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Los Angeles

Predictors of Psychopathic Traits

A dissertation submitted in partial satisfaction of the requirements for the degree

Doctor of Philosophy in Psychology

by

Whitney Allison Brammer

2016

ABSTRACT OF THE DISSERTATION

Predictors of Psychopathic Traits

by

Whitney Allison Brammer

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2016

Professor Steve Sung-Yul Lee, Chair

Despite replicated evidence on the predictive validity of psychopathic traits with respect to later antisocial behavior (ASB), little is known about potential risk factors for youth psychopathic traits. This knowledge may facilitate the development of targeted interventions to ameliorate and possibly prevent later ASB and related problems. Functional genotypes regulating dopamine neurotransmission are strong candidates for causal influences on psychopathic traits; similarly, parenting behavior may not only independently predict psychopathic traits, but it may also interact with youth genotype. Second, reflecting dynamic exchanges secondary to temperament (e.g., evocative gene-environment correlation), parenting behavior may mediate the association of temperament and psychopathic traits. This dissertation tested gene-environment interplay for psychopathic traits and identified mechanisms in their development using multi-method measures of parenting behavior and biologically plausible polymorphisms from candidate genes (i.e., DAT1, DRD4, DRD2). Based on two independent, yet complementary prospective longitudinal samples of 235 youth with and without ADHD who were assessed after

two years (UCLA ADHD and Development Study) and 2,500 adolescents followed across 12 years (National Longitudinal Study of Adolescent Health), we aimed to identify potential risk factors for psychopathic traits and tested parenting behavior as a mediator of their emergence from temperament dimensions. Primary findings were three-fold: (1) across both samples, both negative and positive parenting practices uniquely predicted the development of psychopathic traits into later childhood and adulthood; (2) negative and positive parenting practices significantly moderated the associations of biologically-plausible polymorphisms from dopaminergic genes with later psychopathic traits, callous-unemotional traits, and narcissism, although patterns of moderation were not fully parallel across samples; (3) parenting behavior significantly mediated the associations of early temperament dimensions with later callous-unemotional traits and, in particular, narcissism. In addition to providing evidence for negative and positive parenting as well as dopaminergic functioning underlying psychopathic traits, these results are unique insofar as identifying putative mediators to psychopathic traits. We discuss the insight provided by our preliminary findings into risk factors for psychopathic traits, implications for evidence-based interventions, as well as the dynamic processes underlying their development as well as additional research required to further clarify pathways of risk and resilience underlying youth psychopathic traits.

The dissertation of Whitney Allison Brammer is approved.

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2016

Table of Contents

LIST OF TABLES & FIGURES.....	VI
ACKNOWLEDGMENTS.....	VII
INTRODUCTION	1
PROPOSED STUDIES	5
REFERENCES	7
CHAPTER ONE: ASSOCIATION OF DAT1, DRD4, DRD2, AND PSYCHOPATHIC TRAITS: MODERATION BY PARENTING BEHAVIOR.....	12
STUDY 1: INTERACTION BETWEEN POSITIVE AND NEGATIVE PARENTING BEHAVIOR WITH DAT1 AND DRD4 GENOTYPE: PREDICTIONS OF YOUTH CALLOUS-UNEMOTIONAL TRAITS AND NARCISSISM	13
Method	19
Results	24
Discussion	28
References	38
STUDY 2: INTERACTIVE EFFECTS OF PARENTING WITH DAT1, DRD4, AND DRD2 GENOTYPES ON YOUNG ADULT PSYCHOPATHIC TRAITS	51
Method	57
Results	62
Discussion	67
References	75
CHAPTER TWO: THE ASSOCIATION OF TEMPERAMENT AND PSYCHOPATHIC TRAITS: MEDIATION BY PARENTING BEHAVIOR	83
STUDY 3: POSITIVE AND NEGATIVE PARENTING BEHAVIOR MEDIATES PREDICTIONS OF CHILDHOOD PSYCHOPATHIC TRAITS FROM PROSOCIALITY AND DARING	84
Method	89
Results	94
Discussion	98
References	108
STUDY 4: PARENTING BEHAVIOR MEDIATES PREDICTIONS OF ADULT PSYCHOPATHIC TRAITS FROM CHILDHOOD SELF-REGULATION	119
Method	123
Results	128
Discussion	129
References	140
CONCLUSIONS	150
REFERENCES	155

List of Tables & Figures

Study 1, Table 1. Means and Percentages for Demographic Variables.....	34
Study 1, Figure 1. Predicted Wave 2 Parent+Teacher Composite of Narcissism from Observed Praise and Additive DAT1 Genotype Interaction.....	35
Study 1, Figure 2. Predicted Wave 2 Parent+Teacher Composite of Narcissism from Observed Negative Talk and Additive DAT1 Genotype Interaction.....	36
Study 1, Figure 3. Predicted Wave 2 Parent+Teacher Composite of Narcissism from Observed Praise and Dominant DAT1 Genotype Interaction.....	37
Study 2, Table 1. Demographic Information for National Longitudinal Study of Adolescent Health (Add Health)	72
Study 2, Figure 1. Predicted Wave III Narcissism from Parental Closeness and Additive DAT1 Genotype Interaction.....	73
Study 2, Figure 2. Predicted Wave IV Callous-Unemotional Traits from Parental Involvement and DRD4 Genotype Interaction.....	74
Study 3, Table 1. Means and Percentages for Demographic Variables.....	104
Study 3, Table 2. Mediation by Negative and Positive Parenting Behavior on Temperament Dimensions with Callous-Unemotional Traits and Narcissism.....	105
Study 3, Figure 1. Multiple Mediator Model of Wave 2 Callous-Unemotional Traits by Parenting Behaviors and Prosociality (Beta Coefficients).....	106
Study 3, Figure 2. Multiple Mediator Model of Wave 2 Narcissism by Parenting Behaviors and Prosociality (Beta Coefficients).....	107
Study 4, Table 1. Demographic Information for National Longitudinal Study of Adolescent Health.....	135
Study 4, Table 2. Mediation of the Prediction of Wave I Self-Regulation on Psychopathic Traits, Callous-Unemotional Traits, and Narcissism via Wave II Positive Parenting Behavior and Maltreatment.....	136
Study 4, Figure 1. Multiple Mediator Model of Wave IV Psychopathic Traits by Parenting Behaviors and Wave I Self-Regulation (Beta Coefficients).....	137
Study 4, Figure 2. Multiple Mediator Model of Wave IV Callous-Unemotional Traits by Parenting Behaviors and Wave I Self-Regulation (Beta Coefficients).....	138
Study 4, Figure 3. Multiple Mediator Model of Wave III Narcissism by Parenting Behaviors and Wave I Self-Regulation (Beta Coefficients).....	139

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SELECTED PRESENTATIONS

Brammer, W. A., & Lee, S. S. (2015, July). Association of temperament and psychopathic traits mediated by parenting behavior. *Poster presented at the International Society for Research on Child and Adolescent Psychopathology 17th Scientific Meeting*. Portland, Oregon.

Galan, C., Brammer, W.A., & Lee, S.S. (2013, November). Child ADHD and ODD mediate parental ADHD and marital satisfaction. *Poster presented at the 25th Annual Conference on ADHD*. Washington, DC.

Lee, S. S., Brammer, W. A., & Tung, I. (2013, June). Parenting behavior mediates association of parent ADHD and offspring externalizing behavior. *Symposium presented at the International Society for Research on Child and Adolescent Psychopathology 16th Scientific Meeting*. Leuven, Belgium.

Brammer, W. A., & Lee, S. S. (2013, June). Interactive effects of observed parental praise & DAT1 on child callous-unemotional traits. *Poster presented at the International Society for Research on Child and Adolescent Psychopathology 16th Scientific Meeting*. Leuven, Belgium.

Moses, J.O., Brammer, W. A., & Lee, S. S. (2012, April). The potential moderating role of callous-unemotional traits between reactive and proactive aggression on conduct disorder. *Poster presented at the Annual Stanford Undergraduate Psychology Conference*. Palo Alto, California.

Brammer, W. A., & Lee, S. S. (2011, June). Interactive effects of ADHD and psychopathic traits on externalizing behavior in children. *Poster presented at the International Society for Research on Child and Adolescent Psychopathology 15th Scientific Meeting*. Chicago, Illinois.

Brammer, W. A., & Lee, S. S. (2011, March). Social functioning in children with attention-deficit/hyperactivity disorder and callous-unemotional traits. *Poster presented at the Society for Research in Child Development 2011 Biennial Meeting*. Montreal, Canada.

Brammer, W. A., & Lee, S. S. (2009, May). Impairment in children with and without ADHD: Contributions from aggression and callous-unemotional traits. *Paper presented at the 9th Annual Stanford Undergraduate Psychology Conference*. Palo Alto, California.

Introduction

Youth antisocial behavior (ASB), including aggression, property destruction, and delinquency, constitutes the most costly (e.g., criminal justice, social services) mental health problem in North America (Welsh et al., 2008). Consisting of individual differences in callous-unemotional (CU) traits (i.e., low empathy, shallow emotions), narcissism (i.e., grandiose and egocentric sense of self), and impulsivity, psychopathic traits frequently co-occur with ASB and they predict violent and severe ASB across the lifespan (Frick, Bodin, & Barry, 2000; Frick, Marsee, & Patrick, 2006). Despite considerable evidence on predictions of outcome *from* psychopathic traits, far less is known about correlates of youth psychopathic traits. Individual differences in psychopathic traits appear early in development (Kimonis, Frick, Fazekas, & Loney, 2006) and therefore represent a potentially promising target for intervention, an important consideration given that adult psychopathy is highly treatment resistant (Lynam, 1997). Given its clinical and public health significance, particularly when accompanied by psychopathic traits, identifying risk factors for youth ASB is likely to facilitate innovations in the development and delivery of targeted intervention and prevention efforts.

Negative parenting behavior, including harsh parenting and maltreatment (e.g., physical, sexual, and emotional abuse and neglect), strongly predicts youth ASB. In a meta-analysis, parenting behavior overall, and negative parenting in particular, explained 11% of the variance in delinquency (Hoeve et al., 2009). Although negative parenting behavior, including maltreatment, is associated with psychopathic traits (Barker, Oliver, Viding, Salekin, & Maughan, 2011; Bernstein, Stein, & Handelsman, 1998), less is known about the association of *positive* parenting behavior (e.g., warmth) and psychopathic traits, despite its factorial independence from negative parenting behavior. Preliminary evidence suggests that positive parenting behavior inversely

predicts psychopathic traits over time, even with control of demographic factors (Pardini, Lochman, & Powell, 2007). Thus, overall, surprisingly little is known about the incremental validity of negative and positive parenting behavior, with control of key covariates, with respect to youth psychopathic traits. Previous studies were limited by the use of self-or youth-reported parenting behavior; thus, observational measures of parenting behavior should be prioritized given that they are less susceptible to reporting biases and significantly increment predictions of child outcome beyond maternal self-report (Zaslow et al., 2006). Finally, a recent review highlighted that whereas parenting behavior prospectively predicted CU traits per se (Waller, Gardner, & Hyde, 2013), far less is known regarding its prediction of other psychopathy facets, most notably narcissism. To directly address these limitations, the current proposal examined the association of self-reported and objectively-coded observed positive and negative parenting behavior with respect to CU traits and narcissism, controlling for key correlates of psychopathic traits, including attention-deficit/hyperactivity disorder (ADHD).

Individual differences in youth psychopathic traits are substantially heritable (Bezdjian, Raine, Baker, & Lynam, 2011; Viding, Blair, Moffitt, & Plomin, 2005). However, genetic association studies are necessary to elucidate the underlying pathophysiology of psychopathic traits, potentially signaling targets for intervention. Given that dopamine neurotransmission is central to the structure and connectivity of neural structures involved in the modulation of negative emotion, aggression, and psychopathy (Beiderbeck et al., 2012; Buckholtz et al., 2010), functional genetic variants regulating dopamine are biologically plausible risk factors. This dissertation focused specifically on polymorphisms in the dopamine transporter (DAT1), dopamine receptor D4 (DRD4), and dopamine receptor D2 (DRD2) genes, given their association with antisocial-spectrum phenotypes across the lifespan (e.g., violent delinquency,

novelty-seeking, criminal arrests, impulsivity) (Basoglu et al., 2011; Beaver, Wright, & Walsh, 2008; Burt & Mikolajewski, 2008; Guo et al., 2007; Reiner & Spangler, 2011).

Beyond heritability, gene-environment correlation (rGE) and gene x environment interaction (G x E) are likely relevant to psychopathic traits (Rutter & Silberg, 2002). Given evidence that parenting behavior is associated with psychopathic traits, and the centrality of dopamine neurotransmission to both constructs (Guo et al., 2007; Sheese et al., 2007), the current proposal prioritized individual differences in caregiving behavior (e.g., positive/negative parenting; maltreatment) as biologically plausible moderators of the association of DAT1, DRD2, and DRD4 with respect to youth psychopathic traits. Specifically, we evaluated a theoretical model of G x E known as differential susceptibility (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky, 2005). Traditional G x E studies assume a diathesis-stress framework where genetic vulnerability exacerbates predictions of negative outcome from environmental adversity. However, differential susceptibility contends that some genetic variants (i.e., “plasticity” genes) simultaneously amplify sensitivity to negative *and* positive environments (see Figure 1). Thus, both adverse and enriching environmental factors may moderate genetic effects. There is preliminary correlational and experimental (i.e., intervention) evidence for differential susceptibility, across multiple plasticity dimensions (e.g., genes, physiological reactivity) and environmental factors (e.g., maternal care, poverty), for both children and adults (Belsky & Pluess, 2013). More specifically, there is meta-analytic evidence suggestive of differential susceptibility where interactions of youth DAT1, DRD4, DRD2 with parenting behavior predicted outcomes ranging from ADHD and externalizing and behaviors to sensation-seeking and prosocial behavior (Bakermans-Kranenburg & van IJzendoorn, 2011). Although DAT1, DRD4, and DRD2 are associated with psychopathic traits (Beaver et al., 2008;

Benning, Patrick, Hicks, Blonigen, & Krueger, 2003), it is unclear whether or not G x E effects reflect a diathesis stress framework (e.g., specifically confers vulnerability) and/or a differential susceptibility framework. This proposal thus evaluated G x E with respect to DAT1, DRD2, and DRD4 and parenting behavior in predictions of psychopathic traits, including careful evaluation of established criteria for differential susceptibility (Belsky et al., 2007).

Beyond testing putative G x E effects for psychopathic traits, I evaluated explanatory factors underlying (i.e., mediating) genetic influences on psychopathic traits. Temperament, defined as individual differences in reactivity and self-regulation expressed as emotionality, motor activity, and attention, is substantially heritable (Rothbart & Derryberry, 1981; Saudino, 2005) and reliably associated with psychopathic traits and common forms of psychopathology (Glenn, Raine, Venables, & Mednick, 2007; Lahey et al., 2008). Dimensions of temperament are evident early in development, stable, substantially heritable, and they prospectively predict psychopathic traits (Glenn et al., 2007; Lahey et al., 2008), although the mechanisms underlying these associations are unclear. In addition to its association with psychopathology, temperament similarly is associated with differences in exposure to parenting behaviors. Observed maternal sensitivity was more strongly inversely associated with teacher-reported child externalizing behavior in first grade for youth with difficult temperament (i.e., high negative emotionality and reactivity) relative to those with easy temperament (Bradley & Corwyn, 2008). The association of temperament dimensions and exposure to differences in parenting behavior is thought to reflect processes such as evocative gene-environment correlation (rGE) where child genetically-based traits such as temperament elicit environmental responses. A recent meta-analysis of the determinants of parenting behavior highlighted the role of child genetic and environmental influences on parenting behavior, further substantiating evocative rGE (Klahr & Burt, 2013). The

current proposal directly addressed whether individual differences in dimensions of temperament are linked to liability to the experience of negative and positive parenting that may in turn predict later psychopathology and psychopathic traits.

Overall, despite replicated evidence on the predictive validity of psychopathic traits with respect to later ASB and negative outcomes, little is known about potential risk factors for youth psychopathic traits. Functional genotypes regulating dopamine neurotransmission are strong candidates for causal influences on psychopathic traits; similarly, parenting behavior may not only independently predict psychopathic traits, but it may also interact with youth genotype. Potential interactive effects, including G x E, may reflect diathesis-stress and differential susceptibility. Second, reflecting dynamic exchanges secondary to temperament (e.g., evocative rGE), parenting behavior may mediate the association of temperament and psychopathic traits. This dissertation tested gene-environment interplay for psychopathic traits and identified mechanisms in their development using multi-method measures of parenting behavior and functional polymorphisms from strong candidate genes (i.e., DAT1, DRD4, DRD2). Based on two independent, yet complementary prospective longitudinal samples of 235 youth with and without ADHD followed across four years and 2,500 adolescents followed across 12 years, I aimed to discern potential risk factors underlying psychopathic traits, as well as parenting behavior as a mediator of their emergence from temperament dimensions. Crucially, this knowledge may facilitate the development of targeted intervention and prevention efforts to ameliorate and possibly prevent later ASB and other negative outcomes associated with psychopathic traits.

Proposed Studies

My dissertation consisted of two chapters and two studies within each chapter. To evaluate biologically-plausible gene-environment interplay underlying psychopathic traits, Chapter One examined whether positive and negative parenting behavior moderated the association of dopaminergic genes on psychopathic traits, in addition to careful evaluation of diathesis-stress or differential susceptibility criteria. To elucidate potential causal mechanisms, Chapter Two evaluated whether dimensions of parenting behavior mediated the association of child temperament facets (i.e., daring, prosociality, self-regulation) and psychopathic traits. Each chapter utilized two independent and complementary samples: the UCLA ADHD and Development Study and the National Longitudinal Study of Adolescent Health (Add Health). The ADHD study consists of a prospective longitudinal design with approximately 235 5 to 10 year-olds with and without ADHD who were assessed after two years (7 to 12 years-old; Wave 2). Add Health consists of a prospective longitudinal study of 2,500 adolescents in grades 6 through 12 followed across 12 years (24-32 years-old). Each sample provides individual strengths well-suited to addressing these research questions, such as structured diagnostic interviews and multi-method measures of constructs in the UCLA ADHD Study and a large sample of children followed into adulthood in Add Health.

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Chapter One

Association of DAT1, DRD4, DRD2, and Psychopathic Traits: Moderation by Parenting Behavior

Study 1: Interaction between Positive and Negative Parenting Behavior with DAT1 and DRD4 Genotype: Predictions of Youth Callous-Unemotional Traits and Narcissism

Abstract

Although callous-unemotional (CU) traits and narcissism predict negative outcomes (e.g., recidivism), little is known about their etiology. Polymorphisms of the dopamine transporter (DAT1) and dopamine receptor D4 genes, along with negative and positive parenting, have been implicated in their development. We explored interactive associations between multi-method parenting measures with DAT1 and DRD4 genotypes on multi-informant ratings of CU traits and narcissism in a longitudinal study of 230 school-age youth followed into early adolescence; in the presence of significant gene x environment interaction, we also evaluated empirical criteria for differential susceptibility. Controlling for sex and externalizing behavior, negative and positive parenting behavior separately moderated the association of DAT1 with parent- and teacher-rated CU traits and narcissism, with partial support for differential susceptibility. We consider parenting in the context of efficacious interventions for antisocial behavior as well as evidence of gene-environment interplay for psychopathic traits.

Youth antisocial behavior (ASB) constitutes the most costly mental health problem in North America (Welsh et al., 2008). Consisting of callous-unemotional (CU) traits (i.e., diminished remorse and empathy), narcissism (i.e., grandiosity, egocentricity), and impulsivity, psychopathic traits frequently co-occur with ASB and predict severe and violent expressions of ASB (i.e., delinquency, recidivism, adult psychopathy) across the lifespan (Frick, Marsee, & Patrick, 2006). Despite considerable work on predictions of youth outcome *from* psychopathic traits, less is known regarding predictors of psychopathic traits. This is especially true with respect to narcissism, given that etiological models have prioritized CU traits. Given the clinical and public health significance of ASB and the putative role of psychopathic traits, identifying their risk factors will facilitate innovations in the development of targeted interventions for ASB.

Negative parenting behavior reliably predicts youth ASB and psychopathic traits (Barker, Oliver, Viding, Salekin, & Maughan, 2011; Bernstein, Stein, & Handelsman, 1998; Hoeve et al., 2009). In a community sample, negative parenting specifically predicted key facets (i.e., CU traits, narcissism) of psychopathic traits (Frick, Kimonis, Dandreaux, & Farell, 2003). Youth with life-course persistent ASB were also more likely to exhibit attention-deficit/hyperactivity disorder (ADHD) symptoms relative to youth with adolescent-limited ASB (Moffitt, Caspi, 2001), substantiating ADHD as a risk factor for “fledgling psychopathy” (Fowler et al., 2009; Lynam, 1996) and suggesting that samples enriched for ADHD are appropriate for the study of psychopathic traits. A cross-lagged analysis of 1,562 twins indicated that child-directed parental criticism and dismissal predicted growth in psychopathic traits over five years (Tuvblad, Bezdjian, Raine, & Baker, 2013). Less is known about the association of *positive* parenting behavior (e.g., praise, warmth) and psychopathic traits, despite its factorial independence from negative parenting (Borden et al., 2014) and evidence that positive parenting was uniquely

associated with reduced psychopathic traits over time, with control of demographic factors (Pardini, Lochman, & Powell, 2007). After accounting for child abuse and neglect, maternal care was inversely associated with CU traits; moreover, youth with high CU traits and low maternal care exhibited the highest levels of ASB (Kimonis, Cross, Howard, & Donoghue, 2013), although the association of positive parenting with narcissism specifically is unknown. Notably, many studies did not account for concurrent ASB, limiting inferences about the incremental validity of parental behaviors to psychopathic traits beyond child ASB. Similarly, because parent- and child-reported parenting behavior is vulnerable to reporting biases (Zaslow et al., 2006), the current study employed ratings of objectively-coded negative and positive parenting from a validated structured parent-child interaction task.

Although individual differences in youth psychopathic traits are moderately heritable (Bezdjian, Raine, Baker, & Lynam, 2011; Viding, Blair, Moffitt, & Plomin, 2005), genetic association studies are necessary to elucidate their underlying pathophysiology. Evidence from human and animal models suggests that genetic variants affecting dopamine neurotransmission are important. During a resident-intruder task, rats selectively bred for low anxiety demonstrated elevated aggression and increased local dopamine release; a local infusion of a dopamine D2 receptor antagonist effectively reduced rats' aggression, crucially indicating that experimentally-manipulated dopamine significantly influenced aggression (Beiderbeck et al., 2012). Given that dopamine neurotransmission is central to the structure and connectivity of neural structures involved in the modulation of negative emotion, aggression, and psychopathy (Beiderbeck et al., 2012; Buckholtz et al., 2010), functional genetic variants regulating dopamine are biologically plausible. Given their association with antisocial-spectrum phenotypes (e.g., violent delinquency, novelty-seeking, criminality), we examined the 40 base pair (bp) variable number of tandem

repeat (VNTR) polymorphism located in the 3' untranslated region of exon 15 of the dopamine transporter (DAT1) gene and 48 bp VNTR polymorphism in exon 3 of the dopamine receptor D4 (DRD4) gene (Basoglu et al., 2011; Reiner & Spangler, 2011).

DAT1 shares a common genetic liability with psychopathy (Bornovalova, Hicks, Iacono, & McGue, 2010; Hicks, Krueger, Iacono, McGue, & Patrick, 2004). The DAT1 10-repeat allele was positively associated with criminal behavior (Beaver, Wright, & Walsh, 2008) whereas male carriers of the 10-repeat allele of DAT1 had elevated violent delinquency compared to males homozygous for the 9-repeat allele (Guo et al., 2007). Carriers of the DAT1 9-repeat allele also demonstrated reduced novelty-seeking, a key personality trait among psychopaths (Basoglu et al., 2011), relative to those without a 9-repeat allele (Sabol et al., 1999). There is also meta-analytic evidence that DAT1 10-repeat allele is associated with ADHD (Gizer, Ficks, & Waldman, 2009), a correlate of psychopathic traits (Fowler et al., 2009). Although there is replicated evidence that the 10-repeat allele is associated with antisocial phenotypes, evidence also suggests positive associations of the 9-repeat allele and externalizing behavior in children (Young et al., 2002) and adults (Gerra et al., 2005). Given the replicated association of the 10-repeat allele of DAT1 and externalizing phenotypes, it may be similarly linked to psychopathic traits, although this specific association has not yet been explicitly tested.

Similarly, DRD4 is associated with externalizing phenotypes related to psychopathy (Bornovalova et al., 2010). Adult carriers of the 7-repeat allele self-reported elevated impulsivity, a key dimension of psychopathy in children and adults (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Frick, Bodin, & Barry, 2000; Reiner & Spangler, 2011). In a high-risk community sample of men, the 7-repeat allele was also associated with self-reported novelty-seeking (Laucht, Becker, El-Faddagh, Hohm, & Schmidt, 2005), a reliable correlate of

psychopathic traits (Basoglu et al., 2011). Using transmission disequilibrium test analyses, the 7-repeat allele was significantly associated with comorbid ADHD and ASB, providing additional support for their common genetic vulnerability (Holmes et al., 2002). Preschoolers exposed to maternal insensitivity with the 7-repeat allele were seven times more likely to exhibit oppositional/aggressive behaviors relative to unexposed or non 7-repeat youth (Bakermans-Kranenburg & Van IJzendoorn, 2006), suggesting that the DRD4 7-repeat allele may amplify the association of negative parenting and externalizing problems. Similarly, 7-repeat allele children exposed to elevated prenatal maternal stress exhibited higher rates of disruptive behavior disorders relative to youth with low levels of prenatal maternal stress and non-carriers of the 7-repeat allele (Zohsel et al., 2014). Given that the DRD4 7-repeat allele is associated with ASB and related correlates of psychopathic traits, its specific association with psychopathic traits requires further evaluation.

Beyond heritability, gene-environment correlation (rGE) and gene x environment interaction (G x E) are likely relevant to psychopathic traits (Rutter & Silberg, 2002). Given emergent evidence on the association of parenting behavior and psychopathic traits, and the centrality of dopamine neurotransmission to both constructs (Guo et al., 2007; Sheese, Voelker, Rothbart, & Posner, 2007), the current study prioritized individual differences in negative and positive parenting as biologically plausible moderators of the association of DAT1 and DRD4 with respect to youth psychopathic traits. We also formally tested differential susceptibility (Belsky, 2005; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007), which contends that some genetic variants (i.e., ‘plasticity’ genes) amplify sensitivity to negative *and* positive environments. Thus, both adverse and enriching environmental factors may moderate genetic effects. There is correlational and experimental evidence for differential susceptibility, across

multiple plasticity dimensions (e.g., genes, physiological reactivity) and environmental factors (e.g., maternal care, poverty) for both children and adults (Belsky & Pluess, 2013). Notably, there is meta-analytic evidence suggestive of differential susceptibility where interactions of youth DAT1 and DRD4 with parenting behavior predicted outcomes ranging from ADHD and externalizing and behaviors to sensation-seeking and prosocial behavior (Bakermans-Kranenburg & van Ijzendoorn, 2011). Observed parenting also moderated the association of DAT1 on a latent ADHD factor, such that praise was positively associated and negativity was inversely associated with ADHD in youth with at least one 9-repeat allele (Li & Lee, 2013). Although DAT1 and DRD4 are associated with psychopathic traits (Beaver et al., 2008; Benning et al., 2003), it is unclear whether parenting moderates these predictions and whether G x E effects reflect a diathesis stress framework and/or a differential susceptibility framework.

Current Study and Hypotheses

Functional genotypes regulating dopamine neurotransmission are strong candidates for causal influences on psychopathic traits; similarly, parenting behavior may not only independently predict psychopathic traits, but may also interact with youth genotype. Potential interactive effects must be further tested from both diathesis-stress and differential susceptibility models. The current study tested gene-environment interplay predicting psychopathic traits, using multi-method measures of parenting behavior as well as DAT1 and DRD4 in a prospective longitudinal sample of 230 youth with and without ADHD followed across two years into early adolescence. We predicted that self-reported and observed positive and negative parenting behavior would moderate the association of DAT1 and DRD4 with psychopathic traits, such that both negative and positive parenting would be most strongly associated with psychopathic traits in carriers of the 10-repeat allele of DAT1 and 7-repeat allele of DRD4. Moreover, we predicted

that that these models would reflect differential susceptibility, with carriers of the 10-repeat allele of DAT1 and 7-repeat allele of DRD4 exhibiting more susceptibility to negative and positive parenting behavior.

Method

Participants

At baseline (i.e., Wave 1), 230 ethnically diverse (55% Caucasian; 7% African American; 9% Hispanic; 3% Asian; 23% other) six to ten year-old ($M=7.4$, $SD=1.1$) youth with and without ADHD were recruited using advertisements in local schools and public locations in addition to referrals from local medical and mental health service providers (Table 1). Inclusion criteria consisted of children living with at least one biological parent at least half time, full-time school enrollment, and English fluency. Exclusion criteria consisted of a Full Scale IQ (FSIQ) < 70 , an autism spectrum, seizure, or any neurological disorder. ADHD proband status was based on a fully structured diagnostic interview with the parent. To avoid recruiting a sample of improbably high-functioning youth, non-ADHD comparison children who met diagnostic criteria for any disorder other than ADHD were placed into a group of non-ADHD comparison youth. All participants were recruited, screened, and assessed using identical procedures.

Procedures

Families who contacted the study at baseline first completed a telephone screener to determine their eligibility based on the inclusion and exclusion criteria listed above. Rating scales were mailed to families who satisfied these criteria ($n = 230$) and they were subsequently invited to our research laboratory for assessments. Following signed consent and assent procedures for the parent and child, respectively, clinical psychology graduate students or B.A.-level trained staff assessed children while a second member of the research staff concurrently

interviewed parents about their child's psychopathology. All interviewers were initially blind to the child's diagnostic status, but the blind could not always be preserved given the extensive information gathered about the child. Parents were asked to rate each child based on his/her unmedicated behavior. Approximately 85% of children were evaluated without their medication during the assessment (most were treated with stimulant medication).

Approximately two years later (i.e., Wave 2), 91% ($n = 209$) of the families returned with their eight to 12 year-old children for a follow-up, which featured procedures highly parallel to Wave 1. Relevant domains of inquiry included family functioning, youth academic achievement, and child psychopathology. Relative to non-participating families, Wave 2 families reported a higher average number of child ADHD symptoms, $t(226) = -2.08$, $p = .04$; no other significant differences were observed with respect to the child's age and sex as well as parent sex, race-ethnicity, depression, and ADHD. The IRB approved all study procedures.

Measures

Genotype. DNA was extracted from saliva using DNA Genotek Oragene Self-Collection Kits (DNA Genotek, Inc., Ottawa, CA). First, the 40-bp variable VNTR polymorphism in the 3'untranslated region of exon 15 was genotyped yielding the two most common alleles for DAT1: the 9-repeat (440 bp) and 10-repeat (480 bp) polymorphisms. DAT1 genotype frequencies in our sample were distributed as follows: 9/9 (7.3%), 9/10 (36%), and 10/10 (56.7%). These frequencies were in Hardy-Weinberg equilibrium, $\chi^2(1) = 0.25$, $p = .62$. Second, the 48 bp VNTR polymorphism in located on chromosome 11p15.5 in exon 3 was genotyped. Given that the 4-repeat and 7-repeat polymorphisms are the most common, genotype frequencies of DRD4 in our sample were distributed as follows: 7/7 (6.8%), 7/4 (36.35%), 4/4 (57.5%). These frequencies were in Hardy-Weinberg equilibrium, $\chi^2(1) = 0.17$, $p = .68$. DAT1 was

additively coded 0, 1, and 2 for the 9/9, 9/10, and 10/10 genotypes, respectively, whereas DRD4 was coded 0 and 1 for genotypes without a 7-repeat allele and genotypes with a 7-repeat allele, respectively.

Self-reported parenting behavior. Parents self-reported 42 parenting behaviors on the Alabama Parenting Questionnaire (APQ) at Wave 1 on the frequency of parental involvement, positive parenting, inconsistent parenting, poor monitoring, and corporal punishment dimensions (Frick, 1991). APQ factors converged with observed parenting (Hawes & Dadds, 2006; Chronis-Tuscano et al., 2008). Given that factor structure of the APQ has varied greatly across different studies (Hinshaw et al., 2000; Hawes & Dadds, 2006; Shelton, Frick, & Wootton, 1996), we factor analyzed the APQ data at Wave 1 and uncovered four factors: positive reinforcement, parental involvement, parental monitoring/supervision, and corporal punishment (blinded for review). We did not examine parental monitoring/supervision as it is largely applicable to adolescent ASB (Frick, Christian, & Wootton, 1999). The Cronbach's alphas were .78 for positive reinforcement, .78 for parental involvement, and .61 for corporal punishment at Wave 1. To reduce Type I error and account for their intercorrelation ($r = .53, p < .01$), positive reinforcement and parental involvement were standardized and summed to create a composite positive parenting dimension.

Observed parenting behavior. At Wave 1, parents and child completed a structured parent-child interaction task. Eighty-eight percent of parents who participated in the task were mothers. Measures of dimensional observed parenting were coded using the Dyadic Parent Child Interaction Coding System (DPCICS), yielding the following composites: parental negative talk and praise (Eyberg, Nelson, Duke, & Boggs, 2005). Negative talk was coded in response to parents expressing a verbal criticism or using sarcastic or rude speech toward the child. Praise

was coded in response to a labeled or unlabeled positive judgment of the parent toward the child. Total counts of parental behaviors were counted and divided by total minutes coded. Six research assistants were intensively trained in the DPICS coding protocols for two months until 70% agreement was reached. Specifically, coders received a full day of training followed by weekly coding meetings over 2 months where each coding category was discussed with reviews/quizzes in order to increase reliability and to resolve disagreements. Twenty percent of the videos were randomly selected and coded by two raters in order to estimate reliability. The DPICS has been established as valid in studies of youth with externalizing behaviors (Robinson & Eyberg, 1981) and exhibits moderate to high interrater and test–retest reliability (Chronis-Tuscano et al., 2008). The intraclass correlations (ICC) were .75 for negative talk and .88 for praise.

Psychopathic traits. At Waves 1 and 2, parents and teachers separately completed the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), a 20-item rating scale of youth psychopathic traits yielding three factors: CU traits, narcissism, and impulsivity/conduct problems (Frick et al., 2000). The impulsivity/conduct problems factor was excluded given its redundancy with ASB. Narcissism was estimated from the sum of seven items: “Emotions seem shallow,” “Braggs excessively,” “Uses or ‘cons’ others,” “Teases or makes fun of others,” “Can be charming,” “Becomes angry when corrected,” and “Seems to think he is more important.” Recent confirmation of the three factor model and acceptable psychometric properties were indicated in clinical and community samples (Dong, Wu, & Waldman, 2013), with narcissism demonstrating stability and convergence between parent and teacher ratings in multiple samples (Frick et al., 2000). We combined separate ratings of narcissism using the higher score from parent and teacher reports to generate a composite measure of narcissism (P+T; Piacentini, Cohen, & Cohen, 1992), an approach that previously predicted youth delinquency (Barry et al.,

2007; Frick & Hare, 2001) and also conservatively reduces the number of statistical tests (Lahey et al., 1998). The P+T narcissism scale at Wave 2 had a Cronbach alpha of .74.

At Wave 2, parents and teachers also completed the Inventory of Callous-Unemotional Traits (ICU), a 24 item measure of CU traits rated on a 4-point Likert scale (Frick, 2004). The ICU exhibits high internal consistency (Hawes et al., 2014) as well as appropriate convergent validity with sensation seeking, delinquency, and divergent validity with physiological and self-report measures of emotional reactivity across multiple samples (Essau, Sasagawa, & Frick, 2006; Kimonis et al., 2008). Similar to narcissism, we created a composite measure of CU traits by using the higher score from parent and teacher reports of CU traits (Piacentini et al., 1992). The Cronbach alpha for P+T CU traits at Wave 2 was .90.

ADHD and ODD. At Waves 1 and 2, we administered the Diagnostic Interview Schedule for Children, Version IV (DISC-IV) to each child's parent to derive the diagnostic status of ADHD (any subtype) and oppositional defiant disorder (ODD; Shaffer et al., 2000). The DISC-IV is an extensively-validated and psychometrically sound structured interview. In the DSM-IV Field Trials, test-retest reliability for DISC-IV ADHD ranged from .51 to .64 (Lahey et al., 1994).

Data Analytic Plan

Our goal was to test the independent association of Wave 1 observed parenting behaviors and dopamine genotypes with respect to Wave 2 CU traits and narcissism. Separate models predicted P+T rated CU traits and narcissism from DAT1 and DRD4 genotype, along with Wave 1 observed and self-reported parenting dimensions. We entered main effects for Wave 1 positive parenting (i.e., reported positive parenting or observed praise), negative parenting dimensions (i.e., reported corporal punishment or observed negative talk) and genotype at Step 2. Finally, we

entered separate interactions of genotype with positive and negative parenting behaviors in Step 3 as predictors of Wave 2 CU traits and narcissism. Parenting variables were centered using the sample mean (Aiken & West, 1991). Given that parent- and teacher-reported CU traits were normally distributed, we employed multiple linear regression in tests of CU traits. Parent- and teacher-reported narcissism, however, were significantly skewed; thus, we fit a negative binomial regression in predictions of child narcissism. We controlled for Wave 1 ADHD diagnostic status (i.e., ADHD versus non-ADHD comparison) and concurrent Wave 1 ASB (e.g., ODD) to improve specificity. Child sex was also controlled in all models.

To evaluate differential susceptibility, we utilized established criteria (Kochanska et al., 2011) by first calculating regions of significance for each parenting x genotype interaction that predicted CU traits and narcissism (Aiken & West, 1991; Hayes & Matthes, 2009). Second, we graphed predicted regression lines at ± 2 SD below and above the mean for parenting behavior variables, and identified regions of significance via shading within figures to indicate below which and above which values of the parenting behavior variable groups of youth with different genotypes significantly differed in regards to the P+T CU traits and narcissism outcomes (Belsky & Pluess, 2013; Kochanska et al., 2011).

Results

Population Stratification

Population stratification secondary to admixture can threaten the internal validity of studies. Population stratification requires that race-ethnicity is significantly associated with DAT1 and DRD4, parenting behaviors, and psychopathic trait outcomes (Hutchison, Stallings, McGeary, & Bryan, 2004). Race-ethnicity was unrelated to DAT1 ($\chi^2(10) = 13.09, p = .22$) and DRD4 ($\chi^2(5) = 6.23, p = .29$); it was similarly unrelated to Wave 1 observed praise ($B = -0.02$,

$SE = 0.02, p = 0.17$), observed negative talk ($B = -0.01, SE = 0.03, p = 0.65$), or reported positive parenting ($B = -0.01, SE = 0.02, p = 0.77$), but was significantly associated with corporal punishment ($B = 0.27, SE = 0.13, p = 0.04$). Lastly, race-ethnicity was not significantly associated with Wave 2 P+T CU traits ($B = 0.02, SE = 0.02, p = 0.28$) nor narcissism ($B = -0.04, SE = 0.07, p = 0.62$). Given that the criteria for population stratification were not met, race-ethnicity was not included as a covariate.

DAT1 on CU Traits: Moderation by Parenting Behaviors

First, we evaluated whether Wave 1 observed praise and negative talk, the additively-coded DAT1 genotype (i.e., 9/9 vs. 9/10 vs. 10/10), and the separate DAT1 x praise and DAT1 x negative talk interactions predicted Wave 2 CU traits, controlling for child sex and Wave 1 ADHD and ODD diagnostic status. There were no significant main effects for observed praise or negative talk ($B = -0.07, SE = 0.19, p = 0.70$ and $B = -0.69, SE = 0.62, p = 0.27$, respectively); we observed a marginal main effect of DAT1 on CU traits, $F(2, 102) = 2.37, p = .10$. The praise x DAT1 and negative talk x DAT1 interactions were unrelated to Wave 2 CU traits [$F(2, 102) = 0.18, p = .83$ and $F(2, 102) = 1.90, p = .15$, respectively].

This model was reproduced with Wave 1 self-reported positive parenting and corporal punishment, youth DAT1, and separate youth DAT1 x positive parenting and youth DAT1 x corporal punishment interactions in predictions of Wave 2 CU traits. There was a significant main effect of DAT1, $F(2, 127) = 6.59, p < 0.01$, a marginal main effect of corporal punishment ($B = -7.01, SE = 3.98, p = 0.08$), but positive parenting was unrelated to CU traits ($B = 0.96, SE = 1.21, p = 0.43$). Although the positive parenting x DAT1 interaction was not significant, $F(2, 127) = 0.99, p = .39$, there was a marginally significant corporal punishment x DAT1 interaction on CU traits, $F(2, 127) = 2.31, p = .10$. Given the limited evidence base on correlates of youth

psychopathic traits, especially with regards to measured gene-environment interplay, we chose to further probe this interaction when DAT1 was also coded recessively (i.e., 9/9 vs. 9/10 and 10/10). When reproducing the model with recessive DAT1, there was a marginal main effect of corporal punishment and recessive DAT1 ($B = -6.85$, $SE = 4.11$, $p = 0.10$, and $B = 6.26$, $SE = 3.83$, $p = 0.10$, respectively), but not positive parenting ($B = 0.95$, $SE = 1.25$, $p = 0.45$). Although there was no significant positive parenting x DAT1 interaction, $F(1, 130) = 1.09$, $p = .30$, the corporal punishment x DAT1 interaction did significantly predict CU traits [$F(1, 130) = 4.40$, $p = .04$], such that corporal punishment was positively associated with CU traits in 9/10 and 10/10 youth ($B = -6.85$, $SE = 4.11$, $p = 0.10$), but not in 9/9 youth ($B = 1.85$, $SE = 0.73$, $p = 0.01$). After evaluating regions of significance at ± 2 SD of the corporal punishment mean score, 9/9 versus 9/10 and 10/10 genotype groups differed significantly at lower levels of corporal punishment, thus contraindicating the presence of differential susceptibility.

DAT1 on Narcissism: Moderation by Parenting Behaviors

Next, we evaluated whether Wave 1 observed praise and negative talk, DAT1, and the separate DAT1 x praise and DAT1 x negative talk interactions significantly predicted Wave 2 narcissism. Although praise was unrelated to narcissism ($B = -0.02$, $SE = 0.01$, $p = 0.16$), negative talk and DAT1 each significantly predicted narcissism ($B = 0.04$, $SE = 0.02$, $p = 0.05$ and $\chi^2(2) = 13.35$, $p < .01$, respectively), in addition to a marginal praise x DAT1 interaction [$\chi^2(2) = 5.35$, $p = .07$], such that praise was marginally and positively associated with Wave 2 narcissism in the 10/10 genotype ($B = 0.04$, $SE = 0.02$, $p = 0.07$), but not in the 9/9 and 9/10 youth ($B = -0.11$, $SE = 0.08$, $p = 0.20$ and $B = -0.02$, $SE = 0.03$, $p = 0.58$, respectively). Regions of significance within ± 2 SD of the praise mean score are summarized in gray shading in Figure 1. The presence of both a lower and upper region of significance are consistent with differential

susceptibility. The negative talk x DAT1 interaction significantly predicted narcissism [$\chi^2(2) = 6.79, p = .03$] such that negative talk was marginally and positively associated with Wave 2 narcissism in 9/9 youth ($B = 0.24, SE = 0.14, p = 0.08$), but not in 9/10 and 10/10 genotypes ($B = -0.05, SE = 0.04, p = 0.17$ and $B = 0.06, SE = 0.04, p = 0.13$, respectively). The presence of a lower and upper bounded region of significance was consistent with differential susceptibility (Figure 2).

Given no evidence with respect to gene-environment interplay for narcissism, we conducted follow-up tests with DAT1 coded dominantly (i.e., 9/9 and 9/10 vs. 10/10) and recessively (i.e., 9/9 vs. 9/10 and 10/10). First, we examined whether Wave 1 observed praise and negative talk, DAT1, and the interactions of DAT1 with praise and negative talk predicted Wave 2 narcissism. There were no significant main effects of praise, negative talk, or DAT1 ($B = -0.01, SE = 0.01, p = 0.30, B = 0.01, SE = 0.01, p = 0.56$, and $B = 0.11, SE = 0.13, p = 0.41$, respectively). Whereas the negative talk x DAT1 interaction was unrelated to narcissism, $\chi^2(1) = 0.31, p = .58$, the praise x DAT1 interaction predicted narcissism, $\chi^2(1) = 3.89, p = .05$, such that praise was marginally and positively associated with narcissism in the 10/10 genotype ($B = 0.05, SE = 0.03, p = 0.07$), but not in the 9/9 and 9/10 genotype ($B = -0.03, SE = 0.03, p = 0.30$). Regions of significance analyses indicated that genotypes only differed at higher levels of praise, contraindicating evidence for differential susceptibility (Figure 3). Second, we evaluated whether Wave 1 observed praise and negative talk, recessive DAT1, and their interactions predicted Wave 2 narcissism. There was a marginal effect of observed negative talk ($B = 0.04, SE = 0.02, p = 0.08$) as well as significant effect of DAT1 on narcissism ($B = -0.55, SE = 0.22, p = 0.01$), but no effect of observed praise ($B = -0.02, SE = 0.01, p = 0.19$). Although the negative talk x DAT1 interaction was not significant, $\chi^2(1) = 2.34, p = .13$, the praise x DAT1 interaction marginally

predicted narcissism, $\chi^2(1) = 3.30$, $p = .07$ such that praise was unrelated to Wave 2 narcissism in the 9/9 genotypes ($B = -0.10$, $SE = 0.09$, $p = 0.22$) and 9/10 and 10/10 genotypes ($B = 0.03$, $SE = 0.02$, $p = 0.15$). Given that DAT1 genotypes did not differ along the dimension of observed praise (i.e., non-significant regions of significance), differential susceptibility was not indicated.

DRD4 on CU Traits & Narcissism: Moderation By Parenting Behaviors

We then investigated highly similar models to those reported above but with DRD4 (presence of 7-repeat allele vs. not). First, controlling for child sex and Wave 1 ADHD and ODD diagnostic status, observed praise, negative talk, and DRD4 were each unrelated to CU traits ($B = -0.13$, $SE = 0.09$, $p = 0.17$, $B = 0.21$, $SE = 0.14$, $p = 0.12$, and $B = 1.73$, $SE = 2.01$, $p = 0.39$, respectively). The praise x DRD4 and negative talk x DRD4 interactions were similarly unrelated to CU traits, $F(1, 105) = 0.09$, $p = .76$ and $F(1, 105) = 0.00$, $p = .96$, respectively.

Next, we evaluated whether Wave 1 observed praise and negative talk, DRD4, and their separate interactions predicted Wave 2 narcissism. Observed praise, negative talk, and DRD4 genotype were each unrelated to narcissism ($B = 0.01$, $SE = 0.01$, $p = 0.42$, $B = 0.01$, $SE = 0.01$, $p = 0.27$, and $B = -0.18$, $SE = 0.15$, $p = 0.22$, respectively). The praise x DRD4 and negative talk x DRD4 interactions were similarly unrelated to narcissism, $\chi^2(1) = 2.10$, $p = .15$ and $\chi^2(1) = 0.62$, $p = .43$, respectively.

Discussion

Despite evidence that dopamine neurotransmission is relevant to psychopathic traits, and that parenting may moderate these associations, these relations have not been formally tested. To address this gap, we evaluated whether positive and negative parenting behavior moderated the associations of DAT1 and DRD4 with parent and teacher-rated CU traits and narcissism two years later. Among a sample originally consisting of 230 ethnically-diverse youth followed

prospectively for two years with 91% retention, Wave 1 observed negative talk and DAT1 positively predicted narcissism. Notably, Wave 1 parenting behaviors significantly moderated the association of DAT1 with separate Wave 2 ratings of CU traits and narcissism. Among youth with the DAT1 10/10 genotype, observed praise positively predicted narcissism two years later, even with control of sex, observed negative parenting, as well as ADHD and ODD diagnostic status. In youth with the 9/9 genotype, observed negative parenting positively predicted narcissism, controlling for observed praise and other key covariates. Moreover, corporal punishment positively predicted CU traits among youth with the DAT1 10/10 genotype. In regards to DRD4, observed parenting did not moderate the association of DRD4 with CU traits or narcissism. These findings provide preliminary evidence that the associations of dopamine polymorphisms with later CU traits and narcissism were moderated by negative and positive parenting.

These findings are consistent with recent evidence that negative parenting behavior predicts the development of CU traits and narcissism (Tuvblad et al., 2013). However, the specific dimensions of negative parenting (e.g., inconsistent, harsh parenting) that predict CU traits and narcissism remain unclear. Moreover, given that positive and negative parenting behavior are factorial independent and that little is known about the putative role of positive parenting and psychopathic traits (Chronis et al., 2007), we addressed this question directly. Interestingly, among youth with the DAT1 10/10 genotype, observed praise positively and uniquely predicted combined parent and teacher-rated child narcissism. This is consistent with recent research that parental overevaluation was a risk factor for later narcissism in a community sample of 565 youth (Brummelman et al., 2015). Future research must clarify the specific processes underlying these observed associations to discern the association of parental praise and

narcissism specifically in youth with the 10/10 DAT1 genotype. For example, 10/10 genotype youth may be more prone to internalize parental over-evaluation, facilitating the development of grandiosity and exaggerated self-perception (Brummelman et al., 2015); praise may also be ineffective and detrimental when it is perceived as insincere (Henderlong & Lepper, 2002). However, the specific mechanisms underlying the relations of parental praise and narcissism in genetically distinct youth as well as how these relations develop from infancy into early adulthood remain unknown. Indeed, efforts have been made to identify predictors of trajectories of CU traits across development (Fontaine, Rijdsdijk, McCrory, & Viding, 2010), but these investigations have not yet been extended to narcissism. Lastly, given evidence that the relations of negative and positive parenting with CU traits and narcissism may be more robust in subgroups of youth (e.g., youth with 10/10 DAT1 genotype in this study and Lahey et al., 2011), future investigation into whether these relations extend to other subgroups of youth at-risk for psychopathic traits may also be useful. For example, reduced amygdala activation while viewing fearful faces represents another early physiological signature of psychopathic traits (White et al., 2012), and thus additional inquiry into disrupted physiological responses to fearful affect in relation to parenting, dopaminergic genes, and psychopathic traits may facilitate further development of interventions for youth at-risk for the development of CU traits and narcissism.

One implication from these preliminary findings is that negative talk and praise are prospective risk factors for youth narcissism. Although current evidence-based treatments for oppositional youth prioritize reduced parental negative talk, praise is typically promoted (Garland, Hawley, Brookman-Frazee, & Hurlburt, 2008). However, it remains unclear to what extent praise is beneficial to narcissistic youth, particularly among specific subgroups (e.g., DAT1 10/10 youth). For example, typical evidence-based parent training practices for aggressive

and defiant youth – such as praise – are challenged given that psychopathic traits predict reduced treatment engagement, elevated attrition, and less favorable treatment outcomes (O’Neill, Lidz, & Heilbrun, 2003; Waschbusch, Carrey, Willoughby, King & Andrade, 2007). More recent evidence suggests that focusing on emotional recognition and providing more intensive parenting interventions may be necessary to effectively treat youth with psychopathic traits (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2014; McDonald, Dodson, Rosenfield, & Jouriles, 2011). However, most interventions have prioritized the treatment of CU traits and psychopathic traits overall, rather than the specific role of youth narcissism. Thus, it remains unclear if tailored interventions are necessary for youth with elevated narcissism.

Interestingly, neither positive nor negative parenting behavior significantly moderated the associations of DRD4 with respect to CU traits and narcissism. However, given evidence that DRD4 interacts with the dopamine receptor D2 (DRD2) gene (i.e., epistasis) in the prediction of ASB (i.e., adolescent CD, adult ASB) in multiple large, population-based samples (Beaver et al., 2007; Boutwell et al., 2014), the current sample is poorly positioned to test this directly. Also of note is the fact that relative to narcissism, CU traits were less sensitive to parenting x genotype interactions. Although corporal punishment positively predicted CU traits in youth with at least one 10-repeat allele, additional investigation is needed to clarify these relations. This may reflect that genes involved in serotonin neurotransmission (e.g., serotonin transporter gene), relative to dopamine neurotransmission, are more heavily implicated within the development of CU traits (Glenn, 2011; Sadeh et al., 2010), although future research is needed to clarify these relations.

Based on established criteria (Kochanska et al., 2011), we observed support for differential susceptibility in the case of the DAT1 x praise and DAT1 x negative talk interactions for narcissism. However, we note that although praise was marginally and positively associated

with narcissism in youth with the 10/10 genotype, negative talk was positively associated with narcissism in youth with the 9/9 *genotype*. Thus, results suggest that the 9 and 10-repeat alleles may both be susceptible to parenting behaviors. One possible explanation of the association of the 9-repeat allele with negative talk and the 10-repeat allele with praise and corporal punishment is allele flipping, or positive associations being found between opposite alleles (e.g., 9-repeat and 10-repeat alleles of DAT1; Clarke & Cardon, 2010). The associations of both the 9-repeat and 10-repeat allele being susceptible to parenting behaviors may also be suggestive of the presence of linkage disequilibrium (i.e., alleles on neighboring loci are non-randomly associated; Pritchard & Przeworski, 2001). The 9-repeat allele is also less prevalent in the population relative to the 10-repeat allele, suggesting larger samples or studies that over-sample the 9-repeat allele, are needed to explore these relations. In contrast, the interaction of DAT1 with corporal punishment for CU traits was consistent with a diathesis stress framework whereby higher levels of corporal punishment conferred more vulnerability for CU traits in youth with the 10/10 genotype. If replicated, current interventions for at-risk youth that target reduction of corporal punishment (McDonald et al., 2011) may be more efficacious in youth with the 10/10 DAT1 genotype, although these questions have yet to be empirically interrogated. Overall, these results suggest multiple pathways to youth CU traits and narcissism.

These findings should be considered in the context of several limitations. First, the modest sample size is underpowered to detect main effects and interactive associations in genetic epidemiology (Duncan & Keller, 2011). Moreover, despite multi-method assessment of parenting behavior (i.e., self-reported and observed), parents may have exhibited socially desirable response styles (Bennett, Sullivan, & Lewis, 2006), thus limiting the generalizability of these parenting data with respect to naturally-occurring parenting behavior (Gardner, 2000). In

addition, the current study only assessed parenting from one caregiver (i.e., mother or father), indicating that the parenting behaviors reported and observed in the current study may not be capturing the full dimension of parenting behaviors that may be present across multiple caregivers that may be present in the home. Lastly, the majority of the sample was male, underscoring the need to characterize the presentation and etiology of psychopathic traits (Wang, Baker, Gao, Raine, & Lozano, 2012).

The current study sought to gain a better understanding of the potential interactive effects of biologically-plausible gene-environment interplay underlying psychopathic traits. These preliminary findings suggest that positive and negative parenting behaviors may moderate the associations of DAT1 and DRD4 with child CU traits and narcissism. Among youth with the DAT1 10/10 genotype, observed praise positively predicted parent- and teacher-reported narcissism whereas corporal punishment positively predicted parent- and teacher-reported CU traits. In youth with the DAT1 9/9 genotype, observed negative talk was positively associated with parent- and teacher-reported narcissism. These models also stringently controlled for co-occurring parenting behaviors, child ADHD and ODD diagnostic status, and sex. Across models, partial support for the theory of differential susceptibility was indicated. We contend that these models provide insight into gene-environment interactive effects underlying youth CU traits and narcissism and that, if replicated, improved intervention and prevention efforts may follow from considering gene-environment interplay for psychopathic traits and associated antisocial outcomes.

Study 1, Table 1

Means and Percentages for Demographic Variables

Variable	Wave 2 ADHD (<i>n</i> =129)	Wave 2 Non- ADHD (<i>n</i> =80)	<i>F</i> / χ^2	<i>P</i>
Age (<i>SD</i>)	7.26 (1.13)	7.46 (1.12)	1.58	.21
% Males	78.21	64	4.57	.03
% Caucasian	47.50	48.06	.01	.94
% Household income < \$70,000	36.25	37.98	.06	.80
WISC-IV FSIQ (<i>SD</i>)	102.68 (15.93)	109.75 (13.86)	10.52	.001
% Wave 2 ODD diagnosis	32.50	10.85	14.95	.001
% Wave 2 CD diagnosis	3.75	0	2.33	.13

Note. Mean differences evaluated by one-way ANOVA or chi-square. *SD* = standard deviation;

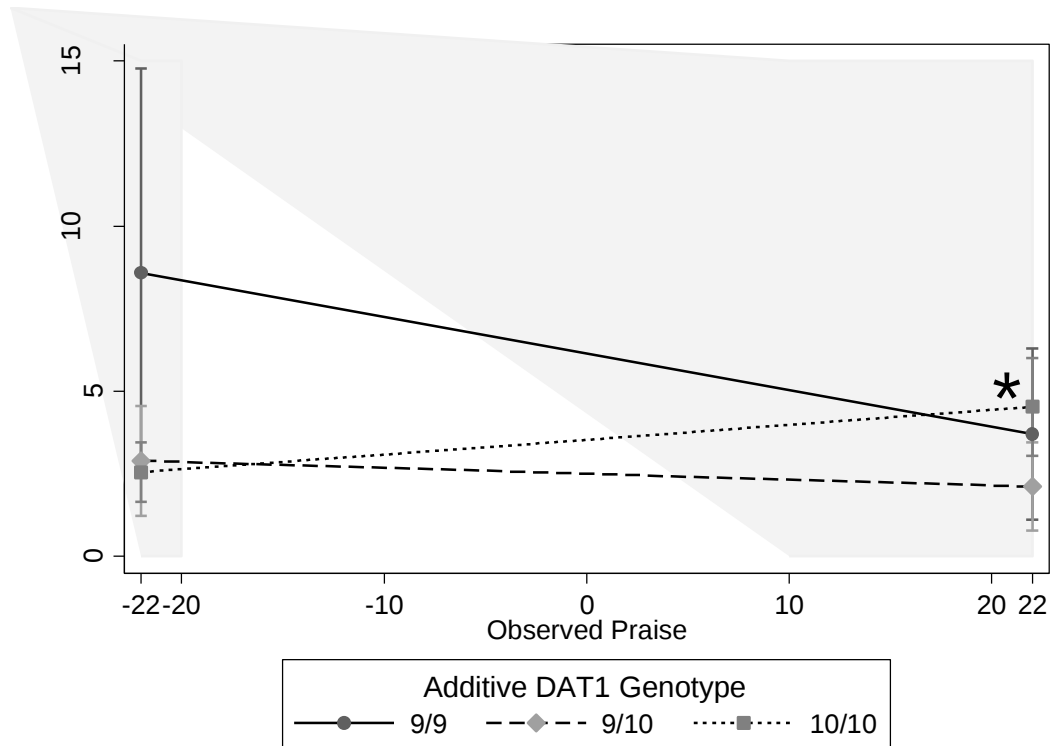
Age = average age at Wave 1 (range: 5–10); WISC-IV FSIQ = average Wechsler Intelligence

Scale for Children, Fourth Edition, Full Scale IQ at Wave 1; ODD = oppositional defiant

disorder; CD = conduct disorder.

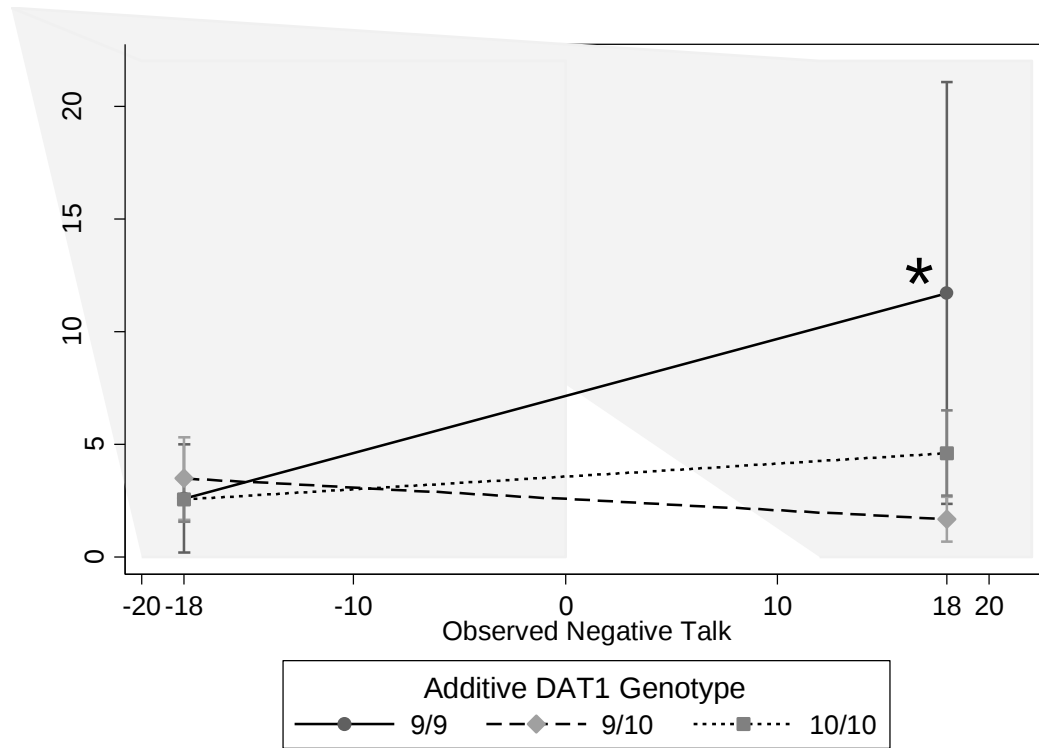
Study 1, Figure 1

Predicted Wave 2 Parent+Teacher Composite of Narcissism from Observed Praise and Additive
DAT1 Genotype Interaction



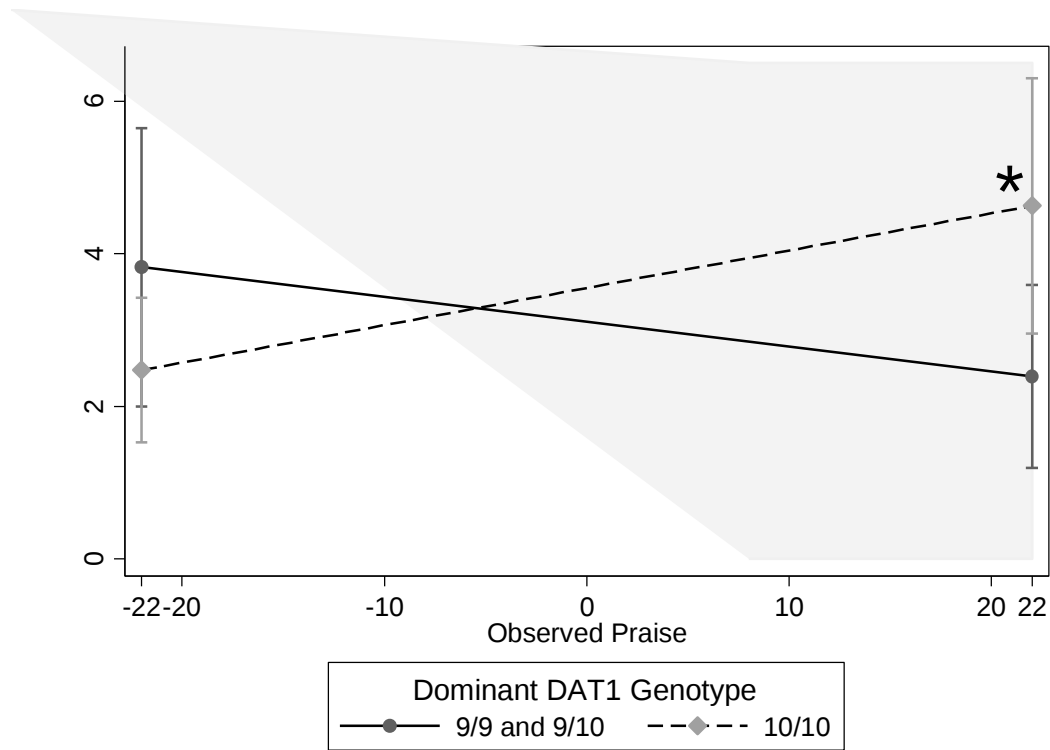
Study 1, Figure 2

Predicted Wave 2 Parent+Teacher Composite of Narcissism from Observed Negative Talk and Additive DAT1 Genotype Interaction



Study 1, Figure 3.

Predicted Wave 2 Parent+Teacher Composite of Narcissism from Observed Praise and Dominant DAT1 Genotype Interaction



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Study 2: Interactive Effects of Parenting with DAT1, DRD4, and DRD2 Genotypes on Young Adult Psychopathic Traits

Abstract

Objective: When accompanied by psychopathic traits, antisocial behavior is particularly persistent and intractable to intervention. Consequently, identifying risk factors for psychopathic will facilitate innovations in interventions for antisocial behavior. Method: Using a population-based sample of 2,488 adolescents (12-21 years) followed prospectively for 13-14 years, we tested the independent association of the 40 base pair (bp) variable number of tandem repeat (VNTR) polymorphism of the dopamine transporter (DAT1) gene, the 48 bp VNTR polymorphism of the dopamine receptor D4 (DRD4) gene, and the Taq1A polymorphism of the dopamine receptor D2 (DRD2) gene with psychopathic traits overall, its individual facets (i.e., callous-unemotional [CU] traits, narcissism), as well as their moderation by negative (i.e., maltreatment) and positive parenting (i.e., closeness, involvement, monitoring) behavior. We also evaluated criteria for differential susceptibility. Results: Parental closeness during adolescence inversely predicted adult psychopathic traits and narcissism specifically; maltreatment histories positively predicted adult psychopathic traits. Parental closeness significantly moderated the association of DAT1 with narcissism, consistent with differential susceptibility. In addition, parental involvement moderated the associations of DRD4 and DRD2 with CU traits; parental monitoring similarly moderated the association of DRD2 and CU traits, but there was no evidence of differential susceptibility. Conclusions: We discuss evidence of gene-environment interplay for psychopathic traits, including partial support for differential susceptibility, as well the putative role of parenting behavior with respect to interventions for youth at-risk for the development of psychopathic traits.

Psychopathic traits, consisting of callous-unemotional (CU) traits (e.g., diminished remorse and empathy), narcissism (e.g., grandiosity, egocentricity), and impulsivity, predict violence and severe antisocial behavior (ASB), including recidivism and adult psychopathy (Frick, Marsee, & Patrick, 2006). To date, studies have prioritized the predictive validity of psychopathic traits, but relatively less is known about their potential risk factors. This is particularly true with respect to narcissism, despite being a central facet of youth and adult psychopathic traits (Cooke & Michie, 2001). Given the clinical and public health significance of psychopathic traits, identifying their risk factors will facilitate the development of efficacious interventions for ASB.

Individual differences in youth psychopathic traits are sensitive to negative parenting. In the Dunedin birth cohort, boys with childhood-onset ASB frequently experienced inadequate parenting (i.e., harsh/inconsistent discipline) and exhibited more psychopathic traits at age 26 relative to boys with adolescent-limited ASB (Moffitt, Caspi, Harrington, & Milne, 2002). Similarly, poor parental monitoring predicted short-term growth in childhood CU traits in a large community sample (Hawes, Dadds, Frost, & Hasking, 2011). However, given these studies did not account for concurrent ASB, it is unclear whether negative parenting behaviors uniquely predicted psychopathic traits. Beyond negative parenting more broadly, maltreatment (i.e., physical abuse, sexual abuse, neglect, and emotional abuse) is specifically associated with psychopathic traits (Cicchetti & Toth, 2005). Violent offenders with antisocial personality disorder and psychopathy reported more childhood severe physical abuse (but not emotional or sexual abuse) relative to violent offenders with antisocial personality disorder only and non-offenders (Kolla et al., 2013), suggesting that maltreatment is uniquely associated with psychopathic traits. However, the continued reliance on aggregate psychopathic traits or

exclusively on CU traits obfuscates which dimensions of psychopathic traits (i.e., CU traits and narcissism) are sensitive to negative parenting generally and maltreatment specifically.

Despite being factorially independent from negative parenting (Borden et al., 2014), only recently has there been commensurate effort to examine if positive parenting behavior (e.g., praise, warmth) is associated with psychopathic traits. Among fifth graders, CU traits decreased more substantively in one year among youth who experienced elevated parental warmth (Pardini, Lochman, & Powell, 2007) and following a parent training intervention consisting of positive reinforcement and behavioral management, parent-reported CU traits decreased for a subset of youth, although reliable predictors of treatment responsiveness were not readily identified (Hawes & Dadds, 2007). This preliminary evidence suggests that interventions promoting positive parenting behavior uniquely predict youth psychopathic traits, although as noted above, it remains unclear if positive parenting is uniquely related to CU traits and narcissism specifically when co-occurring ASB is stringently controlled. The current study addresses this important limitation directly.

Despite its broadsense heritability (Bezdjian, Raine, Baker, & Lynam, 2011; Viding, Blair, Moffitt, & Plomin, 2005), genetic association studies are necessary to discern the underlying pathophysiology of psychopathic traits. Human and non-human animal models converge around the centrality of dopamine (DA) within the structure and connectivity of neural regions involved in the modulation of negative emotion, aggression, and psychopathy (Beiderbeck et al., 2012). Amphetamine-induced DA release was positively associated with psychopathic traits (Buckholtz et al., 2010) and rats bred for low anxiety exhibited elevated aggression and local DA release; moreover, following an infusion of a DA D2 receptor antagonist, their aggression decreased, providing causal evidence of the role of DA within

antisocial-spectrum phenotypes (Beiderbeck et al., 2012). Genes that functionally alter the availability and regulation of DA are thus biologically-plausible candidates in the etiology of psychopathic traits.

The 40 base pair (bp) variable number of tandem repeat (VNTR) polymorphism located in the 3' untranslated region of exon 15 of the DA transporter (DAT1) gene is a plausible variant for psychopathic traits as DAT1 accounted for 11.5% of individual differences in ventral striatum reactivity during a reward-based laboratory task, which predicted self-reported impulsivity (Forbes et al., 2009). The DAT1 10 repeat allele was positively associated with violent ASB (Guo, Roettger, & Shih, 2007) and 10 repeat homozygotes exhibited more rule-breaking behavior relative to carriers of the 9 repeat allele (Burt & Mikolajewski, 2008), although the cross-sectional design prevented inferences about the role of DAT1 in prospective change in ASB. However, there is also evidence implicating the 9 repeat allele with ASB (Gerra et al., 2005). Despite its association with constructs closely related to psychopathy (e.g., impulsivity, severe ASB), prospective longitudinal designs, with rigorous control of ASB, are necessary to consider the association of DAT1 with CU traits and narcissism across development. Improved understanding of the relation of DAT1 with CU traits and narcissism facilitates the development of targeted interventions in subgroups of youth.

The 7 repeat 48 bp VNTR polymorphism in exon 3 of the DA receptor D4 (DRD4) gene is positively associated with impulsivity and novelty-seeking across development, which are central to youth and adult psychopathy (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Laucht, Becker, El-Faddagh, Hohm, & Schmidt, 2005; Reiner & Spangler, 2011). Neurobiological evidence implicates DRD4 with ASB-related phenotypes: in twins, carriers of the 7 repeat allele had significantly lower BOLD signal change in regions associated with

inhibitory control (e.g., right anterior prefrontal cortex) when inhibiting a prepotent response (Mulligan, Kristjansson, Reiersen, Parra, & Anokhin, 2014). Meta-analytic evidence suggests the 7 repeat allele is associated with attention-deficit/hyperactivity disorder (ADHD; Gizer, Ficks, & Waldman, 2009), another key correlate of psychopathy (Fowler et al., 2009). The 7 repeat allele was significantly associated with comorbid ADHD and ASB, providing additional support for shared genetic influences (Holmes et al., 2002). The 7 repeat allele was also positively associated with oppositionality in youth exposed to maternal insensitivity in a sample of preschoolers (Bakermans-Kranenburg & Van Ijzendoorn, 2006), suggesting that the DRD4 7 repeat allele may influence sensitivity to negative parenting. Given its association with constructs strongly correlated psychopathic traits, additional investigation is required to assess whether DRD4 is similarly associated with psychopathic traits.

Third, the DA receptor D2 (DRD2) gene is also thought to influence externalizing phenotypes associated with psychopathic traits (Hicks, Krueger, Iacono, McGue, & Patrick, 2004). The DA D2 receptor antagonist (i.e., haloperidol) successfully reduced ASB (Beiderbeck et al., 2012), and using a cumulative genetic risk approach, a multilocus DA profile score including the DRD2 Taq1A polymorphism (rs1800497), as well as DAT and DRD4, accounted for 10.9% of ventral striatum reactivity in participants' responses during a reward-based card guessing paradigm (Nikolova, Ferrell, Manuck, & Hariri, 2011). Men heterozygous for the DRD2 exhibited more violent delinquency relative to A1 or A2 homozygotes (Guo et al., 2007). A recent meta-analysis of 61 studies concluded that that Taq1A polymorphism was significantly associated with alcohol dependence (Wang, Simen, Arias, Lu, & Zhang, 2013), which shares a common genetic liability with antisocial-spectrum phenotypes (Hicks et al., 2004). Given that

DRD2 is associated with externalizing phenotypes that share a general liability with psychopathic traits, DRD2 is plausible candidate gene for psychopathic traits.

Genetic influences on psychopathology are contingent on environmental experiences (i.e., gene x environment [G x E] interaction), including potential epigenetic processes. For example, nurturing behaviors (i.e., licking/grooming) in rat dams modified gene expression in offspring via DNA methylation (Kappeler & Meaney, 2010). Caregiving behavior is also implicated more directly in offspring DA neurotransmission: rats exposed to early stress (e.g., maternal separation) demonstrated elevated basal DA and reactivity in the nucleus accumbens, an area implicated in reward processing, relative to rates exposed to positive environments (Brake, Zhang, Diorio, Meaney, & Gratton, 2004). Moreover, among young adults, following a psychosocial stressor, individuals who reported less early maternal care exhibited elevated dopamine release in the nucleus accumbens relative to those who experienced early maternal care (Pruessner, Champagne, Meaney, & Dagher, 2004). Therefore, DA genes regulating DA neurotransmission may influence psychopathic traits more readily when biologically plausible environmental conditions, such as parenting, are considered. Differential susceptibility (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007) contends that certain genetic variants (i.e., “plasticity” genes) simultaneously increase susceptibility to negative and positive environments (i.e., accentuates risk and amplifies protection). We know of no study that has evaluated established criteria for differential susceptibility (Belsky et al., 2007; Kochanska, Kim, Barry, & Philibert, 2011) with respect to psychopathic traits. One essential meta-analysis found that individuals with the DAT1 10 repeat allele, DRD4 7 repeat allele, and DRD2 A1 allele were more susceptible to both positive and negative rearing environments in the context of outcomes such as ASB and prosociality (Bakermans-Kranenburg & van Ijzendoorn, 2011). Despite

indications of DA genes affecting responsivity to positive and negative parenting simultaneously, their relation to differential susceptibility with respect to psychopathic traits remains unknown.

Although genotypes implicated in altering DA neurotransmission are plausibly related to the development of psychopathic traits, parenting behaviors (i.e., positive parenting, maltreatment) may moderate these associations. Yet, surprisingly few studies have examined G x E for psychopathic traits, CU traits, and narcissism. Using a longitudinal study of 2,488 adolescents followed for 13-14 years into young adulthood, we hypothesized that positive parenting and maltreatment would be most strongly predictive of later psychopathic traits, CU traits, and narcissism in carriers of the 10 repeat allele of DAT1, the 7 repeat allele of DRD4, and the A1 allele of DRD2. We also predicted that, consistent with differential susceptibility, these same genotypes would simultaneously confer more susceptibility to both positive parenting and maltreatment in predictions of psychopathic traits.

Method

Participants

The National Longitudinal Study of Adolescent Health (Add Health) is an ongoing prospective study of a stratified random sample of adolescents from U.S. high schools (Harris, 2013). At Wave I, 20,745 adolescents (grades 7-12, ages 12-21 years; 49.2% male) were interviewed. Wave II (i.e., approximately two years later) includes 14,738 adolescents (ages 13-22), Wave III (seven to eight years after Wave I) includes 15,197 young adults (ages 18-27), and Wave IV (i.e., 13-14 years after Wave I) includes 15,701 young adults (ages 24-32). Details of the study design can be obtained at <http://www.cpc.unc.edu/projects/addhealth>. Saliva samples were collected from an ethnically diverse subsample of full siblings and twins at Wave III ($n = 2,488$; 74.5% male; 60.9% Caucasian, 8.9% Hispanic, 1% African-American, 6.2% Asian, 0.1%

Native American, and 0.7% “Other”), which the current study utilized. Adolescents with genetic data were somewhat younger at Wave I [$F(1,20,345) = 5.65, p < .001$] relative to adolescents without genetic data, but were comparable with respect to sex [$F(1,20345) = 2.81, p = .09$] and family income [$F(1,20345) = 2.35, p = .13$].

Measures

Genotype. Saliva samples were collected from full siblings or twins to genotype for several candidate polymorphisms. Genomic DNA was isolated from buccal cells using standard methods. First, the 40-bp variable VNTR polymorphism in the 3'untranslated region of exon 15 was genotyped, yielding the two most common alleles for DAT1: the 9 repeat (440 bp) and 10 repeat (480 bp) polymorphisms. DAT1 genotype frequencies were distributed as follows: 9/9 (4.9%), 9/10 (34%), and 10/10 (61.1%). These frequencies were in Hardy-Weinberg equilibrium, $\chi^2(1) = 0.13, p = .71$. Second, the 48 bp VNTR polymorphism in located on chromosome 11p15.5 in exon 3 was genotyped, yielding loci of 2 to 11 repeats. Given that the 4 repeat and 7 repeat polymorphisms are the most common, genotype frequencies of DRD4 in our sample were distributed as follows: 7/7 (4.9%), 7/4 (33.1%), and 4/4 (62%). These frequencies were in Hardy-Weinberg equilibrium, $\chi^2(1) = 0.75, p = .71$. Third, the DRD2 TaqIA (rs1800497) polymorphism located on chromosome 11q22.3 was genotyped, yielding A1 and A2 alleles of the following distribution: A1/A1 (7.9%), A1/A2 (37.6%), A2/A2 (54.6%). These frequencies were in Hardy-Weinberg equilibrium, $\chi^2(1) = 3.84, p = .06$.

Parenting. At Wave I, youth reported their exposure to multiple dimensions of parenting behavior. Responses regarding maternal parenting were prioritized for youth living within two-parent households. Exploratory factor analyses of 27 items (Tung & Lee, in press) uncovered four factors measuring perceived emotional closeness, warmth, and communication (7 items,

e.g., “You are satisfied with the way your mother/father and you communicate with each other,” “Most of the time, your mother/father is warm and loving toward you, “How close do you feel to your mother/father”), involvement in daily activities (4 items, e.g., “In the past four weeks, my mother/father and I have had a talk about a personal problem you were having” and “talked about your school work or grades”), and monitoring and autonomy (7 items, e.g., “Do your parents let you make your own decisions about the time you must be home on weekend nights” and “Do your parents let you make your own decisions about how much television you watch”). Items were measured on a 5-point Likert scale (1 = *not at all*, 5 = *very much*) and summed to form separate totals for perceived parental closeness, involvement, and monitoring. This scale demonstrated predictive validity with offspring outcomes including self-esteem, depression, and juvenile delinquency (Bynum & Kotchick, 2006). The Cronbach alpha for closeness, involvement, and monitoring were .95, .99, and .99, respectively.

Maltreatment. Maltreatment prior to age 18 was retrospectively assessed at Wave IV. Items include: “before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?” “how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?” 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 more than once and positive items were then summed to create a maltreatment score (Haberstick et al., 2005). 49.3% of youth reported no mistreatment history and 50.7% reported at least one episode.

Psychopathic traits, CU traits, and narcissism. Psychopathic traits were collected from respondents at Wave IV and were derived from personality items reflecting the five factor model

(John & Srivastava, 1999), which usefully characterizes dimensions of psychopathy (Lynam et al., 2005). Personality items were previously factor analyzed, yielding 23 items that loaded onto a single scale of total psychopathy (Beaver, Barnes, May & Schwartz, 2011; Cronbach alpha = .80) and was associated with genetic risk and parental negativity in prior studies (Beaver et al., 2011). Items were measured on a 5-point Likert scale (1 = *strongly agree*, 5 = *strongly disagree*) and include: “I feel others’ emotions,” “I lose my temper,” “I live my life without much thought for the future,” and “I sympathize with others’ feelings.” We employed confirmatory factor analysis to discern a CU traits facet from the overall psychopathic traits scale. Results suggested a 4-factor model (results available upon request) provided the best fit (standardized root mean square residual = 0.03, Coefficient of Determination = 0.74). Based on face validity, the first factor appeared to represent emotional regulation (9 items; e.g., “I get angry easily,” “I get stressed out easily,” “I lose my temper”) whereas the second factor reflected the interpersonal/behavioral dimension of psychopathy (7 items; e.g., “I am not really interested in others,” “When making a decision, I go with my “gut feeling” and don’t think much about the consequences of each alternative,” “I live my life without much thought for the future”). The last factor included the following two items: “I talk to a lot of different people at parties” and “I keep in the background;” none of these three factors were utilized in subsequent analyses. The third factor was utilized as a measure of CU traits, which included the following items: “I sympathize with others’ feelings,” “I am not interested in other people’s problems” (reverse-coded), “I feel others’ emotions,” and “I am not really interested in others.” These four items were summed to estimate CU traits (Cronbach alpha = .71). Despite its modest reliability, this scale showed convergent validity with key constructs including Wave IV angry hostility ($B = 0.18$, $SE = 0.01$, $p < 0.001$) and arrest history ($B = 0.04$, $SE = 0.002$, $p < 0.001$).

Narcissism was measured from respondents using the Add Health Narcissism Scale at Wave III (Davis & Brunell, 2012). Consisting of 22 items, including items such as: “I can usually get people to believe me, even when what I am saying isn’t quite true,” “I have leadership abilities,” and “How intelligent are you?” The scale exhibits appropriate predictive validity with risk-taking and aggression, above and beyond the effect of self-esteem, and convergent validity with multiple other established measures of narcissism (Davis & Brunell, 2012). Items were standardized and summed to create a total score. The Cronbach alpha for narcissism was .79.

ASB. Delinquency was assessed at Wave I and included 15 items, such as “how often have you deliberately damaged property that didn’t belong to you in the past 12 months,” “how often did you get into a serious physical fight”, and “how often did you hurt someone badly enough to need bandages or care from a doctor or nurse?” Items were measured on a 4-point scale (0 = *never*, 1 = *1 or 2 times*, 2 = *3 or 4 times*, 3 = *5 or more times*), and were summed to form a total score of delinquency.

Data Analytic Plan

Analyses were performed in Stata using the cross-sectional weight at Wave IV. Hierarchical linear regressions tested the independent association of parenting behaviors and three separate genotypes [DAT1 (i.e., 9/9 vs. 9/10 vs. 10/10), DRD4 (i.e., 7 repeat allele vs. not), and DRD2 (i.e., A1/A1 vs. A1/A2 vs. A2/A2)] with respect to psychopathic traits overall and again in individual predictions of CU traits and narcissism. Parenting variables were centered using the sample mean. Given that total psychopathic traits, CU traits, and narcissism were normally distributed, multiple linear regression was used. Separate models predicted psychopathic traits, CU traits, and narcissism from the interactions of parenting dimensions with

individual genotypes of DAT1, DRD4, and DRD2. We controlled for Wave 1 delinquency, participant sex, and race-ethnicity at Step 1 to improve specificity. We entered main effects for each Wave I positive parenting dimension (i.e., closeness, involvement, monitoring), and the negative parenting dimensions of maltreatment and genotype (i.e., DAT1, DRD4, or DRD2) at Step 2. Finally, we simultaneously entered separate positive parenting x genotype and negative parenting x genotype interactions in Step 3 to test interactive effects, leading to a total of nine models. Non-significant interactions were dropped from the final models.

To evaluate differential susceptibility, we utilized established criteria (Kochanska et al., 2011) by first calculating regions of significance for each significant parenting x genotype interaction in predictions of psychopathic traits, CU traits, and narcissism outcomes. Second, we graphed predicted regression lines using ± 2 SD below and above the mean for parenting behavior variables, and identified regions of significance via shading within figures to indicate below which and above which values of the parenting behavior variable groups of youth with different genotypes significantly differed in regards to the outcomes (Kochanska et al., 2011).

Results

Population Stratification

Given evidence that racial-ethnic differences in allele frequencies (i.e., population stratification) can threaten internal validity, we assessed for the relation of race-ethnicity with key variables. Population stratification requires that race-ethnicity is significantly associated with DAT1, DRD4, and DRD2, as well as parenting behaviors and psychopathic trait outcomes (Hutchison, Stallings, McGeary, & Bryan, 2004). Race-ethnicity was significantly associated with DAT1 ($\chi^2(10) = 62.69, p < .05$), DRD4 ($\chi^2(5) = 102.87, p < .05$), and DRD2 ($\chi^2(10) = 176.71, p < .05$). Race-ethnicity was unrelated to maltreatment ($B = 0.001, SE = 0.02, p = 0.95$),

closeness ($B = 0.002$, $SE = 0.002$, $p = 0.22$), involvement ($B = -0.01$, $SE = 0.01$, $p = 0.11$), and monitoring ($B = 0.01$, $SE = 0.01$, $p = 0.56$). Lastly, race-ethnicity was also unrelated to psychopathic traits ($B = -0.001$, $SE = 0.001$, $p = 0.20$), CU traits ($B = 0.003$, $SE = 0.004$, $p = 0.51$), and narcissism ($B = -0.002$, $SE = 0.003$, $p = 0.45$). Despite the criteria for population stratification not being met, we conservatively controlled for race-ethnicity across models.

DAT1 on Psychopathic Traits, CU Traits, and Narcissism: Moderation by Parenting

First, we evaluated whether Wave I parenting, DAT1 genotype (i.e., 9/9 vs. 9/10 vs. 10/10), and the separate interactions of DAT1 with closeness, maltreatment, involvement, and monitoring simultaneously predicted Wave IV psychopathic traits, controlling for sex, race-ethnicity, and Wave I delinquency. Maltreatment positively predicted psychopathic traits ($B = 1.21$, $SE = .39$, $p < 0.01$), but DAT1 did not [$F(2, 125) = 0.37$, $p = .69$]; closeness, involvement, and monitoring each inversely predicted psychopathic traits ($B = -0.27$, $SE = 0.09$, $p < 0.01$, $B = -0.36$, $SE = 0.17$, $p < 0.05$, and $B = -0.53$, $SE = 0.24$, $p = 0.03$, respectively).

We then reproduced this model predicting Wave IV CU traits. Once again, DAT1 was unrelated to CU traits, $F(2, 125) = 1.03$, $p = .36$ whereas parental involvement and monitoring each inversely predicted CU traits ($B = -0.14$, $SE = 0.05$, $p < 0.01$ and $B = -0.12$, $SE = 0.05$, $p = 0.02$, respectively). However, closeness and maltreatment were each unrelated to CU traits ($B = -0.01$, $SE = 0.02$, $p = 0.57$ and $B = -0.12$, $SE = 0.11$, $p = 0.28$, respectively).

Next, we investigated whether closeness, maltreatment, involvement, and monitoring moderated predictions of Wave III narcissism from DAT1. Closeness inversely predicted narcissism ($B = -0.95$, $SE = .31$, $p < 0.01$), whereas DAT1, maltreatment, involvement, and monitoring were each unrelated to narcissism [$F(2, 108) = .96$, $p = .38$, $B = 0.15$, $SE = 0.44$, $p = 0.74$, $B = 0.16$, $SE = 0.20$, $p = 0.41$, and $B = -0.35$, $SE = 0.26$, $p = 0.17$, respectively]. Closeness

significantly interacted with DAT1 [$F(2, 109) = 8.25, p < .0001$] such that closeness negatively predicted narcissism in the 9/9 ($B = -0.95, SE = .31, p < 0.01$) and 10/10 genotypes ($B = -0.24, SE = .09, p < 0.01$), but not the 9/10 genotype ($B = 0.05, SE = .10, p = 0.59$). Evaluation of regions of significance within ± 2 SD of the closeness mean score are indicated via gray shading in Figure 1, and the presence of both a lower and upper region of significance are consistent with differential susceptibility. That is, individuals with the 9/9 and the 10/10 genotypes simultaneously exhibited elevated Wave III narcissism if they reported lower levels of Wave I closeness and lower levels of Wave III narcissism if they reported higher levels of Wave III narcissism.

DRD4 on Psychopathic Traits, CU Traits, and Narcissism: Moderation by Parenting

Next, we assessed whether Wave I parenting, DRD4 genotype (i.e., 7-repeat allele vs. not), and the separate interactions of DRD4 with closeness, maltreatment, involvement, and monitoring predicted Wave IV psychopathic traits, controlling for sex, race-ethnicity, and Wave I delinquency. There was a main effect of DRD4 ($B = 1.65, SE = .72, p = 0.02$) such that the 7-repeat allele positively predicted psychopathic traits; closeness inversely predicted ($B = -0.21, SE = .10, p = 0.03$) whereas maltreatment positively predicted psychopathic traits ($B = 1.23, SE = .33, p < 0.001$). Involvement and monitoring were each unrelated to psychopathic traits ($B = -0.14, SE = 0.23, p = 0.56$ and $B = -0.18, SE = 0.33, p = 0.57$, respectively). Involvement marginally interacted with DRD4 in the prediction of psychopathic traits, $F(2, 126) = 3.13, p = .08$, whereas monitoring also marginally interacted with DRD4 in the prediction of psychopathic traits, $F(2, 126) = 2.90, p = .09$.

Next, we reproduced this model but evaluated Wave IV CU traits. There was a main effect of DRD4 ($B = 0.34, SE = .14, p = 0.02$) such that the 7-repeat allele positively predicted

CU traits; monitoring inversely predicted CU traits ($B = -0.11$, $SE = .05$, $p = 0.03$), whereas closeness, maltreatment, and involvement each did not ($B = -0.01$, $SE = 0.03$, $p = 0.78$, $B = -0.13$, $SE = 0.11$, $p = 0.21$, and $B = -0.06$, $SE = 0.06$, $p = 0.31$, respectively). The DRD4 x involvement interaction predicted CU traits, $F(2, 126) = 7.21$, $p < .01$ such that involvement negatively predicted CU traits in carriers of the 7 repeat allele ($B = -0.28$, $SE = .07$, $p < 0.001$) but not in those without the 7 repeat allele ($B = -0.06$, $SE = .06$, $p = 0.31$). Regions of significance analyses indicated that genotypes only differed significantly at higher levels of involvement, contraindicating evidence of differential susceptibility (Figure 2).

We then investigated whether closeness, maltreatment, involvement, and monitoring interacted with DRD4 to predict Wave III narcissism. Closeness inversely predicted narcissism ($B = -0.17$, $SE = .07$, $p = 0.01$), whereas DRD4, maltreatment, involvement, and monitoring were each unrelated to narcissism ($B = -0.40$, $SE = 0.72$, $p = 0.58$, $B = 0.10$, $SE = 0.41$, $p = 0.80$, $B = 0.15$, $SE = 0.18$, $p = 0.40$, and $B = -0.34$, $SE = 0.23$, $p = 0.13$, respectively).

DRD2 on Psychopathic Traits, CU Traits, and Narcissism: Moderation by Parenting

Lastly, we reproduced the above models to determine whether DRD2 (i.e., A1/A1 vs. A1/A2 vs. A2/A2) interacted with closeness, maltreatment, involvement, and/or monitoring in the prediction of Wave IV psychopathic traits, controlling for sex, race-ethnicity, and Wave I delinquency. Closeness inversely predicted psychopathic traits ($B = -0.24$, $SE = 0.11$, $p = 0.03$) whereas maltreatment positively predicted psychopathic traits ($B = 1.18$, $SE = 0.34$, $p = 0.001$). DRD2, involvement, and monitoring were each unrelated to psychopathic traits ($F(2, 125) = 1.40$, $p = .25$, $B = 0.51$, $SE = 0.87$, $p = 0.56$, and $B = -0.44$, $SE = 0.25$, $p = 0.08$, respectively). Involvement significantly interacted with DRD2 [$F(2, 126) = 3.39$, $p = .03$] such that involvement negatively predicted psychopathic traits in those with the A2/A2 ($B = -0.71$, $SE =$

.21, $p < 0.01$) genotype, but not A1/A1 or A1/A2 ($B = 0.51$, $SE = .87$, $p = 0.56$ and $B = 0.08$, $SE = .34$, $p = 0.81$, respectively). Given that DRD2 was unrelated to parental involvement (i.e., no regions of significance), differential susceptibility was not indicated.

We then replicated this model with CU traits as the outcome. Closeness positively predicted CU traits ($B = 0.14$, $SE = 0.06$, $p = 0.03$) whereas involvement did so inversely ($B = -0.13$, $SE = 0.05$, $p < 0.01$). DRD2, maltreatment, and monitoring were each unrelated to CU traits ($F(2, 125) = 1.77$, $p = .17$, $B = -0.15$, $SE = 0.11$, $p = 0.20$, and $B = 0.21$, $SE = 0.14$, $p = 0.14$, respectively). Closeness marginally interacted with DRD2 in the prediction of CU traits, $F(2, 126) = 2.86$, $p = .06$) whereas monitoring significantly interacted with DRD2 in the prediction of CU traits, $F(2, 126) = 3.21$, $p = .04$ such that monitoring inversely predicted CU traits in the A1/A2 genotype ($B = -0.30$, $SE = .11$, $p < 0.01$), but not in the A1/A1 or A2/A2 genotypes ($B = 0.21$, $SE = .14$, $p = 0.14$ and $B = -0.03$, $SE = .06$, $p = 0.57$, respectively; Figure 3). Differential susceptibility was not suggested given that genotypes were similar across parental monitoring.

Lastly, we assessed whether closeness, maltreatment, involvement, and monitoring interacted with DRD2 in the prediction of Wave III narcissism. Closeness inversely predicted narcissism ($B = -0.13$, $SE = 0.06$, $p = 0.03$), whereas monitoring positively did ($B = 1.73$, $SE = 0.67$, $p = 0.01$). DRD2, maltreatment, and involvement were unrelated to narcissism ($F(2, 110) = 0.39$, $p = .68$, $B = -0.03$, $SE = 0.38$, $p = 0.95$, and $B = 0.17$, $SE = 0.18$, $p = 0.33$, respectively). Monitoring significantly interacted with DRD2 in the prediction of narcissism, $F(2, 111) = 7.05$, $p < .01$, such that monitoring positively predicted narcissism in the A1/A1 genotype ($B = 1.73$, $SE = 0.67$, $p = 0.01$) but inversely predicted narcissism in the A1/A2 genotype ($B = -1.25$, $SE = 0.43$, $p = 0.01$); monitoring was unrelated to narcissism in the A2/A2 genotype ($B = 0.23$, $SE = 0.43$, $p = 0.01$).

=0.42, $p = 0.59$). Given that genotypes did not differ significantly along the range of monitoring, differential susceptibility was not indicated.

Discussion

Although genetic variation underlying DA neurotransmission and parenting behaviors predict individual differences in psychopathic traits (i.e., CU traits, narcissism), their interactive effects have not been formally evaluated. We tested whether maltreatment prior to age 18 and Wave I positive parenting behavior during adolescence moderated the associations of DAT1, DRD4, and DRD2 with young adult psychopathic traits, CU traits, and narcissism in a large prospective longitudinal sample of adolescents followed for 13-14 years into adulthood. Parental closeness negatively predicted psychopathic traits and narcissism whereas maltreatment consistently positively predicted young adult psychopathic traits. Parental closeness negatively predicted narcissism, both in young adults with the DAT1 9/9 and 10/10 genotypes, consistent with differential susceptibility. Parental involvement negatively predicted CU traits, but only in carriers of the DRD4 7 repeat allele and DRD2 A2/A2 genotypes, inconsistent with differential susceptibility. Lastly, monitoring negatively predicted CU traits and narcissism in youth with the DRD2 A1/A2 genotype, but positively predicted CU traits in youth with the A1/A1 genotype; again, these interactions were not consistent with a differential susceptibility framework. These results provide preliminary support for gene-environment interplay between positive parenting and DA genes in the prediction of young adult psychopathic traits.

Childhood maltreatment significantly predicted adult psychopathic traits, replicating prior evidence that negative rearing environments are central to psychopathic traits (Cicchetti & Toth, 2005; Hawes et al., 2011). Notably, these predictions survived control of concurrent ASB, a longstanding limitation of previous studies (e.g., Hawes et al., 2011). However, it is unclear

whether these relations extend to negative parenting behaviors that are milder in presentation (e.g., parental harshness, inconsistency), which also predict youth ASB across the lifespan (Burnette, Oshri, Lax, Richards, & Ragbeer, 2012). These preliminary data suggest that interventions targeting psychopathic traits should prioritize the reduction of negative parenting behaviors (McDonald, Dodson, Rosenfield, & Jouriles, 2011) and that the mediational processes through which maltreatment increases psychopathic traits require elucidation. For example, undercontrolled childhood personality (i.e., high impulsivity/emotionally reactivity, low conscientiousness) significantly mediated the association of maltreatment subtypes (e.g., neglect, sexual abuse, physical abuse) on adolescent ASB (Oshri, Rogosch, & Cichetti, 2013). Should similar pathways mediate predictions of psychopathic traits, deficits in emotional reactivity and conscientiousness are logical targets for intervention that may reduce psychopathic traits. Furthermore, although dopamine genes did not moderate the association of maltreatment with psychopathic traits, the serotonin transporter (5-HTTLPR), which is implicated in the etiology of psychopathic traits and ASB (Fowler et al., 2009), interacted with maltreatment to predict ASB (Li & Lee, 2010). Future research that evaluates the putative relations of 5-HTTLPR with negative parenting (i.e., maltreatment) might provide a better understanding of the role of maltreatment and other negative parenting dimensions underlying psychopathic traits.

Despite its independence from negative parenting, less is known about the independent association of positive parenting with respect to young adult psychopathic traits. Indeed, this constituted a key focus of the current study. Consistent with Hawes & Dadds (2007), positive parenting appeared to promote resilience. The negative prediction of psychopathic traits and narcissism from closeness suggests the utility of a warm and communicative parent-child relationship (Pardini et al., 2007) whereas the negative prediction of narcissism and CU traits (at

least in youth with a DRD4 7 repeat allele or the DRD2 A2/A2 allele) from monitoring and involvement, respectively, highlights the importance of parental monitoring with at-risk adolescents (Hawes et al., 2011). Due to the design of the current study, we were unable to examine change in either positive parenting or psychopathic traits. Additionally, elucidation of the mediational pathways through which parenting promotes resilience is a promising direction: for example, positive parenting behaviors such as warmth may enhance empathic responsiveness toward others (Kiang, Moreno & Robinson, 2004), which holds significant implications for interventions for psychopathic traits (McDonald et al., 2011; Miller et al., 2014): for example, tailoring treatment to prioritize parents' increased use of positive parenting behaviors before decreasing their use of negative parenting may be more efficacious, although much more research is required in this domain. Lastly, positive parenting dimensions may be differentially effective in promoting adaptive outcomes as a function of youth sex: mothers use more positive parenting with girls than boys and positive parenting may predict decreased ASB in girls more robustly than in boys (Barnette & Scaramella, 2013). Gaining further insight into sex differences in psychopathic traits over time, as well as their relation to positive parenting beyond concurrent ASB, is a key goal for future research.

Recent meta-analytic provides evidence of the interactive effects of DAT1, DRD4, and DRD2 with rearing environment (e.g., parenting behavior, prenatal smoking) on socioemotional outcomes (e.g., prosociality, ASB, ADHD) in the context of differential susceptibility, with youth with the DAT1 10/10 genotype, DRD4 7 repeat allele, and DRD2 A1 allele being the most susceptible to negative and positive environments (Bakermans-Kranenburg & van Ijzendoorn, 2011). However, the current study found partial evidence for differential susceptibility with DAT1 x parental closeness, using previously established criteria (Kochanska et al., 2011).

Results may reflect restricted variance across parenting measures, which can limit inferences about differential susceptibility (Ellis et al., 2011). Specifically, although DRD4 genotype differences were observed at higher levels of parental involvement (i.e., upper region of significance), genotypes did not differ significantly at lower levels of involvement; however, with additional variance, evaluating for regions of significance within ± 2 SD of the mean of involvement would have more powerfully tested differential susceptibility. This further highlights the need to implement multi-method (e.g., observed parenting) and informant measures of parenting, and also provides a rationale for further developing the methods through which differential susceptibility is tested. Next, although results did not unambiguously confirm differential susceptibility, they revealed that DA genetic variants may confer vulnerability to not only ASB, but also to psychopathic traits (Beiderbeck et al., 2012; Burt & Mikolajewski, 2008; Guo et al., 2007). Additional efforts to refine the pathways through which DAT1, DRD4, and DRD2 impact DA pathways and ultimate phenotype expression would improve understanding of causal influences on psychopathic traits. For example, during a reward-based laboratory task, DAT1 and DRD4 accounted for 11.5% and 9% of the variance of individual differences in reactivity within the ventral striatum, respectively, which was significantly associated with impulsivity (Forbes et al., 2009), suggesting that additional inquiry into neurobiological underpinnings of psychopathic traits related to DA neurotransmission may facilitate innovations in the development of interventions for ASB.

Several limitations should be considered. We relied on adolescent-reported parenting, which may be vulnerable to reporting biases in comparison to observed measures of parenting (Zaslow et al., 2006). Moreover, narcissism data were only available at Wave III, and thus interpretations of predictions of narcissism in the current study do not parallel predictions of

young adulthood psychopathic traits and CU traits from Wave IV; instead, narcissism was assessed during emerging adulthood, indicating a need for individuals to address whether these associations remain into young adulthood. Lastly, although CU traits were derived using empirically- and theoretically-informed approaches, the personality items from which CU traits were distilled were not originally designed to assess psychopathy and CU traits.

Overall, we sought to test the interactive associations of Wave I negative and positive parenting with DAT1, DRD4, and DRD2 with Wave IV psychopathic traits and CU traits as well as Wave III narcissism. Using a large longitudinal sample of adolescents followed 13-14 years, positive parenting inversely whereas maltreatment inversely predicted adult psychopathic traits. DAT1, DRD4, and DRD2 all moderated the associations of positive parenting dimensions with psychopathic traits facets, with some evidence of differential susceptibility. The current findings provide a better understanding of the relations of parenting with DA genes in the context of psychopathic traits and its subfactors; if replicated, the findings provide additional insight into current interventions and preventions for youth at-risk for ASB and psychopathic traits.

Study 2, Table 1

Demographic Information for National Longitudinal Study of Adolescent Health (Add Health)

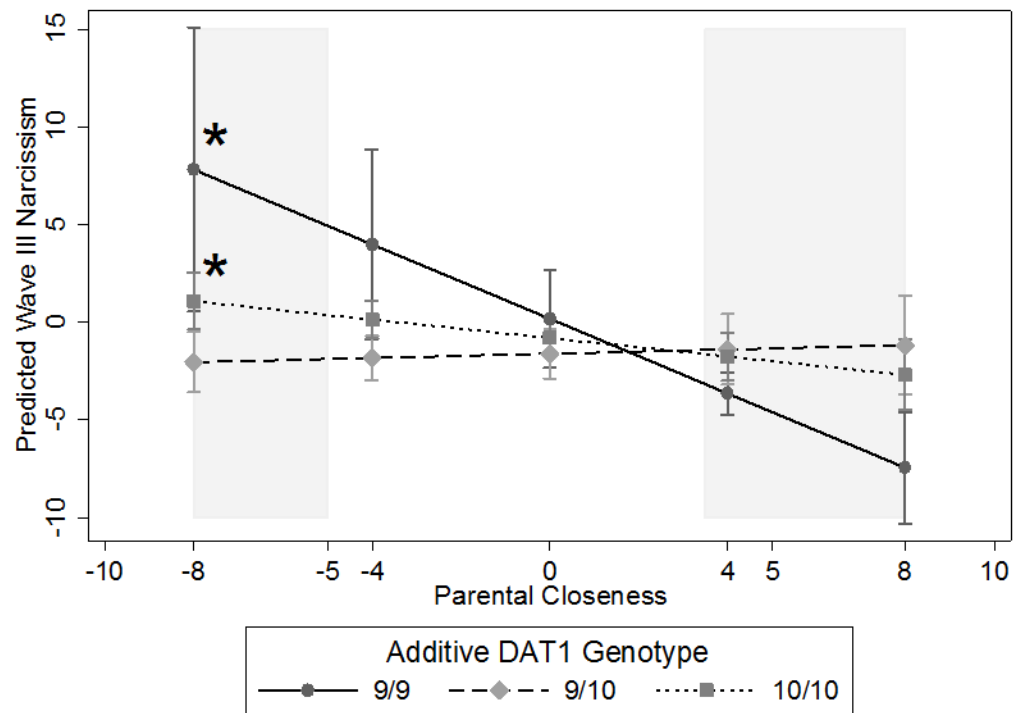
Variable	(<i>n</i> = 2,488)	Range
Wave I Age (<i>SD</i>)	15.99 (0.19)	12 – 21
% Male	49.2	–
% Caucasian	74.5	–
Wave I Delinquency (<i>SD</i>)	4.21 (0.19)	0 – 45
Wave IV Psychopathic Traits (<i>SD</i>)	56.74 (0.58)	23 – 91
Wave IV Callous-Unemotional Traits (<i>SD</i>)	8.50 (0.17)	4 – 20
Wave III Narcissism (<i>SD</i>)	-0.99 (0.41)	-18 – 22
Maltreatment (<i>SD</i>)	0.68 (0.03)	0 – 3
Wave I Closeness (<i>SD</i>)	30.35 (0.18)	10 – 35
Wave I Monitoring (<i>SD</i>)	5.09 (0.09)	0 – 7
Wave I Involvement (<i>SD</i>)	4.00 (0.09)	0 – 10

Note. *SD* = standard deviation; Maltreatment = average maltreatment prior to age 18 score assessed retrospectively at Wave IV.

Study 2, Figure 1

Predicted Wave III Narcissism from Parental Closeness and Additive DAT1 Genotype

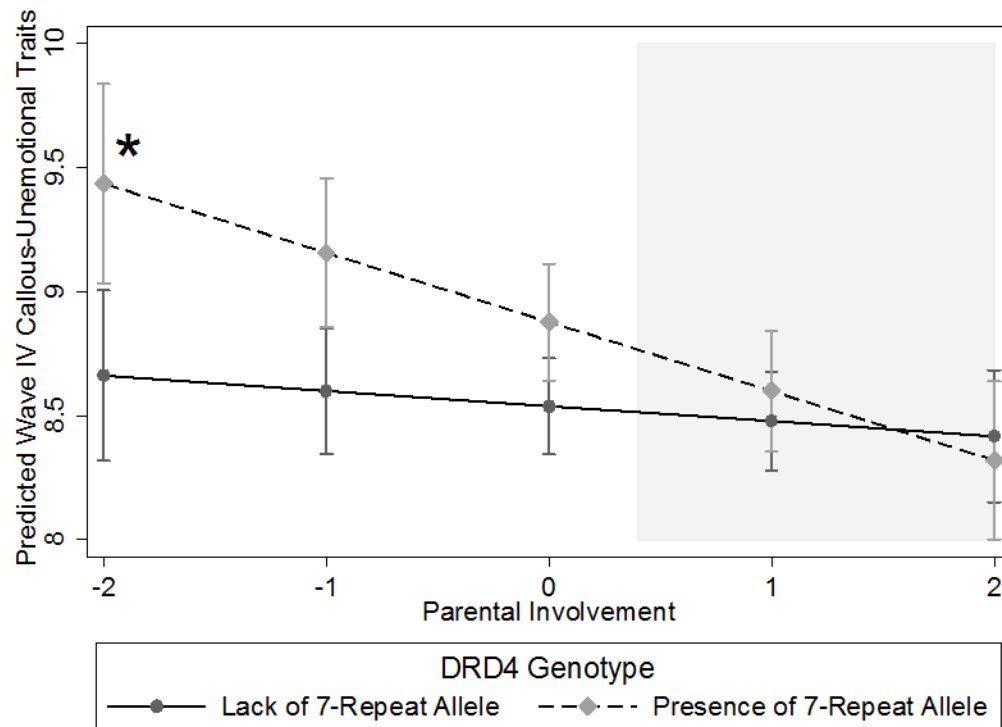
Interaction



Study 2, Figure 2

Predicted Wave IV Callous-Unemotional Traits from Parental Involvement and DRD4 Genotype

Interaction



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Chapter Two

The Association of Temperament and Psychopathic Traits: Mediation by Parenting Behavior

Study 3: Positive and Negative Parenting Behavior Mediates Predictions of Childhood Psychopathic Traits from Prosociality and Daring¹

Abstract

Objective: Although callous-unemotional (CU) traits and narcissism predict negative outcomes (e.g., delinquency, recidivism, adult psychopathy), relatively little is known about their etiology. Given replicated evidence of the inter-correlations of temperament, parenting behavior, and psychopathic traits, we tested whether negative and positive parenting behaviors, collectively and independently, mediated separate predictions of CU traits and narcissism from dimensions of temperament (i.e., prosociality, daring). Method: We collected multi-method measures of positive and negative parenting behavior, temperament, and psychopathic traits in 207 children followed prospectively for two years (i.e., Wave 2). Results: Controlling for concurrent parenting, attention-deficit/hyperactivity disorder and oppositional defiant disorder diagnostic status, and sex, baseline (i.e., Wave 1) positive parenting uniquely mediated the association of prosociality with Wave 2 CU traits whereas Wave 1 observed negative parenting significantly mediated the association of daring with Wave 2 narcissism. Wave 1 observed positive parenting was negatively associated whereas reported negative parenting was positively associated with Wave 2 CU traits; Wave 1 observed negative parenting was positively associated with Wave 2 narcissism. Conclusions: Parenting behavior may partially underlie predictions of psychopathic traits from temperament. We discuss future research directions needed to clarify the contributions of negative and positive parenting on CU traits and narcissism across development.

¹ This manuscript is currently an invited resubmission from *Journal of Psychopathology and Behavioral Assessment*.

Youth antisocial behavior (ASB) is among the most costly mental health problem in North America (Welsh et al., 2008). Consisting of individual differences in callous-unemotional (CU) traits (e.g., diminished remorse and empathy), narcissism (e.g., grandiosity, egocentricity), and impulsivity, psychopathic traits uniquely predict severe and violent ASB (e.g., recidivism, adult psychopathy) across the lifespan (Frick, Marsee, & Patrick, 2006). Despite considerable knowledge about predictions of youth outcome *from* psychopathic traits, less is known about predictors of youth psychopathic traits. Moreover, etiological models have typically prioritized CU traits, despite narcissism being factorially independent from CU traits and uniquely predicting key outcomes (Frick, Bodin, & Barry, 2000; Barry et al., 2007; Ha, Petersen, & Sharp, 2008). Psychopathic traits appear early in development (Kimonis et al., 2006) and therefore represent a promising target for intervention, an important consideration given that adult psychopathy is highly treatment resistant (Lynam, 1997). Given their prediction of outcomes with considerable clinical and public health significance, identifying correlates of psychopathic traits will facilitate innovations in the development and delivery of interventions as well as reduce the burden associated with ASB.

Temperament is central to causal theories of ASB and psychopathic traits. Defined as variation in reactivity (i.e., elevated arousal, motor activity) and self-regulation (i.e., processes regulating attention and inhibition; Rothbart & Derryberry, 1981), temperament dimensions prospectively predict diverse forms of psychopathology including attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD) (Bates, Pettit, Dodge, & Ridge, 1998; Lahey et al., 2008). Youth with ODD and ADHD exhibited elevated negative affect and reduced effortful control (Martel, Gremillion, & Roberts, 2012) and observed fearful inhibition at age 3 inversely predicted psychopathy at age 28 (Glenn, Raine,

Venables, & Mednick, 2007). However, considerable heterogeneity in models of temperament, as well as important differences in its measurement, complicates inferences about its precise role in the development of psychopathic traits. Crucially, the developmental propensity model (Lahey et al., 2008) improve the precision of temperament with respect to psychopathology given its measurement excluded synonyms of symptoms of psychopathology to reduce item overlap. Comprised of prosociality (e.g., prosocial concern for others), daring (e.g., sensation-seeking), and negative emotionality, early prosociality (low), daring (high), and negative emotionality (high) prospectively predicted ODD and CD (Lahey et al., 2008). Moreover, prosociality and daring each predicted a latent antisocial factor derived from diverse indicators of ASB (e.g., official court records, CD symptoms), even with control of initial ASB (Trentacosta, Hyde, Shaw, & Cheong, 2009). In a large representative sample of British youth, prosociality was inversely associated with CU traits, but it was unrelated to ASB; CU traits were significantly associated with ASB and total psychiatric difficulties, substantiating the dissociability of prosociality and CU traits with respect to ASB (Moran et al., 2009). However, this study did not examine daring, thus preventing inferences about the unique associations of prosociality and daring with CU traits. A recent study was the first to replicate the positive association of novelty-seeking as well as an inverse association of cooperativeness with adolescent psychopathy among male offenders with CD (Lennox & Dolan, 2014), indicating a need to further clarify the independent associations of daring and prosociality facets with separate psychopathy facets.

Despite evidence that prosociality and daring predict psychopathic traits, the factors mediating these predictions are poorly understood. Parenting behavior represents one theoretically-plausible intermediate factor underlying predictions of psychopathic traits, given that negative parenting behavior consistently predicts psychopathic traits (Barker, Oliver,

Viding, Salekin, & Maughan, 2011; Frick, Kimonis, Dandreaux, & Farell, 2003; Hawes, Dadds, Frost, & Hasking, 2011). For example, a cross-lagged analysis of 1,562 twins indicated that negative child-directed parental affect (e.g., criticisms, dismissals) significantly predicted growth in psychopathic traits five years later; however, this study did not account for concurrent ASB, limiting inferences regarding the incremental validity of parenting beyond child ASB (Tuvblad, Bezdjian, Raine, & Baker, 2013). Despite its factorial independence from negative parenting (Borden et al., 2014), only recently has the predictive validity of *positive* parenting behavior (e.g., praise, warmth) with respect to psychopathic traits been examined. In a preliminary study, positive parenting inversely predicted psychopathic traits over time, even with control of gender, age, minority status, and family income (Pardini, Lochman, & Powell, 2007). Similarly, maternal care was inversely associated with CU traits, with control of child abuse/neglect, and youth with high CU traits and low maternal care exhibited elevated ASB (Kimonis, Cross, Howard, & Donoghue, 2013). However, far less is known about the association of positive parenting with narcissism, especially with control of co-occurring ASB (Waller, Gardner, & Hyde, 2013); moreover, studies have not employed observed parenting, despite self- and child-report being vulnerable to biases (Zaslow et al., 2006). Although positive and negative parenting behavior are plausible mediators underlying the development of psychopathic traits, studies must employ more stringent models (e.g., ASB) as well as more rigorous parenting measures (e.g., observational).

The plausibility of parenting behavior as a mediator in predictions of psychopathic traits is also strengthened given its association with temperament in the context of child ASB. Elevated child disinhibition and emotionality were associated with harsh parenting, which both predicted ASB five years later (Burnette, Oshri, Lax, Richards, & Ragbeer, 2012). Observed maternal

sensitivity was also more strongly negatively associated with teacher-reported child externalizing behavior in first grade for youth with difficult temperament (i.e., high negative emotionality and reactivity) relative to those with easy temperament (Bradley & Corwyn, 2008). Although temperament and parenting each uniquely predict, as well as transact, to predict psychopathic traits, there are likely to be multiple underlying pathways. Partially reflecting genetic influences, individual differences in temperament elicit non-random responses from the environment (evocative gene-environment correlation; rGE) that contribute to ASB and psychopathic traits. For example, genetically at-risk (based on biological mothers' ASB) adopted children were more frequently exposed to negative parenting (e.g., guilt induction, hostility) from adopted parents from ages 7 through 12 relative to children not genetically at-risk (O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). In particular, meta-analytic evidence indicates substantial effects of child genetic variation and environmental influences on parenting behaviors (e.g., negativity, warmth, control), and additionally theorized the presence of evocative rGE with respect to parenting (Klahr & Burt, 2013). Additional evidence highlighted the role of evocative rGE, beyond passive rGE (i.e., correlated genes of parents and offspring contribute to their correlated behavior), underlying the association of negative parenting and adolescent externalizing problems (Marceau et al., 2013). Thus, the association of temperament with psychopathic traits may reflect diverse patterns with parenting behavior, including evocative interactions and shared causal influences.

Despite replicated evidence of their association with CU traits and narcissism, few studies have tested whether negative and positive parenting behaviors, collectively and independently, mediate separate predictions of CU traits and narcissism from temperament dimensions. We evaluated whether positive and negative parenting behaviors mediated

predictions of psychopathic traits from child temperament (i.e., prosociality, daring) using data from a two-year prospective longitudinal design of 207 5 to 10 year-olds with and without ADHD. We employed rigorous multiple mediation to identify unique effects among multiple mediators, given evidence of its superior statistical properties (e.g., power) relative to traditional mediational methods (Zhao, Lynch, & Chen, 2010); we also stringently controlled for co-occurring ADHD and ODD to ascertain specific associations. We hypothesized that baseline (i.e., Wave 1) positive and negative parenting would each independently mediate the association of Wave 1 temperament dimensions (i.e., prosociality, daring) with psychopathic traits (i.e., CU traits, narcissism) at the two-year follow-up (i.e., Wave 2).

Method

Participants

At baseline (i.e., Wave 1), 230 ethnically diverse (55% Caucasian; 7% African American; 9% Hispanic; 3% Asian; 23% other) five to ten year-old ($M=7.4$, $SD=1.1$) children with and without ADHD were recruited using advertisements in local schools and public locations in addition to referrals from local medical and mental health service providers (Table 1). Inclusion criteria consisted of children living with at least one biological parent at least half time, full-time school enrollment, and English fluency. Exclusion criteria consisted of a Full Scale IQ (FSIQ) < 70, an autism spectrum, seizure, or any neurological disorder. ADHD proband status was based on a fully structured diagnostic interview with the parent. To avoid recruiting a sample of improbably high-functioning youth, non-ADHD comparison children who met diagnostic criteria for any disorder other than ADHD were placed into a group of non-ADHD comparison youth. All participants were recruited, screened, and assessed using identical procedures.

Procedures

Families who contacted the study first completed a telephone screener to determine their eligibility based on the inclusion and exclusion criteria listed above. Rating scales were mailed to families who satisfied these criteria ($n = 230$) and they were subsequently invited to our research laboratory for in-person assessments. Following signed consent and assent procedures for the parent and child, respectively, clinical psychology graduate students or B.A.-level trained staff assessed children using tests of cognitive ability and academic achievement; a second member of the research staff concurrently interviewed parents about their child's psychopathology. All interviewers were initially blind to the child's diagnostic status, but the blind could not always be preserved given the extensive information gathered about the child. Parents were asked to rate each child based on his or her unmedicated behavior. Approximately 85% of children were evaluated without their medication during the in-person assessment (most were treated with stimulant medication). Approximately two years later (i.e., Wave 2), 90% ($n = 207$) of the families returned with their seven to 12 year-old children for a follow-up, which featured parallel procedures to Wave 1. Relevant domains of inquiry included family functioning, youth academic achievement, and child psychopathology. Relative to non-participating families, Wave 2 families reported a higher average number of child ADHD symptoms, $t(226)=-2.08$, $p=.04$; no other significant differences were observed with respect to the child's age and sex as well as parent sex, race-ethnicity, depression, and ADHD. The IRB approved all study procedures.

Measures

Temperament. The Child and Adolescent Dispositions Scale (CADS) is a 48-item parent interview of youth prosociality, daring, and negative emotionality. The CADS was developed for studies of psychopathology by excluding clear synonyms or antonyms of psychiatric symptoms (Lahey et al., 2008). At Wave 1, parents used a 4-point Likert scale to rate 48 items that yielded

three factors: prosociality, daring, and negative emotionality. Given that prosociality and daring are more strongly associated with psychopathic traits than negative emotionality (Lennox & Dolan, 2014; Moran et al., 2009), we excluded the latter from the current study. Previous studies reported high test-retest reliability and external validity (Lahey et al., 2008; Trentacosta et al., 2009). The Cronbach alpha was .87 and .79 for prosociality and daring, respectively.

Self-reported parenting behavior. Parents self-reported 42 parenting behaviors on the Alabama Parenting Questionnaire (APQ) at Wave 1, providing frequencies of parental involvement, positive parenting, inconsistent parenting, poor monitoring, and corporal punishment dimensions (Frick, 1991). The APQ demonstrated convergent validity with observed parenting measures (Hawes & Dadds, 2006; Chronis-Tuscano et al., 2008). However, given that the number of APQ factors varied from three to five (Hawes & Dadds, 2006), we factor analyzed the APQ at Wave 1 and discerned four factors: positive reinforcement, parental involvement, parental monitoring/supervision, and corporal punishment (blinded for review). The current study did not examine parental monitoring/supervision given its specific association with adolescent ASB (Frick, Christian, & Wootton, 1999). The Cronbach's alphas were .78 for positive reinforcement, .78 for parental involvement, and .61 for corporal punishment. To reduce Type I error and account for their intercorrelation ($r = .53, p < .01$), positive reinforcement and parental involvement were standardized and summed to create a composite positive parenting dimension.

Observed parenting behavior. At Wave 1, parents (88% mothers) and children completed a structured parent-child interaction task. Measures of observed parenting were coded using the Dyadic Parent Child Interaction Coding System (DPICS), yielding the following composites: parental negative talk and praise (Eyberg, Nelson, Duke, & Boggs, 2005). Negative

talk was coded in response to parents expressing a verbal criticism or used sarcastic/rude speech toward the child. Praise was coded in response to a labeled or unlabeled positive judgment toward the child. Total counts of parental behaviors were counted and divided by total minutes coded. Six research assistants were intensively trained in the DPICS coding protocols for two months until 70% agreement was reached. Coders received a full day of training followed by weekly coding meetings over 2 months where each coding category was discussed with reviews/quizzes in order to increase reliability and to resolve disagreements. Twenty percent of the videos were randomly selected and coded by two raters to estimate reliability. The DPICS has been established as valid in studies of youth with externalizing behavior and exhibits moderate to high interrater and test–retest reliability (Chronis-Tuscano et al., 2008). The intraclass correlations for our categories were .75 for negative talk and .88 for praise.

Psychopathic traits. At Waves 1 and 2, parents separately completed the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), a 20-item rating scale of youth psychopathic traits in children consisting of three factors: CU traits, narcissism, and impulsivity/conduct problems (Frick et al., 2000). We analyzed the narcissism factor, but excluded impulsivity/conduct problems given its redundancy with ODD. Narcissism was estimated from the sum of seven items: “Emotions seem shallow,” “Braggs excessively,” “Uses or ‘cons’ others,” “Teases or makes fun of others,” “Can be charming,” “Becomes angry when corrected,” and “Seems to think he is more important.” Narcissism was stable in multiple samples, with significant convergence between parent and teacher ratings ($r=.38$, $p<.01$; Frick et al., 2000). More recent confirmation of the three factor model and acceptable psychometric properties were indicated in clinical and community samples (Dong, Wu, & Waldman, 2013). The narcissism scale had a Cronbach alpha of .81 in this sample.

At Wave 2, parents also completed the Inventory of Callous-Unemotional Traits (ICU), a 24 item measure of CU traits rated on a 4-point Likert scale (Frick, 2004). The ICU exhibits high internal consistency (Hawes et al., 2014) as well as appropriate convergent validity with sensation seeking, delinquency, and divergent validity with physiological and self-report measures of emotional reactivity (Kimonis et al., 2008). The Cronbach alpha for the total sum of CU traits was .86.

ADHD and ODD. At Waves 1 and 2, we administered the Diagnostic Interview Schedule for Children, Version IV (DISC-IV) to each child's parent to derive ADHD and ODD diagnostic status (Shaffer et al., 2000). The DISC-IV is a fully structured, extensively-validated and psychometrically sound diagnostic interview. In the DSM-IV Field Trials, test-retest reliability for ADHD from the DISC ranged from .51 to .64 (Lahey et al., 1994).

Data Analytic Plan

We assessed whether Wave 1 multi-method positive and negative parenting behavior mediated the association of Wave 1 temperament (i.e., daring, prosociality) with Wave 2 CU traits and narcissism. We implemented the multiple mediator macro (Preacher & Hayes, 2008) with bootstrapping, a nonparametric re-sampling procedure more statistically powerful than traditional approaches (e.g., Sobel test) (MacKinnon, Krull, & Lockwood, 2000; Zhao et al., 2010). Bootstrapping samples from a dataset k number of times and uses percentages of those distributions to calculate confidence intervals and estimate the indirect effect of mediators. Unlike traditional mediation, recent developments illustrate that mediation does not require a significant direct effect of the predictor on the outcome (MacKinnon et al., 2000; Preacher & Hayes, 2008). Reflecting a "stage sequence" framework (Collins, Graham, & Flaherty, 1998), the predictor first influences the mediator, followed by an effect on the outcome; thus, evaluation

of causal mediation through bootstrapping is not contingent upon a significant direct effect (Zhao et al., 2010). Bootstrapping does not assume normality (Preacher & Hayes, 2008) and thus is robust to skewed data; because the bootstrap distribution is non-symmetrical, it is not equivalent to the traditional t statistic (i.e., betas for the indirect effect cannot be divided by the SE).

Separate models examined whether Wave 1 positive and negative parenting behavior mediated the association Wave 1 *prosociality and daring* with Wave 2 psychopathic traits. Parenting behaviors (i.e., self-reported positive parenting, corporal punishment; observed negative talk, praise) were entered simultaneously to evaluate their collective and independent mediation of separate predictions of CU traits and narcissism from prosociality and daring. Each model separately analyzed prosociality and daring as well as CU traits and narcissism. Next, to improve specificity, sex as well as both Wave 1 ADHD (i.e., ADHD versus non-ADHD control) and ODD diagnostic status were controlled.

Results

Association of Prosociality with CU Traits and Narcissism: Mediation by Parenting Behavior

Observed Parenting. We investigated whether Wave 1 observed positive and negative parenting behavior mediated the association of Wave 1 prosociality with Wave 2 CU traits. Controlling for sex as well as Wave 1 ADHD and ODD diagnostic status, the total effect of prosociality negatively predicted CU traits ($B = -0.74$, $SE = 0.10$, $p < .01$). Prosociality was unrelated to observed praise or negative talk ($B = 0.20$, $SE = 0.19$, $p = .28$ and $B = -0.01$, $SE = 0.13$, $p = .96$, respectively). Observed praise negatively predicted CU traits ($B = -0.12$, $SE = 0.05$, $p = .03$) whereas observed negative talk was unrelated to CU traits ($B = 0.11$, $SE = 0.07$, $p = .14$). There was a significant direct effect of prosociality on CU traits ($B = -0.72$, $SE = 0.10$, p

< .001). We calculated the total and specific indirect effects of prosociality on CU traits through observed praise and negative talk by using 1,000 bootstrap simulation samples, yielding the 95% bias corrected and accelerated confidence intervals for indirect effects. Bootstrapping analyses indicated that neither praise nor negative talk significantly mediated the prediction of Wave 2 CU traits from Wave 1 prosociality.

Next, we similarly examined whether Wave 1 observed praise and negative talk significantly mediated the effect of Wave 1 prosociality on Wave 2 parent-rated narcissism. Controlling for sex, ADHD and ODD diagnostic status, the total effect of prosociality was negatively associated with narcissism ($B = -0.07$, $SE = 0.04$, $p = .03$). Prosociality was unrelated to observed praise or negative talk ($B = 0.22$, $SE = 0.17$, $p = .20$ and $B = -0.07$, $SE = 0.13$, $p = .61$, respectively). Observed negative talk positively predicted narcissism ($B = 0.08$, $SE = 0.02$, $p < .01$), whereas observed praise did not ($B = 0.01$, $SE = 0.02$, $p = .56$). There was a significant direct effect of prosociality on narcissism ($B = -0.07$, $SE = 0.03$, $p = .03$). Bootstrapping analyses indicated that neither praise nor negative talk significantly mediated the association of prosociality and narcissism.

Self-Reported Parenting. We then constructed parallel models to those described above, but this time evaluated *self-reported* positive and negative parenting behavior as a mediator from Wave 1 prosociality to Wave 2 parent-rated CU traits (Table 2). Controlling for sex, ADHD and ODD diagnostic status, the total effect of prosociality was negatively associated with CU traits ($B = -0.61$, $SE = 0.11$, $p < .01$). Prosociality was positively associated with reported positive parenting ($B = 0.13$, $SE = 0.04$, $p < .01$) but was unrelated to corporal punishment ($B = -0.01$, $SE = 0.02$, $p = .69$). Positive parenting marginally and negatively predicted CU traits ($B = -0.40$, $SE = 0.24$, $p = .10$) whereas corporal punishment positively predicted CU traits ($B = 1.49$, $SE =$

0.49, $p < .01$). There was a significant direct effect of prosociality on CU traits ($B = -0.55$, $SE = 0.11$, $p < .01$). Notably, bootstrapping analyses revealed that Wave 1 positive parenting, but not corporal punishment, significantly and uniquely mediated the prediction of Wave 2 CU traits from individual differences in Wave 1 prosociality (Figure 1).

We then examined whether Wave 1 self-reported positive parenting and corporal punishment significantly mediated the effect of Wave 1 prosociality on Wave 2 parent-rated narcissism. Controlling for sex, ADHD and ODD diagnostic status, the total effect of prosociality was negatively associated with narcissism ($B = -0.07$, $SE = 0.03$, $p = .05$). Prosociality was positively associated with positive parenting ($B = 0.10$, $SE = 0.04$, $p = .01$), but unrelated to corporal punishment ($B = -0.02$, $SE = 0.02$, $p = .37$). Positive parenting and corporal punishment were unrelated to narcissism ($B = 0.001$, $SE = 0.07$, $p = .99$ and $B = 0.22$, $SE = 0.14$, $p = .11$, respectively). There was a marginal direct effect of prosociality on narcissism ($B = -0.06$, $SE = 0.03$, $p = .07$). Bootstrapping analyses indicated that neither positive parenting nor corporal punishment significantly mediated the association of prosociality and narcissism.

Association of Daring with CU Traits and Narcissism: Mediation by Parenting Behavior

Observed Parenting. We next investigated whether Wave 1 observed parenting behavior mediated the association of Wave 1 daring with Wave 2 parent-rated CU traits. Controlling for sex, ADHD and ODD diagnostic status, the total effect of daring was unrelated to parent-rated CU traits ($B = -0.08$, $SE = 0.21$, $p = .72$). Daring was marginally and positively associated with observed negative talk ($B = 0.35$, $SE = 0.21$, $p = .10$) but unrelated to observed praise ($B = 0.34$, $SE = 0.31$, $p = .27$). Observed praise negatively predicted CU traits ($B = -0.15$, $SE = 0.06$, $p = .02$) whereas observed negative talk was unrelated to CU traits ($B = 0.12$, $SE = 0.09$, $p = .20$). There was a significant direct effect of daring on CU traits ($B = -0.06$, $SE = 0.20$, $p = .75$).

Bootstrapping analyses indicated that neither praise nor negative talk significantly mediated the association of daring and CU traits.

We then investigated whether Wave 1 observed praise and negative talk significantly mediated the effect of Wave 1 daring on Wave 2 parent-rated narcissism (Table 2). Accounting for sex, ADHD and ODD diagnostic status, the total effect of daring was unrelated to narcissism ($B = 0.60$, $SE = 0.06$, $p = .31$). Daring was marginally and positively associated with observed negative talk ($B = 0.39$, $SE = 0.21$, $p = .07$) and praise ($B = 0.52$, $SE = 0.28$, $p = .07$). Negative talk positively predicted narcissism ($B = 0.08$, $SE = 0.02$, $p < .01$), whereas praise did not ($B = 0.004$, $SE = 0.02$, $p = .80$). There was a non-significant direct effect of daring on narcissism ($B = 0.03$, $SE = 0.06$, $p = .63$). Interestingly, bootstrapping analyses revealed that observed negative talk significantly mediated the association of daring with narcissism (controlling for sex and ADHD and ODD diagnostic status) whereas observed praise did not (Figure 2).

Self-Reported Parenting. We evaluated whether Wave 1 self-reported parenting behavior mediated the effect of Wave 1 daring with Wave 2 parent-rated CU traits. Controlling for sex, ADHD and ODD diagnostic status, the total effect of daring was unrelated to CU traits ($B = 0.07$, $SE = 0.21$, $p = .73$). Daring was unrelated to positive parenting and corporal punishment ($B = 0.04$, $SE = 0.07$, $p = .61$ and $B = 0.04$, $SE = 0.03$, $p = .25$, respectively). Positive parenting negatively predicted CU traits ($B = -0.72$, $SE = 0.25$, $p < .01$) whereas corporal punishment positively predicted CU traits ($B = 1.53$, $SE = 0.54$, $p < .01$). There was no significant direct effect of daring on CU traits ($B = 0.04$, $SE = 0.20$, $p = .85$). Bootstrapping analyses indicated that neither positive parenting nor corporal punishment significantly mediated the association of daring and CU traits.

Lastly, we examined whether Wave 1 positive parenting and corporal punishment significantly mediated the effect of Wave 1 daring on Wave 2 parent-rated narcissism. Controlling for sex, ADHD and ODD diagnostic status, the total effect of daring was positively associated with narcissism ($B = 0.12$, $SE = 0.06$, $p = .03$). Daring was unrelated to positive parenting or corporal punishment ($B = 0.06$, $SE = 0.07$, $p = .36$ and $B = 0.03$, $SE = 0.03$, $p = .39$, respectively). Positive parenting and corporal punishment were unrelated to narcissism ($B = -0.03$, $SE = 0.07$, $p = .62$ and $B = 0.21$, $SE = 0.14$, $p = .13$, respectively). There was a significant direct effect of daring on narcissism ($B = 0.12$, $SE = 0.06$, $p = .04$). Bootstrapping analyses indicated that neither positive parenting nor corporal punishment significantly mediated the association of daring and narcissism.

Discussion

Although dimensions of temperament are central to individual differences in youth CU traits and narcissism, little is known about the explanatory factors underlying these predictions. We evaluated both observed and self-reported positive and negative parenting behavior as mediators of the association of prosociality and daring with respect to CU traits and narcissism, controlling for child sex as well as youth ADHD and ODD diagnostic status. Using a well-characterized sample of 207 youth with and without ADHD followed prospectively for two years, Wave 1 prosociality was positively associated with Wave 1 self-reported positive parenting in models examining predictions of CU traits from prosociality. Next, in predictions of Wave 2 narcissism, Wave 1 daring was marginally positively associated with observed negative parenting. Across models, observed positive parenting was negatively associated whereas reported corporal punishment was positively associated with Wave 2 CU traits; moreover, observed negative talk was positively associated with Wave 2 narcissism. Notably, self-reported

positive parenting uniquely mediated the association of prosociality with CU traits; observed negative parenting independently mediated the association of daring with narcissism, controlling for praise, ADHD and ODD diagnostic status and sex. These findings suggest that positive and negative parenting behaviors contribute to the development of psychopathic traits from dimensions of temperament. We emphasize that this pattern emerged even with stringent control of other parenting behaviors, comorbid ADHD and ODD, and that temperament items did not consist of highly similar language in the psychopathology outcomes they predicted.

To our knowledge, this study is the first of its kind to identify mediators of temperament and youth psychopathic traits. Consistent with hypotheses, negative and positive parenting behaviors mediated cross-time predictions of youth CU traits and narcissism from separate dimensions of temperament. However, given that findings varied across models (e.g., self-reported versus observed parenting), replication and extension is clearly needed. Although these preliminary findings suggest that positive parenting mediated the prediction of CU traits from prosociality, additional constructs between positive parenting and later CU traits must be identified to improve interventions for CU traits. For example, positive parenting (e.g., responsiveness, warmth) facilitates youth prosocial and empathic responding (Kiang, Moreno & Robinson, 2004), which may underlie predictions of CU traits and narcissism from positive parenting. We emphasize, however, multiple additional mediating pathways and processes deserve further inquiry. For example, deviant peer affiliation predicted increased psychopathy from 13 to 24 years (Lynam, Loeber, & Stouthamer-Loeber, 2008) and thus deviant peers may represent yet another pathway from early temperament and subsequent psychopathic traits. Finally, discerning and predicting trajectories of psychopathic traits may improve traction on the phenomenology of psychopathic traits over time. Although trajectories of CU traits have recently

been elucidated (Fontaine, Rijdsdijk, McCrory, & Viding, 2010), these investigations have not yet been extended to narcissism. Given that narcissism uniquely predicts ASB independent of CU traits (Frick et al., 2000; Jezior, McKenzie, & Lee, 2015), prospective change in narcissism must be similarly prioritized in future studies.

The current findings further substantiate bidirectional associations between child temperament and parenting behavior (Burnette et al., 2012), and specifically that child prosociality was associated with positive parenting (controlling for negative parenting) as well as child daring with negative parenting (controlling for positive parenting). Although child temperament and parenting behaviors were collected concurrently at Wave 1, limiting directional inferences, we observed a unique association between child prosociality and positive parenting (and marginally for daring with negative parenting), which is consistent with previous work on reciprocal exchanges between child negative behavior and parenting (Kiff, Lengua, & Zalewski, 2011; Patterson, 1982). Reciprocal relations between temperament and parenting over time in predictions of child adjustment have been previously revealed (e.g., cross-lagged analyses), but similar efforts should be extended to predictions of youth psychopathic traits (Lengua, 2006). The current study's findings also provide further evidence suggesting that difficult youth temperament can elicit negative parenting behaviors just as *positive* youth temperament can elicit positive parenting (Kochanska, Friesenborg, Lange, & Martel, 2004; Wilson & Durbin, 2012). Previous studies have prioritized negative parenting behavior, but dynamic exchanges with positive parenting behavior may also relate to the development of CU traits and narcissism. For example, intervention-induced reductions in CU traits were partially mediated by maternal psychological aggression (i.e., increased maternal warmth; McDonald, Dodson, Rosenfield, &

Jouriles, 2011), highlighting the need to further incorporate positive parenting in the context of psychopathic traits (Hawes, Price, & Dadds, 2014).

The current study revealed that baseline observed praise negatively predicted whereas baseline self-reported negative parenting positively predicted CU traits two years later; similarly, baseline observed negative parenting positively predicted narcissism two years later. This pattern replicates evidence that negative parenting is as a risk factor for psychopathic traits (Tuvblad et al., 2013), although crucially, the current study additionally controlled for concurrent ADHD and ODD. Given the centrality of negative parenting to etiological models of CU traits relative to positive parenting (Waller et al., 2012), these findings highlight a potentially unique role for positive parenting in the development of CU traits (Pardini et al. 2007). Thus, interventions for CU traits may benefit from modules that separately target negative and positive parenting, which is reflected in current evidence-based treatments for oppositional and aggressive youth (Garland, Hawley, Brookman-Frazee, & Hurlburt, 2008). However, typical parent training practices for antisocial youth are less efficacious in treating youth at-risk for the development of psychopathic features (Waschbusch, Carrey, Willoughby, King & Andrade, 2007). These preliminary findings indicate that more intensive parenting training may be necessary in treating youth with emergent CU traits, consistent with recent intervention work (McDonald et al., 2011). However, it remains unclear which components of parenting-focused interventions (e.g., promoting parental warmth, increasing parental consistency) are most efficacious for youth at-risk for ASB and CU traits. Additionally, although positive parenting dimensions were unrelated to narcissism two years later in the current study, recent evidence suggested that elevated levels of parental overvaluation positively predicted childhood narcissism approximately two years later in a community sample (Brummelman et al., 2015). This discrepancy may reflect different constructs underlying positive

parenting overall, although they may share common mediators (e.g., internalized praise may underlie growth in narcissism; Brummelman et al., 2015).

We consider several study limitations: first, temperament and parenting behavior data were collected concurrently, which prevents temporal ordering of predictors and mediators, a requirement for causal mediation (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). However, given the scarcity of mediational models in etiological models of psychopathic traits with temporally-ordered mediators (i.e., parenting) and outcomes (i.e., psychopathic traits), we contend that these preliminary results are valuable and provide testable hypotheses in future studies. Second, although the current study utilized multi-method measures of parenting, temperament and psychopathic traits were limited to parent report. Third, parents can respond in socially desirable ways with respect to their parenting behavior and observed parent-child laboratory tasks, which can constrain the full range of naturally-occurring parenting behaviors (Gardner, 2000). Additionally, parenting in the current study was based largely on mothers. Fourth, the sample primarily consisted of boys, reflecting a longstanding limitation in the field and the urgent need to characterize the phenomenology and etiology of psychopathic traits in girls (Ficks, Dong, & Waldman, 2014). Fifth, the mediational constructs evaluated do not preclude other influences, including gene-environment correlation and interaction that are salient to the development of CU traits, narcissism, and antisocial behavior (Glenn, 2011).

Within the context of multiple mediation with bootstrapping, the current study evaluated parenting behavior as mediators with respect to predictions of CU traits and narcissism from key dimensions of temperament. Using a well-characterized sample of six to nine year-old children with and without ADHD, followed prospectively for two years, both negative and positive parenting emerged as important intermediate phenotypes in the development of psychopathic

traits. Specifically, positive parenting inversely predicted whereas negative parenting positively predicted CU traits two years later, and negative parenting positively predicted narcissism two years later. Moreover, controlling for co-occurring parenting and ASB, self-reported positive parenting mediated predictions of CU traits from prosociality and observed negative parenting mediated predictions of narcissism from daring. We contend that additional research is needed to clarify the contributions of negative as well as positive parenting on CU traits and narcissism, and if replicated, they may constitute targets for future intervention and prevention efforts targeting psychopathic traits and associated antisocial outcomes.

Study 3, Table 1

Means and Percentages for Demographic Variables

Variable	Wave 2 ADHD (<i>n</i> =129)	Wave 2 Non- ADHD (<i>n</i> =80)	<i>F</i> / χ^2	<i>p</i>
Age (<i>SD</i>)	7.26 (1.13)	7.46 (1.12)	1.58	.21
% Males	78.21	64	4.57	.03
% Caucasian	47.50	48.06	.01	.94
% Household income < \$70,000	36.25	37.98	.06	.80
WISC-IV FSIQ (<i>SD</i>)	102.68 (15.93)	109.75 (13.86)	10.52	.001
% Wave 2 ODD diagnosis	32.50	10.85	14.95	.001
% Wave 2 CD diagnosis	3.75	0	2.33	.13

Note. Mean differences evaluated by one-way ANOVA or chi-square. *SD* = standard deviation;

Age = average age at Wave 1 (range: 5–10); WISC-IV FSIQ = average Wechsler Intelligence

Scale for Children, Fourth Edition, Full Scale IQ at Wave 1; ODD = oppositional defiant

disorder; CD = conduct disorder.

Study 3, Table 2

Mediation by Negative and Positive Parenting Behavior on Temperament Dimensions with

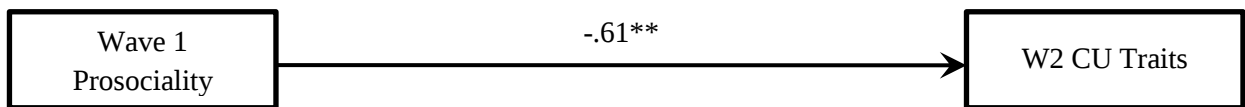
	Point est.	SE	95% BCa bootstrap CI	
			Lower	Upper
Predictions of Callous-Unemotional Traits from Prosociality				
Positive Parenting	-.05	.03	-.12	-.003
Corporal Punishment	-.01	.03	-.09	.05
TOTAL	-.06	.05	-.17	.02
Predictions of Narcissism from Daring				
Observed Praise	.002	.01	-.01	.03
Observed Negative				
Talk	.03	.02	.001	.08
TOTAL	.03	.02	-.001	.09
Callous-Unemotional Traits and Narcissism				

Note. Point est. = point estimate of the indirect effect; SE = standard error; BCa bootstrap CI = Bias corrected and accelerated confidence intervals. ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder. All models controlled for sex, race-ethnicity, and comorbidity. Statistically significant indirect effects bolded.

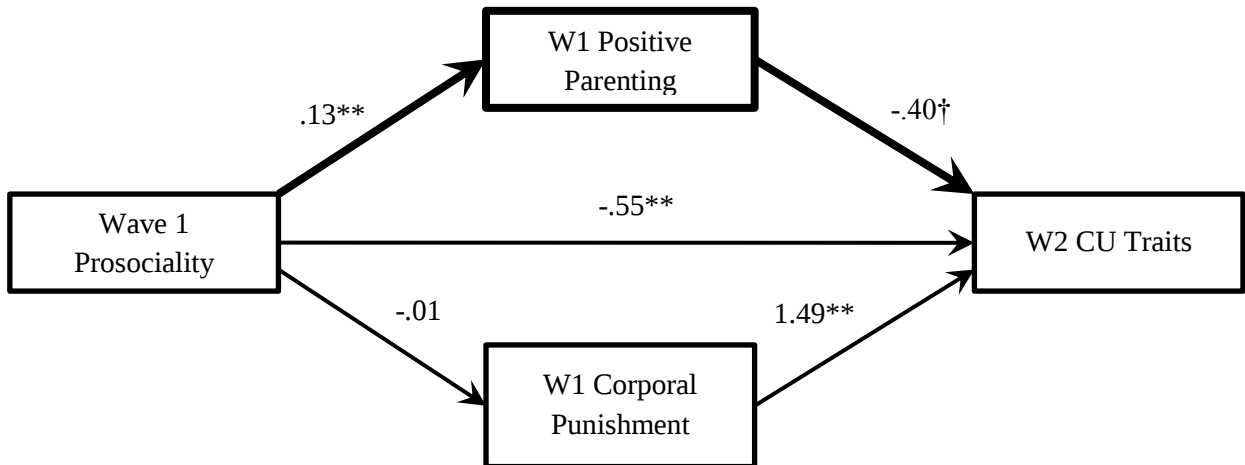
Study 3, Figure 1

Multiple Mediator Model of Wave 2 Callous-Unemotional Traits by Parenting Behaviors and
Prosociality (Beta Coefficients)

A) Total Effect



B) Indirect Effects

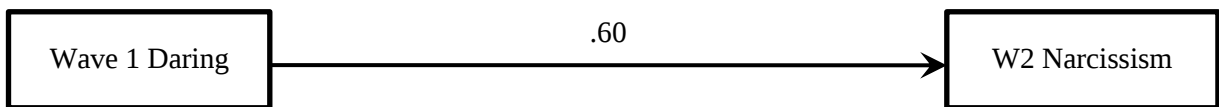


$^{\dagger} p \leq .10$. $* p \leq .05$. $** p \leq .01$.

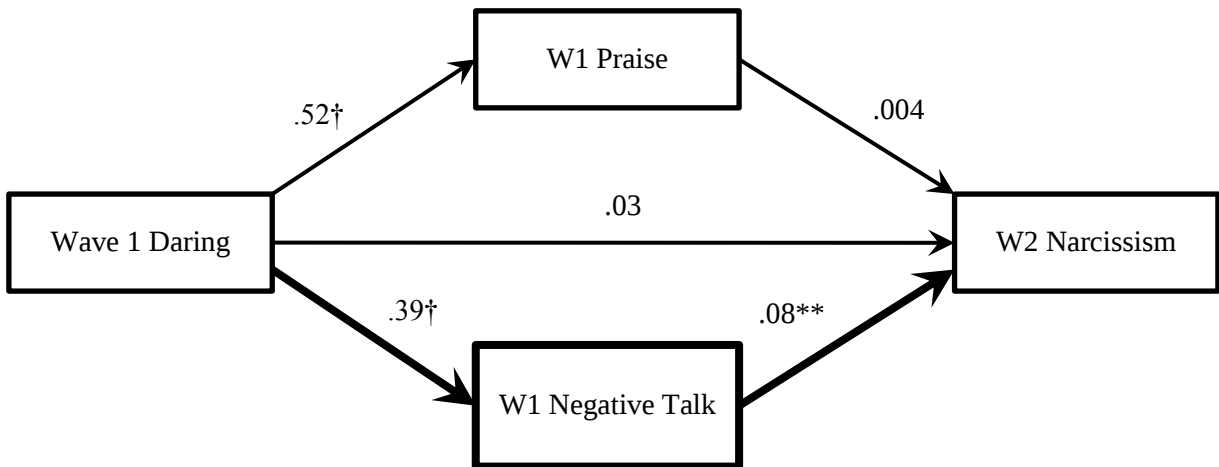
Study 3, Figure 2

Multiple Mediator Model of Wave 2 Narcissism by Parenting Behaviors and Prosociality (Beta Coefficients)

A) Total Effect



B) Indirect Effects



† $p \leq .10$. * $p \leq .05$. ** $p \leq .01$.

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Study 4: Parenting Behavior Mediates Predictions of Adult Psychopathic Traits from Childhood Self-Regulation²

Abstract

Objective: Given their reliable prediction of violence and persistent antisocial behavior, identifying risk factors for psychopathic traits (i.e., callous-unemotional [CU] traits, narcissism) is a priority. By virtue of their prediction of psychopathic traits and interactions with youth temperament (e.g., self-regulation), positive and negative parenting behaviors are plausible mediators of the association of self-regulation and subsequent psychopathic traits. Method: Using a population-based sample of 15,701 adolescents followed for 13-14 years into adulthood, we tested whether childhood maltreatment and positive parenting (i.e., closeness, communication, warmth) during adolescence mediated the associations of childhood self-regulation with adult psychopathic traits, including separate examination of CU traits and narcissism. Results: Using bootstrapping procedures and covarying for concurrent delinquency, childhood maltreatment mediated predictions of adult psychopathic traits (and narcissism specifically) from childhood self-regulation, whereas positive parenting during adolescence additionally mediated predictions of adult narcissism from childhood self-regulation. Neither maltreatment nor positive parenting mediated the association of childhood self-regulation and adult CU traits. Conclusions: We discuss negative and positive parenting behavior as potential mediators underlying predictions of psychopathic traits and narcissism as well as future directions to clarify etiological models of psychopathic traits.

² This manuscript is currently under editorial review.

Individual differences in psychopathic traits reliably predict violence and severe antisocial behavior (ASB) across the lifespan (Frick, Marsee, & Patrick, 2006; Piatigorsky & Hinshaw, 2004). Psychopathic traits consist of callous-unemotional (CU) traits (i.e., deficits in remorse, empathy; shallow emotions) and narcissism (i.e., grandiosity, egocentrism), but it is unclear whether narcissism uniquely predicts ASB (Frick & White, 2008). That is, despite its centrality to adult and youth psychopathy (Cooke & Michie, 2001; Feilhauer & Cima, 2013), studies infrequently test the incremental validity of narcissism beyond CU traits. Notably, although there is considerable knowledge about the predictive validity of psychopathic traits, there is limited knowledge about their determinants, including potential causal influences. Given they predict low treatment engagement/attendance, as well as poor treatment outcomes (O'Neill, Lidz, & Heilbrun, 2003), identifying predictors of psychopathic traits is likely to facilitate innovations in treatment development that are currently underway (e.g., McDonald, Dodson, Rosenfield, & Jouriles, 2011).

Broadly defined as variation in reactivity (i.e., arousal and motor activity) and self-regulation (e.g., regulation of attention, inhibition, self-soothing; Rothbart & Derryberry, 1981; Saltaris, 2002), temperament dimensions prospectively predict oppositional defiant disorder (ODD), conduct disorder (CD), and psychopathic traits, although the mechanisms underlying these predictions are unknown (Glenn, Raine, Venables, & Mednick, 2007; Martel, Gremillion, & Roberts, 2012; Schmeck & Poustka, 2001). Despite its heterogeneity, multiple models of temperament share self regulation as a central component. Defined as modulation of reactivity to external stimuli (Rothbart, Ellis, & Posner, 2004), self-regulation is crucial to psychopathic traits by virtue of spanning multiple systems of attentional, motor, and executive functioning, all of which are independently associated with ASB and psychopathic traits (Rothbart et al., 2004).

Previous research has separately examined facets of self-regulation, including effortful control, behavioral/fearful inhibition, and attention shifting; thus, it is unclear which facets are particularly relevant to emergent ASB and psychopathic traits (Frick & Morris, 2004). For example, poor control, one aspect of self-regulation, prospectively predicted parent- and teacher-reported ASB at ages 9 and 11 (Caspi, Henry, McGee, Moffitt, & Silva, 1995). Fearful inhibition, another dimension of self-regulation, was inversely related to psychopathic traits (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Frick & Morris, 2004; Patrick, 1994): observed fear/inhibition at age 3 was inversely associated with psychopathic personality 25 years later in a community sample (Glenn et al., 2007). Moreover, low effortful control (i.e., inhibition of a prepotent response to instead produce a more prosocial response) significantly predicted two-year growth in adolescent antisocial personality disorder symptoms, although only in neglected youth (Jovev et al., 2013). Although individual facets of self-regulation (e.g., fearful inhibition, effortful control) are associated with ASB and psychopathy, self-regulation overall has yet to be formally evaluated with specific psychopathy facets (i.e., CU traits, narcissism). Moreover, given that self-regulation confers resilience for ASB (Eisenberg, Spinrad, & Eggum, 2011), improved understanding of self-regulation in the context of ASB and psychopathic traits may enhance intervention effects.

Developmental psychopathology prioritizes identification of explanatory mechanisms underlying predictions, which informs the development and delivery of targeted interventions (Rutter & Sroufe, 2000). One theoretically plausible mediator is parenting behavior given its reliable prediction of psychopathic traits: boys with life-course persistent ASB frequently experienced harsh and inconsistent parenting during youth, and subsequently demonstrated elevated psychopathic traits at age 26 (Moffitt, Caspi, Harrington, & Milne, 2002). In a

community sample of 1,000 children, parental monitoring and supervision inversely predicted CU traits after 12 months (Hawes, Dadds, Frost, & Hasking, 2011). Maltreatment, (i.e., physical abuse, sexual abuse, neglect, and emotional abuse) is similarly associated with psychopathic traits (Cicchetti & Toth, 2005), with violent offenders with psychopathy and antisocial personality disorder retrospectively reporting elevated childhood physical abuse (Kolla et al., 2013). However, prior research has typically examined overall psychopathic traits or CU traits specifically, critically limiting knowledge about the association of negative parenting and maltreatment with narcissism. Further, given evidence of their factorial independence, negative and positive parenting (i.e., praise, warmth; Borden et al., 2014) should be dissociated with respect to their potential unique association with psychopathic traits. For example, parental warmth negatively predicted CU traits after a year in a community sample of fifth graders (Pardini, Lochman, & Powell, 2007) and a parent-training intervention (e.g., positive reinforcement, behavioral management) similarly reduced CU traits (Hawes & Dadds, 2007). This preliminary evidence suggests that interventions that facilitate positive parenting behaviors significantly reduced youth psychopathic traits, although their association with narcissism in particular is unknown. Moreover, prior studies inconsistently accounted for concurrent ASB, thus limiting inferences regarding the incremental contribution of negative and positive parenting behaviors to psychopathic traits beyond ASB.

The putative mediating role of parenting behavior is strengthened by evidence that temperament dimensions are correlated with parenting in the context of ASB. Observed maternal sensitivity was more negatively associated with teacher-reported child ASB in first grade for youth with difficult temperament (i.e., high negative emotionality and reactivity) relative to those with easy temperament (Bradley & Corwyn, 2008). A history of neglect predicted growth in

early adolescent antisocial personality disorder symptoms, but only in youth with low effortful control (Jovev et al., 2013). The interactive association of temperament dimensions with parenting behavior reflects diverse processes such as evocative gene-environment correlation (rGE): that is, child genetically-influenced traits, including temperament, elicit non-random environmental responses (e.g., parents, teachers, peers). A recent meta-analysis highlighted the role of child genetic and environmental influences on parenting behavior (e.g., negativity, warmth), further substantiating evocative rGE (Klahr & Burt, 2013). Moreover, the correlation of negative parenting behaviors and adolescent ASB in two samples of Swedish and American families was best accounted for by evocative rGE rather than passive rGE (i.e., correlated genes of parents and offspring contribute to their correlated behavior; Marceau et al., 2013).

To review, individual differences in negative and positive parenting behaviors reliably predict psychopathic traits, and self-regulation similarly is associated with liability in the experience of different parenting behaviors as well as with psychopathic traits. The current study aimed to test negative (i.e., maltreatment) and positive parenting behavior (i.e., closeness) as potential causal pathways from self-regulation to adult psychopathic traits in a stratified random sample of 15,701 adolescents followed for 13-14 years into adulthood. We hypothesized that maltreatment during childhood and Wave II parental closeness would independently mediate the association of Wave I child self-regulation with Wave IV psychopathic traits and its subfacets (i.e., Wave IV CU traits, Wave III narcissism).

Method

Participants

The National Longitudinal Study of Adolescent Health (Add Health) is an ongoing longitudinal study of a stratified random sample of adolescents from U.S. high schools (Harris,

2013). At Wave I, 20,745 adolescents (grades 7-12, ages 12-21 years; 49.2% male) were interviewed. Wave II (i.e., approximately two years later) includes 14,738 adolescents (ages 13-22), Wave III (seven to eight years after Wave I) includes 15,197 young adults (ages 18-28), and Wave IV (i.e., 13-14 years after Wave I) includes 15,701 young adults (ages 24-32). Details of the study design are available at <http://www.cpc.unc.edu/projects/addhealth>. The sample is ethnically diverse (53.4% Caucasian, 17% Hispanic, 21.6% African-American, 6.4% Asian, 0.6% Native American, and 0.9% “Other”).

Measures

Self-regulation. A 23-item measure of self-regulation assessed adolescents’ ability to regulate their attention, feelings, and behavior. Items were answered by respondents (e.g., “do you have trouble paying attention in school?” “difficult problems make you very upset,” and “you never argue with anyone”) and their parents at Wave 1 (e.g., “you can trust your child” and “does your child have a bad temper?”; Beaver, Ratchford, & Ferguson, 2009). A confirmatory factor analysis revealed that all 23 items significantly loaded onto a single factor that has demonstrated adequate reliability as well as convergent validity and discriminant validity with deviant peer affiliation and parenting behavior, respectively (Beaver et al., 2009; Belsky & Beaver, 2011). Items were recoded such that higher values indicated poor self-regulation, standardized, and summed to form a composite score ($\alpha = .79$).

Parental closeness. At Wave II, youth reported their perception of parental closeness, warmth, and communication (7 items; blinded for review). Items include: “You are satisfied with the way your mother/father and you communicate with each other,” “Most of the time, your mother/father is warm and loving toward you, “How close do you feel to your mother/father”; blinded for review). Items were measured on a 5-point Likert scale (1 = *not at all*, 5 = *very*

much) and summed to form a total score of parental closeness. Responses regarding maternal parenting were prioritized for youth living within two-parent households. This scale demonstrated predictive validity with offspring outcomes including self-esteem, depression, and juvenile delinquency (Bynum & Kotchick, 2006). The Cronbach alpha for closeness was .85.

Maltreatment. Maltreatment prior to age 18 was retrospectively assessed at Wave IV. Items include: “before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?” “how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?” 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 more than once and positive items were summed to create a maltreatment score (Haberstick et al., 2005; Li & Lee, 2010). 49.3% of youth reported no mistreatment history and 50.7% reported at least one episode.

Psychopathic traits, CU traits, and narcissism. Psychopathic traits were collected from respondents at Wave IV and derived personality items reflecting the five factor model (Johns & Srivastava, 1999), a valid approach to psychopathy (Lynam et al., 2005). Personality items were factor analyzed, yielding 23 items that reliably loaded onto a reliable scale of total psychopathy (Cronbach alpha ranging from .80-.81; Beaver, Barnes, May & Schwartz, 2011), which was associated with genetic risk and parental negativity in prior studies (Beaver et al., 2011). Items were measured on a 5-point Likert scale (1 = *strongly agree*, 5 = *strongly disagree*) and include: “I feel others’ emotions,” “I lose my temper,” “I live my life without much thought for the future,” and “I sympathize with others’ feelings.” We employed confirmatory factor analysis to discern a CU traits facet from the overall psychopathic traits scale. A 4-factor model (results

available upon request) best fit the data (standardized root mean square residual = 0.03, Coefficient of Determination = 0.74). The first factor reflected emotional regulation (9 items; e.g., “I get angry easily,” “I get stressed out easily,” “I lose my temper”); the second factor reflected the interpersonal/behavioral dimension of psychopathy (7 items; e.g., “I am not really interested in others” and “When making a decision, I go with my “gut feeling” and don’t think much about the consequences of each alternative”); and the last factor only two items (e.g., “I talk to a lot of different people at parties” and “I keep in the background”). Only the third factor from the four-factor model was utilized in subsequent analyses as a measure of CU traits: “I sympathize with others’ feelings,” “I am not interested in other people’s problems” (reverse-coded), “I feel others’ emotions,” and “I am not really interested in others.” These four items were summed to estimate CU traits ($\alpha = .71$). Despite its modest reliability, this scale showed convergent validity with key constructs including Wave IV angry hostility ($B = 0.18$, $SE = 0.01$, $p < 0.001$) and arrest history ($B = 0.04$, $SE = 0.002$, $p < 0.001$).

Narcissism was measured from respondents using the Add Health Narcissism Scale at Wave III (Davis & Brunell, 2012), which consisted of 22 items, including: “I can usually get people to believe me, even when what I am saying isn’t quite true,” “I have leadership abilities,” and “How intelligent are you?” The scale exhibits appropriate predictive validity with risk-taking and aggression, above and beyond the effect of self-esteem, and convergent validity with multiple other established measures of narcissism (Davis & Brunell, 2012). Items were standardized and summed to create a total score ($\alpha = .79$).

ASB. Wave I delinquency included 15 items, such as “how often have you deliberately damaged property that didn’t belong to you in the past 12 months,” “how often did you get into a serious physical fight”, and “how often did you hurt someone badly enough to need bandages or

care from a doctor or nurse?” Items were measured on a 4-point scale (0 = *never*, 1 = *1 or 2 times*, 2 = *3 or 4 times*, 3 = *5 or more times*), and were summed to form a total score of delinquency ($\alpha = .95$). This scale demonstrated appropriate validity with self-control and neuropsychological deficits (Beaver, DeLisi, Vaughn, & Wright, 2008).

Data Analytic Plan

Analyses were performed in Stata using the cross-sectional weight at Wave IV. We assessed whether Wave II positive parenting behavior and maltreatment mediated the association of Wave I self-regulation with Wave IV psychopathic traits and CU traits as well as Wave III narcissism. We utilized structural equation modeling with bootstrapping (using replication weights), a nonparametric re-sampling procedure more statistically powerful than traditional approaches (e.g., Sobel test; MacKinnon, Krull, & Lockwood, 2000; Zhao, Lynch, & Chen, 2010). Bootstrapping samples from a dataset k number of times and uses percentages of those distributions to calculate confidence intervals and estimate the indirect effect of mediators. Unlike traditional mediation, recent developments illustrate that mediation does not require a significant direct effect of the predictor on the outcome (MacKinnon et al., 2000; Preacher & Hayes, 2008). Reflecting a “stage sequence” framework (Collins, Graham, & Flaherty, 1998), the predictor first influences the mediator, followed by an effect on the outcome; thus, evaluation of causal mediation through bootstrapping is not contingent upon a significant direct effect (Zhao et al., 2010). Bootstrapping does not assume normality (Preacher & Hayes, 2008) and thus is robust to skewed data; because the bootstrap distribution is non-symmetrical, it is not equivalent to the traditional t statistic (i.e., betas for the indirect effect cannot be divided by the SE).

Separate models examined whether Wave II positive parenting behavior and maltreatment mediated the association Wave I self-regulation with Wave IV psychopathic traits

and CU traits and Wave III narcissism. Parenting behaviors (i.e., closeness, maltreatment, involvement, monitoring) were entered simultaneously to evaluate their collective and independent mediation of psychopathic traits, CU traits, and narcissism from self-regulation. To improve specificity, sex and Wave I delinquency were controlled across models.

Results

We first evaluated whether Wave II positive parenting behaviors and maltreatment mediated the association of Wave I self-regulation with Wave IV psychopathic traits (Figure 1). Controlling for sex and Wave I delinquency, the total effect of self-regulation positively predicted psychopathic traits ($B = 0.28$, $SE = 0.02$, $p < .001$). Self-regulation negatively predicted closeness ($B = -0.13$, $SE = 0.01$, $p < .001$) but positively predicted maltreatment ($B = 0.01$, $SE = 0.001$, $p < .001$). Next, maltreatment positively predicted psychopathic traits ($B = 1.26$, $SE = 0.17$, $p < .001$); closeness was unrelated to psychopathic traits ($B = -0.03$, $SE = 0.03$, $p = .28$). There was a significant direct effect of self-regulation on psychopathic traits ($B = 0.26$, $SE = 0.02$, $p < .001$). We calculated the total and specific indirect effects of self-regulation on psychopathic traits through parenting by using replication weights to construct 1,000 bootstrap simulation samples, yielding the 95% bias corrected confidence intervals for indirect effects. Bootstrapping analyses indicated that maltreatment, but not closeness, significantly mediated the prediction of Wave IV psychopathic traits from Wave I self-regulation (Table 2).

Second, we similarly examined whether Wave II positive parenting and maltreatment significantly mediated the effect of Wave I self-regulation on Wave IV CU traits (Figure 2). Controlling for sex and Wave I delinquency, the total effect of self-regulation was positively predictive of CU traits ($B = 0.02$, $SE = 0.004$, $p < .001$). Self-regulation again negatively predicted closeness ($B = -0.13$, $SE = 0.01$, $p < .001$) but positively predicted maltreatment ($B =$

0.01, SE = 0.001, $p < .001$). Maltreatment and closeness were both unrelated to CU traits ($B = -0.07$, SE = 0.05, $p = .16$ and $B = -0.01$, SE = 0.01, $p = .16$, respectively). There was a significant direct effect of self-regulation on CU traits ($B = 0.02$, SE = 0.004, $p < .001$). Bootstrapping analyses indicated that neither closeness nor maltreatment significantly mediated the association of self-regulation and CU traits (Table 2).

Lastly, we investigated whether Wave I positive parenting behavior and maltreatment mediated the association of Wave I self-regulation with Wave III narcissism (Figure 3). Controlling for sex and Wave I delinquency, the total effect of self-regulation predicted narcissism ($B = 0.08$, SE = 0.02, $p < .01$). Self-regulation again positively predicted maltreatment ($B = 0.01$, SE = 0.002, $p < .001$) and inversely predicted closeness ($B = -0.12$, SE = 0.01, $p < .001$). Maltreatment positively predicted narcissism ($B = 0.66$, SE = 0.22, $p < .01$); closeness negatively predicted narcissism ($B = -0.11$, SE = 0.04, $p < .01$). There was a significant direct effect of self-regulation on narcissism ($B = 0.06$, SE = 0.02, $p < .05$). Bootstrapping analyses indicated that both closeness and maltreatment significantly mediated the association of self-regulation and narcissism (Table 2).

Discussion

Despite replicated evidence that temperament is associated with psychopathic traits overall and its individual facets of CU traits and narcissism, few studies have tested whether negative and positive parenting mediated these predictive associations. Using a large sample of adolescents followed prospectively into adulthood, we employed multiple mediation with bootstrapping procedures to ascertain whether positive and negative parenting uniquely mediated predictions of psychopathic traits from self-regulation, stringently controlling for concurrent ASB. First, poor self-regulation at Wave I predicted Wave II positive parenting (i.e., closeness,

communication, and warmth) but was positively associated with childhood maltreatment across models; maltreatment positively predicted Wave IV psychopathic traits and Wave III narcissism whereas Wave II positive parenting inversely predicted Wave III narcissism. Second, childhood maltreatment significantly mediated predictions of Wave IV psychopathic traits and Wave III narcissism from Wave I self-regulation, controlling for Wave II positive parenting and Wave I delinquency. Wave II closeness similarly mediated predictions of Wave III narcissism from Wave I self-regulation. However, neither maltreatment nor positive parenting emerged as significant mediators of the associations of self-regulation and Wave IV CU traits. These results provide preliminary evidence of the multiple pathways to psychopathic traits outcomes from self-regulation through parenting behaviors.

To our knowledge, this study is among the first to examine mediators of psychopathic traits, CU traits, and narcissism. Consistent with hypotheses, maltreatment mediated associations of self-regulation with psychopathic traits and narcissism (adjusting for concurrent positive parenting and baseline ASB) whereas positive parenting similarly mediated associations of self-regulation with narcissism (adjusting for concurrent maltreatment and baseline ASB). These findings are strengthened by the fact that predictors, mediators, and outcomes were all temporally ordered, a requirement for causal mediation (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). To identify targets for intervention, the pathways through which maltreatment and positive parenting contribute to psychopathic traits and narcissism from self-regulation require elucidation. For example, negative social cognitive processing styles are thought to mediate predictions of ASB among maltreated youth; should negative social cognitions similarly mediate predictions of psychopathy and narcissism from maltreatment, interventions should remediate these cognitive biases (e.g., hostile attribution bias; Bradshaw & Garbarino, 2004).

Interestingly, contrary to initial hypotheses, negative and positive parenting did not significantly mediate predictions of CU traits from self-regulation. However, different facets of psychopathic traits are etiologically distinct (Larsson, Andershed, & Lichtenstein, 2006; Wang, Baker, Gao, Raine, & Lozano, 2012), further underscoring the need to consider CU traits and narcissism separately. For example, although psychopathy facets were independently sensitive to substantial genetic and non-shared environmental influences, CU traits, but not narcissism, were sensitive to additional unique genetic influences beyond a latent overall psychopathy score (Larsson et al., 2006). Thus, individual differences in CU traits and narcissism may be governed by different mechanisms of risk and protection across development, although this has yet to be examined empirically. We contend that identification shared and unique risk and protective factors for CU traits versus narcissism is critical. For example, maltreatment and positive parenting were more strongly associated with narcissism in the current study; however, other caregiving behaviors (e.g., negative parental affect) may be relatively specific to CU traits (Tuvblad, Bezdjian, Raine, & Baker, 2013). Lastly, given evidence of key sex differences in the presentation of psychopathy (Hicks et al., 2012), sex may be an important moderation of (separable) pathways to CU traits, and narcissism, suggesting etiologically distinct pathways in boys and girls (Wang et al., 2012).

Consistent with evidence that child temperament predicts exposure to later parenting behavior (Bradley & Corwyn, 2008; Kiff, Lengua, & Zalewski, 2011), Wave I poor self-regulation inversely predicted Wave II positive parenting and positively predicted maltreatment history prior to age 18. Given reciprocal exchanges between child temperament and parenting behavior (Burnette et al., 2012), results of the current study do not preclude parent effects that preceded baseline assessments. Although quantitative methods improve traction on reciprocal relations between temperament and parenting (e.g., cross-lagged analyses), these analyses have

yet to be extended in the context of psychopathic traits (Lengua, 2006). Furthermore, self-regulation in the current study is only one facet of temperament whereas others are likely heuristic. The three-factor developmental propensity model (i.e., prosociality, daring, and negative emotionality) was developed excluding language highly synonymous with symptoms of psychopathology (Lahey et al., 2008). Although the self-regulation scale in this study included regulation of cognitions, emotions, and behavior and exhibited convergent validity with parenting behavior and contact with the criminal justice system at earlier ages (Beaver et al., 2009; Belsky & Beaver, 2011), this scale may similarly reflect other individual differences related to psychopathy, including executive functioning and cognitive control (Baskin-Sommers, Brazil, Ryan, Kohlenberg, Neumann, & Newman, 2015; Sadeh & Verona, 2008). Thus, the specificity of effects with respect to self-regulation remains somewhat ambiguous.

The current study substantiates that caregiving behavior is related to psychopathic traits. Specifically, it replicates prior evidence indicating that maltreatment confers risk for adult psychopathic traits and narcissism (Cicchetti & Toth, 2005; Johnson, Cohen, Brown, Smailes, & Bernstein, 1999), and implicates positive parenting behavior during adolescence as a key factor in the development of narcissism, even with control of baseline ASB. Although positive parenting (e.g., praise, warmth) may increase risk for narcissism (Horton, Bleau, & Drwecki, 2006), recent cross-lagged evidence suggests that narcissism may stem from parents' overevaluation of their youth (e.g., interpreting their youth as more special or entitled relative to their peers) rather than parental warmth per se, whereas parental warmth predicts positive self-esteem (Brummelman et al., 2015). Consistent with our findings, positive parenting may then be protective for youth whereas narcissism is more likely to be promoted if parents overvalue their youth (Thomaes & Brummelman, 2015), especially if youth internalize grandiose thoughts

(Brummelman et al., 2015). Moreover, given that maltreatment was a risk factor for psychopathic traits and narcissism, additional research is needed to clarify other dimensions of negative parenting (e.g., harshness, inconsistency) associated with ASB (Burnette et al., 2012) similarly predict psychopathic traits and narcissism. Given that traditional interventions for youth ASB are less efficacious in the presence of psychopathic features (Waschbusch, Carrey, Willoughby, King & Andrade, 2007), innovations in treatment development for this population is sorely needed (McDonald et al., 2011; Miller et al., 2014). These preliminary results indicate that treatments that aim to reduce negative parenting and promote be positive parenting may be efficacious for youth with psychopathic traits and narcissism.

We emphasize several important study limitations. First, the current study relied on youth reported parenting, which may be subject to biases relative to measures of observed parenting behavior (Zaslow et al., 2006). Second, although the CU traits scale was generated using empirically- and theoretically-informed approaches, it was derived from personality data that was not originally intended to assess psychopathy; thus, additional investigation of the validity of this subscale would be informative. Next, the current study tested one of likely several pathways to psychopathic traits outcomes, and additional inquiry is required to clarify gene-environment interplay within the development of ASB and psychopathy (Glenn, 2011). Lastly, psychopathic traits and CU traits outcomes were available only at Wave IV (i.e., ages 24-32) whereas narcissism data was collected only at Wave III (i.e., ages 18-28), thus limiting direct comparisons of CU traits and narcissism in this particular sample.

In sum, we sought to test whether negative and positive parenting mediated predictions of psychopathic traits, CU traits, and narcissism from individual differences in self-regulation using a large longitudinal sample of adolescents followed 13-14 years. With temporally-ordered

predictors, mediators, and outcomes as well as rigorous bootstrapping procedures, childhood maltreatment significantly mediated the prediction of adult psychopathic traits and narcissism from childhood self-regulation. Positive parenting also during adolescence significantly mediated the association of self-regulation and adult narcissism, controlling for baseline ASB. Neither maltreatment nor positive parenting mediated predictions of adult CU traits from childhood self-regulation. The current results provide some clarity with respect to pathways to psychopathic traits, CU traits, and narcissism, and if replicated, provide insight into current interventions for youth at-risk for ASB and psychopathic traits.

Study 4, Table 1

Demographic Information for National Longitudinal Study of Adolescent Health

Variable	(<i>n</i> = 15,701)	Range
Wave I Age (<i>SD</i>)	16.15 (1.74)	12 – 21
% Male	47.2	–
% Caucasian	53.4	–
Wave I Delinquency (<i>SD</i>)	4.32 (5.31)	0 – 45
Maltreatment (<i>SD</i>)	.71 (.82)	0 – 3
Wave II Closeness (<i>SD</i>)	30.02 (4.31)	7 – 35
Wave IV Psychopathic Traits (<i>SD</i>)	56.74 (9.47)	23 – 107
Wave IV Callous-Unemotional Traits (<i>SD</i>)	8.76 (2.42)	4 – 20
Wave III Narcissism (<i>SD</i>)	-0.29 (6.72)	-21 – 30

Note. *SD* = standard deviation; Maltreatment = average maltreatment prior to age 18 score assessed retrospectively at Wave IV.

Study 4, Table 2

Mediation of the Prediction of Wave I Self-Regulation on Psychopathic Traits, Callous-Unemotional Traits, and Narcissism via Wave II Positive Parenting Behavior and Maltreatment

	Point est.	SE	<i>p</i>	95% BC bootstrap CI	
				Lower	Upper
Predictions of Wave IV Psychopathic Traits					
Parental Closeness	.004	.003	.29	-.003	.01
Maltreatment	.01	.002	<.001**	.01	.02
TOTAL	.02	.004	<.001**	.01	.03
Predictions of Wave IV Callous-Unemotional Traits					
Parental Closeness	.002	.001	.14	-.001	.003
Maltreatment	-.001	.001	.18	-.002	.0003
TOTAL	.001	.001	.50	-.002	.003
Predictions of Wave III Narcissism					
Parental Closeness	.01	.01	.01**	.002	.02
Maltreatment	.01	.002	.01**	.002	.01
TOTAL	.02	.01	.001**	.01	.03

Note. Point est. = point estimate of the indirect effect; SE = standard error; BC bootstrap CI = Bias corrected confidence intervals following bootstrapping using replication weights.

Maltreatment = average maltreatment prior to age 18 score assessed retrospectively at Wave IV.

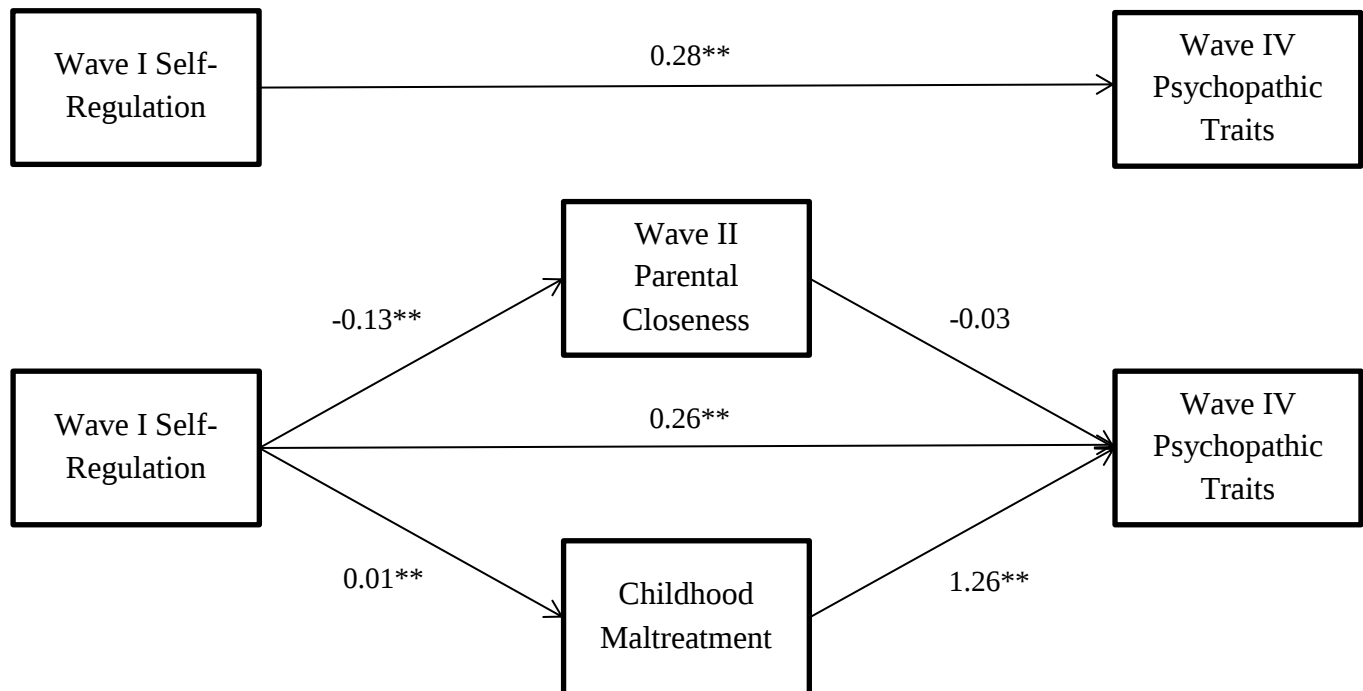
Parental closeness score assessed at Wave II. All models controlled for sex and Wave I

delinquency. * $p \leq .05$. ** $p \leq .01$.

Study 4, Figure 1.

Multiple Mediator Model of Wave IV Psychopathic Traits by Parenting Behaviors and Wave I

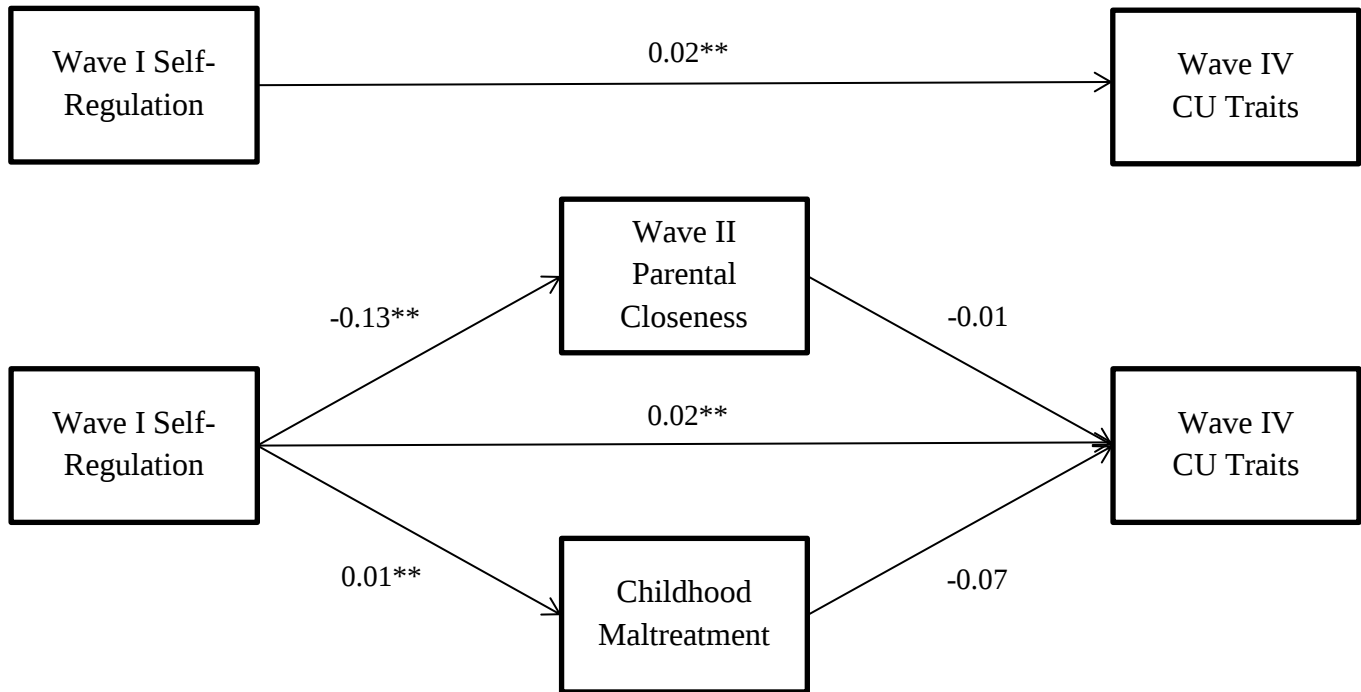
Self-Regulation (Beta Coefficients)



Note. * $p \leq .05$. ** $p \leq .01$. Model controlled for sex and Wave I delinquency.

Study 4, Figure 2.

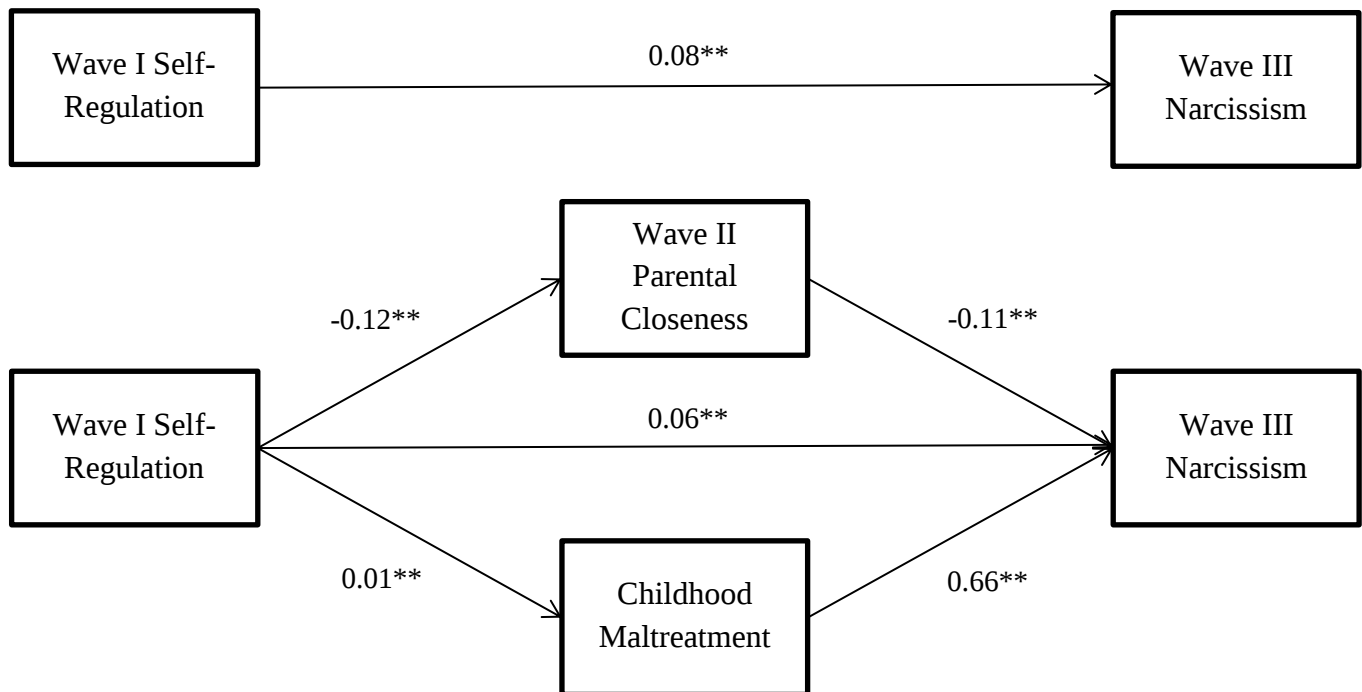
Multiple Mediator Model of Wave IV Callous-Unemotional Traits by Parenting Behaviors and Wave I Self-Regulation (Beta Coefficients)



Note. * $p \leq .05$. ** $p \leq .01$. Model controlled for sex and Wave I delinquency.

Study 4, Figure 3

Multiple Mediator Model of Wave III Narcissism by Parenting Behaviors and Wave I Self-Regulation (Beta Coefficients)



Note. * $p \leq .05$. ** $p \leq .01$. Model controlled for sex and Wave I delinquency.

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Conclusions

Whereas extant evidence has prioritized testing the predictive validity of psychopathic traits, the current dissertation sought to elucidate their risk factors across development. Gaining insight into the etiological models for psychopathic traits facilitates the development of more effective and enduring intervention and prevention efforts in youth at-risk for psychopathic traits and antisocial behavior (ASB). I utilized two complementary prospective longitudinal samples (i.e., ADHD Study, Add Health) that are well-matched to evaluating the current research questions. Whereas the UCLA ADHD Study features multi-method (i.e., objective, self-reported) and multi-informant (i.e., parent, teacher) measures of parenting and ASB, Add Health is a large population-based design. Furthermore, Study 1 captured the relations of candidate genes, parenting, and psychopathic traits in middle to late childhood whereas Study 2 identified these relations from early adolescence into adulthood. To review, using the ADHD Study and Add Health, we first investigated whether parenting behavior moderated the associations of functional polymorphisms regulating dopamine neurotransmission with psychopathic traits, CU traits, and narcissism (i.e., Chapter One). Second, we examined whether parenting behaviors mediated predictions of psychopathic traits, CU traits, and narcissism from individual differences in temperament (i.e., prosociality, daring, self-regulation; i.e., Chapter Two). We highlight the major findings and discuss their key implications in more detail below.

First, across both samples, these results persuasively suggest that negative and positive parenting behavior uniquely predict the development of psychopathic traits. Specifically, in the UCLA ADHD Study, Wave 1 negative parenting positively predicted Wave 2 narcissism and CU traits; similarly, in Add Health, childhood maltreatment positively predicted adult psychopathic traits and specifically narcissism. In addition to replicating evidence of negative parenting as a

risk factor for psychopathic traits (Cicchetti & Toth, 2005; Tuvblad et al., 2013), these results survived careful control of concurrent externalizing behavior. Alternatively, positive parenting behavior inversely predicted Wave 2 CU traits in the ADHD Study and Wave III narcissism in Add Health, implicating its potential role in psychopathic traits separate from negative parenting (Pardini, Lochman, & Powerll, 2007; Thomaes & Brummelman, 2015). If replicated, these results suggest that interventions that effectively reduce negative parenting practices and promote positive parenting practices may reduce the considerable burden associated with psychopathic traits. These findings are noteworthy given that traditional ASB interventions are less efficacious in youth with psychopathic traits (Waschbusch, Carrey, Willoughby, King & Andrade, 2007), highlighting the need for more innovative and targeted treatments than those currently available. Given that our results suggest that intensive parenting interventions may remediate CU traits and narcissism, consistent with recent work (McDonald et al., 2011; Miller et al., 2014), additional research is required to identify which components of parent-focused interventions (e.g., increasing parental warmth and consistency, decreasing parental harshness and corporal punishment) are most efficacious in these at-risk youth.

Second, parenting behaviors moderated the relations of DAT1, DRD2, and DRD4 with later psychopathic traits, CU traits, and narcissism. Although Study 1 investigated DAT1 and DRD4, Study 2 (i.e., Add Health) provided data for DAT1, DRD4, and DRD2 in a large sample better positioned for genetic association studies. First, negative parenting dimensions of negative talk and corporal punishment moderated the relations of DAT1 with narcissism and CU traits, respectively, in Study 1. With respect to positive parenting, positive parenting dimensions (i.e., involvement, monitoring) moderated the associations of DRD4 and DRD2 with adult narcissism and of DRD2 with later CU traits in Study 2. Across both samples, positive parenting

dimensions similarly moderated the association of DAT1 with later narcissism, with control of concurrent ASB; however, in youth with the 10/10 DAT1 genotype, positive parenting positively predicted later narcissism in Study 1 (i.e., ADHD Study), whereas it *negatively* predicted later narcissism in Study 2 (i.e., Add Health). Although the patterns of moderation were not fully parallel across samples, key sample differences may complicate direct comparisons. That is, whereas Study 1 provided more refined measures of parenting (e.g., observed and self-reported parenting), Study 2 utilized adolescent self-reported parenting and associations were tested prospectively into adulthood. Thus, differences in results may be accounted for by methodological differences in assessment methods, statistical power, and/or developmental period. For example, positive parenting dimensions of involvement and monitoring may be more heavily predictive of psychopathic traits in adolescence relative to earlier in childhood, consistent with evidence of relations of parenting and ASB more broadly (Laird, Pettit, Dodge, & Bates, 2003). Future research that seeks to clarify these discrepancies are clearly needed. Moreover, DAT1, DRD4, and DRD2 were also implicated within emerging CU traits and narcissism in Studies 1 and 2, further substantiating evidence of dopaminergic functioning within etiological models of ASB as well as psychopathy (Beiderbeck et al., 2012; Burt & Mikolajewski, 2008; Guo et al., 2007). Although these preliminary results suggest that dimensions of both negative and positive parenting overall moderated the associations of dopaminergic genes with later narcissism and CU traits, additional inquiry is required to clarify the pathways through which individual differences in DAT1, DRD4, and DRD2 impact neurobiological dopamine pathways and their ultimate expression as psychopathic traits (Forbes et al., 2009).

Third, parenting behavior significantly mediated the associations of early temperament dimensions with later psychopathic traits, CU traits, and narcissism. In Studies 3 and 4, negative parenting dimensions significantly mediated predictions of narcissism from facets of temperament (i.e., daring in Study 3; self-regulation in Study 4), controlling for concurrent ASB. Moreover, adjusting for baseline ASB, positive parenting mediated predictions of CU traits from prosociality in Study 3 whereas positive parenting mediated predictions of narcissism from self-regulation in Study 4. We again note that each study captured these relations across different developmental stages (Study 3 from middle to late childhood; Study 4 from early adolescence into adulthood) and utilizing different temperament dimensions; despite these differences, negative parenting mediated the association of childhood temperament with later narcissism in both samples. To our knowledge, these results are unique insofar as identifying putative mediators to youth psychopathic traits. To promote more refined evidence-based interventions for psychopathic traits, the pathways through which negative and positive parenting contribute to psychopathic traits and its subfacets from temperament dimensions require additional attention. For example, whereas negative parenting may lead to negative social cognitive processing styles (e.g., hostile attribution bias; Bradshaw & Garbarino, 2004), positive parenting may lead to more prosocial and empathic responding (Kiang, Moreno & Robinson, 2004); if negative processing biases and/or empathic responding also mediated predictions of psychopathic traits (and particularly narcissism) from negative and positive parenting, interventions should seek to also remediate these intermediate constructs of empathy and negative cognitions.

Overall, given that significant social, public health, and financial costs affiliated with severe and violent ASB that accompanies psychopathic traits (Frick, Marsee, & Patrick, 2006; Welsh et al., 2008), there is a need to gain a better understanding of risk and protective factors of

psychopathic traits, CU traits, and narcissism. Preliminary results across two independent samples of youth and adolescents indicated that both negative and positive parenting practices significantly predicted the development of facets of psychopathy into later childhood and adulthood. Moreover, negative and positive parenting practices significantly moderated the associations of biologically-plausible polymorphisms from dopaminergic genes (i.e., DAT1, DRD4, DRD2) with psychopathic traits, narcissism, and CU traits. Lastly, negative and positive parenting also serve as key intermediate constructs in predictions of psychopathy facets from individual differences in temperament dimensions. Although additional research is needed to further clarify pathways of risk and resilience underlying youth psychopathic traits, these preliminary findings provide important insight into risk factors for psychopathic traits as well as the dynamic processes underlying their development.

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