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Psychopathic Traits Mediate the Association of Serotonin Transporter Genotype and Child Externalizing Behavior

Whitney A. Brammer^{1,*}, Kristen L. Jezior¹, and Steve S. Lee¹

¹Department of Psychology, University of California, Los Angeles, 1285 Franz Hall, Box 951563, Los Angeles, CA 90095-1563, USA.

Abstract

Although the promoter polymorphism of the serotonin transporter (5-HTTLPR) gene is associated with externalizing behavior, its mediating pathways are unknown. Given their sensitivity to serotonin neurotransmission and unique association with attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD), we tested callous-unemotional (CU) traits and narcissism as separate mediators of the association of 5-HTTLPR with ADHD and ODD. We evaluated 209 5–9 year-old children with and without ADHD at baseline; approximately two years later (i.e., Wave 2), parents and teachers separately rated ADHD and ODD symptoms and youth self-reported antisocial behavior. Controlling for race-ethnicity and baseline ADHD/ODD, narcissism uniquely mediated predictions of multi-informant rated Wave 2 ADHD and ODD from variation in 5-HTTLPR; CU traits mediated predictions of Wave 2 ADHD from variations in 5-HTTLPR, but did not mediate the associations of 5-HTTLPR with ODD or youth self-reported antisocial behavior. Specifically, the number of 5-HTTLPR long alleles positively predicted CU traits and narcissism; narcissism was positively associated with Wave 2 ADHD and ODD symptoms, whereas CU traits were positively associated with Wave 2 ADHD. Child sex also moderated indirect effects of CU traits and narcissism, such that narcissism mediated predictions of ADHD/ODD in girls but not boys. Psychopathic traits may represent a relevant pathway underlying predictions of prospective change in ADHD and ODD from 5-HTTLPR, particularly in girls. We consider the role of psychopathic traits as a potential intermediate phenotype in genetically-sensitive studies of child psychopathology.

Keywords

Callous-unemotional traits; narcissism; 5-HTTLPR; ADHD; ODD

Childhood externalizing behavior, including attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD), are common referrals for child mental health services in the U.S. (Achenbach, Dumenci, & Rescorla, 2003; Loeber, Burke, Lahey, Winters, & Zera, 2000). In two nationally representative studies (Nock, Kazdin, Hiripi, & Kessler, 2006; Nock, Kazdin, Hiripi, & Kessler, 2007), 10.2% and

*Correspondence concerning this article should be addressed to Whitney A. Brammer, Department of Psychology, University of California, Los Angeles, 1285 Franz Hall, Box 951563, Los Angeles, CA 90095-1563. wbrammer@ucla.edu.

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9.5% of adults had a lifetime diagnosis of ODD and CD, respectively. Beyond their core features, clinically significant oppositional and conduct problems independently predict substantial impairment (Burke, Waldman, & Lahey, 2010) including family conflict and poor school performance (Burt, Krueger, McGue, & Iacono, 2003; Pardini & Fite, 2010). Similarly, the presence of severe conduct problems predicts elevated risk for injury, substance abuse, depression, incarceration, and death by homicide and suicide (Loeber & Farrington, 1998). Given their considerable clinical and public health significance, improved understanding of the etiology and development of conduct problems is necessary to facilitate innovations in intervention and prevention.

Although conduct problems are better predictors of persistent antisocial behavior than ADHD, children with ADHD are crucial to examining the development of conduct problems given its centrality to early-onset antisocial behavior (Burke et al., 2010). First, ADHD is a potent risk factor for conduct problems, given that ADHD youth are 6.6 and 56.3 times more likely to be diagnosed with ODD in boys and girls, respectively, than non-ADHD youth, even with control of other comorbid conditions (e.g., anxiety, depression; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Next, ADHD and co-occurring oppositionality is more treatment resistant than conduct problems only (Biederman et al., 2008). Moreover, early ADHD with oppositionality may reflect unique genetic influences and explanatory processes (e.g., familial genetic liability, chaotic family environment) relative to oppositionality without ADHD (Caspi et al., 2008; Tuvblad, Zheng, Raine, & Baker, 2009). Given the clinical significance of oppositionality and antisocial behavior more generally in youth with ADHD, and evidence that their co-occurrence may reflect unique causal influences, ADHD is a compelling and appropriate subgroup of youth through which to examine early expressions of conduct problems.

There is meta-analytic evidence that the serotonin transporter promoter polymorphism (5-HTTLPR) is associated with ADHD and antisocial behavior (Ficks & Waldman, 2014; Gizer, Ficks, & Waldman, 2009). Because ADHD features prominently in the development of ODD (Burke et al., 2010) and that the covariation of ADHD with ODD largely reflects shared genetic influences (Caspi et al., 2008), 5-HTTLPR may also be relevant to ODD and conduct problems. However, the association of 5-HTTLPR with respect to conduct problems is inconsistent: whereas long allele homozygotes exhibit the highest aggressive and rule-breaking behavior in preadolescence (Nobile et al., 2007), heterozygotes display the most CD symptoms in community-based adolescents (Malmberg, Wargelius, Lichtenstein, Orelund, & Larsson, 2008); short allele homozygotes exhibit the most violent behavior in a forensic sample of adults (Retz, Retz-Junginger, Supprian, Thome, & Rösler, 2004). 5-HTTLPR may affect conduct problems via disruption of neural structures and circuitry (e.g., amygdala, perigenual cingulate) critical to negative affect and stress reactivity (Pezawas et al., 2005). For example, in a sample of 671 adolescents, short allele homozygotes had significantly more severe ADHD six years later than long allele homozygotes at elevated stress levels, whereas there was no difference at low levels of stress (van der Meer et al., 2014). Although 5-HTTLPR is a biologically plausible biomarker for ADHD and conduct problems, theoretically-derived *mediating* factors underlying 5-HTTLPR with respect to externalizing behavior are largely unknown (Rutter, 2006). Intermediate phenotypes are often conceptualized as putative mediators of genetic influences on psychopathology, but

they are rarely formally evaluated as mediators, especially with respect to genetic influences on attention and conduct problems (see Brammer & Lee, 2013, Li & Lee, 2014, and Martel et al., 2011 for key exceptions).

Consisting of individual differences in narcissism, callous-unemotional (CU) traits, and impulsivity/conduct problems (Frick, Bodin, & Barry, 2000; Vitacco, Rogers, & Neumann, 2003), psychopathic traits are plausible intermediate phenotypes underlying the association of 5-HTTLPR with ADHD and conduct problems given that they are significantly heritable and show strong predictive validity with relevant outcomes (e.g., delinquency, aggression, frequency of police contact; Frick, Marsee, & Patrick, 2006; Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005; Frick & White, 2008; Viding, Blair, Moffitt, & Plomin, 2005). Notably, psychopathic traits appear early in development (as early as age 3), are relatively stable over time, and also predict later aggression, validating their role as a precursor to subsequent ADHD and ODD (Frick, Kimonis, Dandreaux, & Farell, 2003; Kimonis et al., 2006). ADHD moderated the association of ODD with psychopathic traits and aggression in a sample of psychiatrically hospitalized youth, such that the presence of ADHD was marginally associated with elevated CU traits and significantly associated with both reactive and proactive aggression (Becker, Luebke, Fite, Greening, & Stoppelbein, 2013). Controlling for age and initial aggression, childhood ADHD, ODD, and CD, child psychopathic traits uniquely predicted adolescent delinquency severity five years later (Piatigorsky & Hinshaw, 2004). Similarly, in a nationally-representative sample, CU traits predicted growth in conduct problems three years later with control of baseline conduct problems and key covariates (Moran et al., 2009). Unlike CU traits, however, far less is known about the association of narcissism with respect to ADHD and ODD (Frick & White, 2008). That is, despite the centrality of narcissism to conceptual models of adult and youth psychopathy (Cooke & Michie, 2001; Feilhauer & Cima, 2013), studies infrequently address the potential incremental contribution of youth narcissism, beyond CU traits, to critical outcomes. In community- and clinical-based samples, childhood narcissism demonstrated predictive validity even with control of CU traits (Barry et al., 2007; Frick et al., 2000; Ha, Petersen, & Sharp, 2008). Among 85 detained adolescent boys, narcissism was positively associated with proactive aggression, controlling for CU traits, whereas CU traits were unrelated to aggression (Muñoz Centifanti, Kimonis, Frick, & Aucoin, 2013). However, the covariation of narcissism and conduct problems may be inflated due to item overlap (e.g., “Uses or ‘cons’ others” and “Becomes angry when corrected”). Thus, the incremental validity of narcissism with overlapping items excluded, beyond CU traits and other key covariates (e.g., ADHD), with respect to prediction of conduct problems and related antisocial behavior remains largely unknown.

In addition to their reliable association with externalizing behavior, psychopathic traits are sensitive to serotonin neurotransmission. 5-HTTLPR is a valid biomarker for antisocial behavior and psychopathy (Gunter, Vaughn, & Philibert, 2010), although there is less clarity regarding which specific genotype increases susceptibility. Homozygosity of the short allele of 5-HTTLPR was associated with CU traits, even with control of comorbid CD, although the sample consisted entirely of adolescents diagnosed with ADHD five years previously (Fowler et al., 2009). However, in two samples, homozygotes for the 5-HTTLPR long allele from low socioeconomic status backgrounds exhibited the highest rates of CU traits and

narcissism (Sadeh et al., 2010), which is consistent with theoretical work (Glenn, 2011). Crucially, despite their covariation, Sadeh et al. (2010) separately examined the association of 5-HTTLPR with respect to narcissism and CU traits, thus preventing inferences about the unique association of 5-HTTLPR with control of the other dimension. The association of the 5-HTTLPR long allele and psychopathic traits has been replicated in forensic male patients; however, this study separately revealed that the short allele was associated with impulsivity (another facet of psychopathic traits; Sadeh, Javdani, & Verona., 2013). Similarly, healthy adults homozygous for the 5-HTTLPR long allele exhibited less prosocial empathic responding and lower cardiovascular and electrodermal activity when they viewed others in distress than adults homozygous for the 5-HTTLPR short allele (Gyurak et al. 2013), further substantiating that 5-HTTLPR is a plausible biomarker for youth and adult psychopathic traits. Given their sensitivity to 5-HTTLPR, psychopathic traits are strong candidate mediators with respect to the development of ADHD and conduct problems. The current study addresses this issue directly.

Beyond genetic influences, there are reliable sex differences in externalizing behavior (Lahey et al., 2006; Moffitt & Caspi, 2001) and an emergent literature with respect to psychopathic traits (Cale & Lilienfeld, 2002; Viding, Simmonds, Petrides, & Frederickson, 2009). Specifically, girls typically exhibit a later onset of antisocial behavior (i.e., at adolescence) and demonstrate lower psychopathic traits relative to boys, although relational aggression may be an important exception (Lilienfeld & Andrews, 1996; Pechorro et al., 2013; Silverthorn & Frick, 1999). Psychopathic traits may manifest uniquely in boys and girls, especially across development: whereas boys may frequently develop antisocial personality disorder symptoms, girls may develop signs of borderline personality disorder (Sprague, Javdani, Sadeh, Newman, & Verona, 2012). That is, reflecting heterotypic continuity, psychopathic traits in girls may represent a latent construct of broader emotion dysregulation and negative emotionality, consistent with evidence of somatization being more strongly linked to secondary psychopathy in girls than in boys (Lilienfeld & Hess, 2001). Although genetic and environmental influences on psychopathic traits may be comparable in boys and girls (Ficks, Dong, & Waldman, 2014), other putative causal risk factors may be divergent (Krischer & Sevecke, 2008; see Verona & Vitale, 2006 for review). Reduced skin conductance responses were positively associated with the interpersonal facet of psychopathic traits (including narcissism) in boys but not girls during a countdown task (Isen et al., 2010). Similarly, men homozygous for the 5-HTTLPR short allele, but not women, were more likely to administer higher shocks (i.e., aggression) to a confederate under stress (Verona, Joiner, Johnson, & Bender, 2006). Given that hormones (e.g., androgen) are implicated in the density of serotonin transporter receptor sites in the brain (Fink, Sumner, Rosie, Wilson, & McQueen, 1999), sex differences in externalizing behavior and psychopathic traits may be partially related to variation in 5-HTTLPR.

Despite replicated evidence of their association with attention and conduct problems, few studies have tested whether CU traits and narcissism, collectively and independently, constitute empirically distinct pathways from 5-HTTLPR to individual differences in the prospective change of ADHD and ODD over time. A key innovation of the current study is the evaluation of causal mediation given the temporally-ordered predictors, mediators, and outcomes (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). Specifically, candidate

mediators (i.e., CU traits and narcissism) were assessed at baseline (i.e., Wave 1) and multi-informant rated outcomes (i.e., ADHD, ODD, antisocial behavior) were assessed at a two-year prospective follow-up (i.e., Wave 2). Moreover, multiple mediation with bootstrapping, a superior approach to more traditional mediational methods due to enhanced power (Zhao, Lynch, & Chen, 2010), was implemented to identify unique effects among multiple mediators. Using a well-characterized sample of school-age children with ($n = 80$) and without ADHD ($n = 129$), we examined whether individual differences in Wave 1 CU traits and narcissism uniquely and collectively mediated the association of 5-HTTLPR with Wave 2 parent- and teacher-rated ADHD and ODD symptoms, as well as youth self-reported antisocial behavior. We also tested whether child sex moderated these mediational models. Models conservatively controlled for Wave 2 ODD in predictions of Wave 2 ADHD, Wave 2 ADHD in predictions of Wave 2 ODD, and both Wave 2 ADHD and ODD in predictions of Wave 2 youth self-reported antisocial behavior. We predicted that Wave 1 narcissism and CU traits would each uniquely mediate the association of 5-HTTLPR with respect to Wave 2 ADHD, ODD, and youth-reported antisocial behavior, such that the long allele of 5-HTTLPR would positively predict Wave 1 CU traits and narcissism, and that sex would moderate these indirect effects (i.e., conditional indirect effects). Due to a nascent literature on sex differences in psychopathic traits, directional hypotheses were not made with respect to conditional indirect effects. Likewise, we predicted that CU traits and narcissism would positively predict Wave 2 ADHD and ODD symptoms and antisocial behavior.

Method

Participants

At baseline (i.e., Wave 1), 230 ethnically diverse (i.e., parents identified as 55% Caucasian; 7% African American; 9% Hispanic; 3% Asian; 23% mixed/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 < 70 , an autism spectrum, seizure, or any neurological disorder. ADHD proband status was based on a fully structured diagnostic interview with the parent. At Wave 1, 52.4% of youth initially met diagnostic criteria for ADHD, whereas 28.4% of youth met diagnostic criteria for ODD. We contend that a sample enriched for ADHD (and comorbid ODD) is appropriate for the study of psychopathic traits given the co-occurrence of ADHD and psychopathic traits (Fowler et al., 2009). 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

At baseline, 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 informed consent and assent procedures for the parent and child, respectively, clinical psychology graduate students or B.A.-level trained staff assessed children's cognitive ability and academic achievement; a second research staff member 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. Approximately 85% of children were evaluated without their medication during the in-person assessment (most regularly-medicated youth were treated with stimulant medication). Parents were asked to rate each child based on his or her unmedicated behavior.

Approximately two years later (i.e., Wave 2), 91% ($n = 209$) of the families returned with their seven to 12 year-old children for a follow-up assessment, 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 Institutional Review Board approved all study procedures. A total of 174 families had complete data in the analyses below.

Measures

Genotype—DNA was extracted from saliva using DNA Genotek Oragene Self-Collection Kits (DNA Genotek, Inc., Ottawa, CA). The 48-base pair (bp) insertion/deletion polymorphism in the promoter region of 5-HTTLPR was genotyped using standard primers, which produced fragments of either 484 or 528 bp (Heils et al., 1996). The long variant (528 bp) has approximately three times basal activity than the shorter promoter (484 bp) (Lesch et al., 1996). Because the precise mode of transmission for 5-HTTLPR is unknown (i.e., dominant, recessive, heterosis), we compared the following three genotypes: long/long (32.2%, $n = 56$), short/long (43.1%, $n = 75$), and short/short (24.7%, $n = 43$). These frequencies did not significantly deviate from Hardy-Weinberg equilibrium ($\chi^2 = 3.1$, $df = 1$, $p > .05$).

Psychopathic traits—At Wave 1, parents completed the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), a 20-item rating scale of psychopathic traits in children consisting of three factors: CU traits, narcissism, and impulsivity/conduct problems (Frick et al., 2000). CU traits and narcissism were stable in multiple samples, with significant convergence between parent and teacher ratings ($r = .38$, $p < .01$; Frick et al., 2000). We analyzed the CU traits and narcissism factors, but excluded impulsivity/conduct problems given its redundancy with ODD and antisocial behavior. The CU traits factor consisted of the sum of six items: "Is concerned about how well he/she does at school/work" (reverse scored; [R]), "Is good at keeping promises" (R), "Feels bad or guilty when he/she does something wrong" (R), "Is concerned about the feelings of others" (R), "Does not show feelings or emotions", and "Keeps the same friends" (R). Narcissism was estimated from the sum of these five items: "Emotions seem shallow," "Braggs excessively," "Teases or makes

fun of others,” “Can be charming,” and “Seems to think he is more important.” To minimize shared source variance between similar narcissism and ODD items, the following items (i.e., “Uses or ‘cons’ others” and “Becomes angry when corrected”) were excluded. However, results were identical when the seven original narcissism items were used. CU traits and narcissism scales had Cronbach alphas of .59 and .76, respectively. The level of CU traits and narcissism in the current study were comparable to community samples and samples of youth at moderate risk for antisocial behavior (Allen, Briskman, Humayun, Dadds, & Scott, 2013; Frick et al., 2000)

ADHD and ODD—At Wave 2, we administered the ADHD and ODD modules from the Diagnostic Interview Schedule for Children, Version IV (DISC-IV) to each child’s parent (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 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). Given that the predictive validity of dimensionally-rated ADHD and ODD was superior to diagnostic designations (Fergusson & Horwood, 1995), we used the total number of parent-reported Wave 2 ADHD and ODD symptoms as separate dependent variables.

At Wave 2, parents and teachers separately completed the Disruptive Behavior Disorder (DBD) Rating Scale (Pelham, Gnagy, Greenslade, & Milich, 1992). All 45 items, keyed to DSM-IV ADHD and ODD, were rated on a five-point scale from “not at all” to “very much.” Items rated as “pretty much” or “very much” were scored as a symptom (Lahey et al., 1994). The DBD has strong predictive validity (Lahey et al., 2004). Given its validity (Lahey et al., 1998; Piacentini, Cohen, & Cohen, 1992; Shemmashian & Lee, 2015), we combined parent and teacher ratings of ADHD and ODD using the ‘or rule’ (i.e., symptom is present if endorsed by either the parent or teacher) to generate a composite measure of ADHD and ODD symptoms from both parent and teachers (parent+teacher or P+T). This strategy is a valid method of assessing youth ADHD and disruptive behavior disorders and also conservatively reduces the number of statistical tests (Lahey et al., 1998). Cronbach alphas were .96 for parent+teacher ADHD and .80 for parent+teacher ODD in this study.

Self-reported antisocial behavior—At Wave 2, we administered the Self-Report of Antisocial Behavior Scale (SRA), an interview assessment of youth self-reported antisocial behavior in the previous 6 months (Loeber, Stouthamer-Loeber, Van Kammen, & Farrington, 1989). Youth were asked to report whether they participated in the behavior “never,” “once,” “twice,” or “more often” in the past six months, yielding a dimensional measure of antisocial behavior severity. This scale demonstrated strong concurrent and predictive validity in community and clinical samples (Hinshaw, Simmel, & Heller, 1995; Loeber et al., 1989). The Cronbach alpha was .79 for antisocial behavior severity in this sample.

Data Analytic Procedures

To review, we assessed whether individual differences in Wave 1 CU traits and narcissism mediated the association of 5-HTTLPR with the number of parent- and teacher-rated Wave 2 ADHD and ODD symptoms, as well as youth-reported antisocial behavior. We note that the key constructs are temporally ordered given that genotype “precedes” Wave 1 psychopathic

trait mediators and Wave 2 outcomes, thus allowing for conceptualizations of causal mediation (Kraemer et al., 2001). To enhance specificity of mediated effects, we implemented stringent covariates: (1) Wave 2 ADHD diagnostic status (i.e., DISC-IV ADHD versus non-ADHD control) in predictions of Wave 2 ODD; (2) Wave 2 ODD diagnostic status in predictions of Wave 2 ADHD; and (3) Wave 2 ADHD and ODD status in predictions of youth-reported antisocial behavior. Race-ethnicity and family income were also controlled. We used a multiple mediator macro with bootstrapping (i.e., a nonparametric re-sampling procedure that repeatedly samples from a dataset k number of times and empirically estimates a sampling distribution of the indirect effect of mediators) that is superior to traditional mediation (e.g., Sobel test; Hayes, 2013; Preacher & Hayes, 2008; Zhao et al., 2010). Unlike traditional mediation, recent developments illustrate that mediation does not require a significant association between the predictor and outcome. Instead, mediation follows a “stage sequence” framework (Collins, Graham, & Flaherty, 1998), in which the predictor initially affects the mediator, which then affects the outcome. Accordingly, a significant direct effect of the predictor on the outcome is not required to discern mediation (Zhao et al., 2010).

5-HTTLPR was coded 0, 1, and 2 for the long/long, short/long and short/short genotypes, respectively. CU traits and narcissism were entered simultaneously as mediators whereas the outcomes separately consisted of the number of Wave 2 parent-rated ADHD and ODD symptoms from the DISC-IV. This exact model was then reproduced with Wave 2 P+T rated ADHD and ODD using the ‘or rule’ from the DBD Rating Scale and then again with youth-reported antisocial behavior from the SRA. Crucially, bootstrapping is robust to non-normal data (Preacher & Hayes, 2008), a key consideration with psychopathology data (e.g., symptom counts). We also assessed whether child sex moderated each mediation model; when conditional indirect effects were significant, we present confidence intervals (CI) for an index of moderated mediation and posthoc differences of indirect effects in both boys and girls (Hayes, 2014). Below, we first present the total effect of 5-HTTLPR on ADHD and ODD (excluding CU traits and narcissism), followed by the independent association of CU traits and narcissism with respect to Wave 2 ADHD and ODD symptoms, as well as youth self-reported antisocial behavior. Second, we report the direct effect of 5-HTTLPR on ADHD, ODD, and antisocial behavior (i.e., the effect of 5-HTTLPR including mediators). We conclude by reporting the bootstrap-derived indirect effects of 5-HTTLPR with outcomes through each mediator as well as results of moderated mediation by child sex.

Results

Descriptive Information

Descriptive data for the current sample are summarized in Table 1. We examined whether key demographic variables significantly differed according to Wave 2 ADHD diagnostic status. As expected, youth diagnosed with ADHD via the DISC-IV at Wave 2 were significantly more likely to be male ($\chi^2 = 4.57, p = .03$) and be diagnosed with Wave 2 ODD relative to youth without ADHD ($\chi^2 = 14.95, p < .01$). Accordingly, we covaried for Wave 2 ADHD diagnostic status in models predicting Wave 2 ODD symptoms and for Wave 2 ODD diagnostic status in models predicting Wave 2 ADHD symptoms.

Population Stratification

Given racial-ethnic differences in allele frequencies of 5-HTTLPR (Gelernter, Cubells, Kidd, Pakstis, & Kidd, 1999), population stratification is a potential threat to the internal validity of genetic association studies. Population stratification requires that race-ethnicity be significantly associated with 5-HTTLPR as well as with outcomes of ADHD and ODD (Hutchison, Stallings, McGeary, & Bryan, 2004). Although race-ethnicity was significantly related to 5-HTTLPR ($X^2 = 29.33$, $df = 10$, $p < .01$), it was unrelated to Wave 2 parent-reported ADHD diagnostic status ($X^2 = 3.22$, $df = 6$, $p = .78$), ODD diagnostic status ($X^2 = 9.30$, $df = 6$, $p = .16$), the number of parent-rated ADHD ($B = -0.10$, $SE = 0.53$, $p = .16$) and ODD symptoms ($B = -0.12$, $SE = 0.18$, $p = .10$), the number of parent + teacher-rated ADHD ($B = -0.07$, $SE = 0.58$, $p = .36$) and ODD symptoms ($B = -0.08$, $SE = 0.17$, $p = .28$), and youth self-reported antisocial behavior ($B = -0.01$, $SE = 0.19$, $p = .90$). Although population stratification was unlikely to confound the current study, we nevertheless controlled for race-ethnicity in all mediational models.

5-HTTLPR and Wave 2 ADHD: Mediation by Wave 1 CU Traits and Narcissism

We began by testing the association of 5-HTTLPR and parent-rated Wave 2 ADHD symptoms through Wave 1 CU traits and narcissism, controlling for Wave 2 ODD, race-ethnicity, and family income (Figure 1). There was no significant total effect of 5-HTTLPR (i.e., excluding the mediators from the model) on Wave 2 ADHD symptoms ($B = -0.54$, $SE = 0.55$, $p = .33$), but the number of 5-HTTLPR long alleles was positively associated with CU traits ($B = -0.45$, $SE = 0.20$, $p = .02$) and narcissism ($B = -0.47$, $SE = 0.22$, $p = .03$). Second, CU traits significantly ($B = 0.65$, $SE = 0.22$, $p < .01$) and narcissism marginally positively predicted the total number of Wave 2 ADHD symptoms ($B = 0.37$, $SE = 0.20$, $p = .07$). Third, there was no direct effect of 5-HTTLPR on Wave 2 ADHD when CU traits and narcissism were included in the model ($B = -0.07$, $SE = 0.54$, $p = .90$). We calculated the total and specific indirect effects of 5-HTTLPR on Wave 2 ADHD symptoms through CU traits and narcissism by specifying 1,000 bootstrap simulation samples and calculating point estimates and the 95% bias corrected and accelerated (BCa) CIs for each indirect effect. The point estimate of the difference between the total effect and direct effect through the two mediators (i.e., total indirect effect) differed significantly from zero such that CU traits and narcissism independently mediated the association of 5-HTTLPR and the total number of parent-rated Wave 2 ADHD symptoms (Table 2). Next, we tested whether child sex moderated the mediation of CU traits and narcissism on Wave 2 ADHD symptoms from 5-HTTLPR (i.e., putative conditional indirect effects of CU traits and narcissism). The index of moderated mediation indicated that sex moderated the indirect effects of narcissism (CI: 0.02 to 1.08), but not CU traits (CI: -0.50 to 0.91). Specifically, the indirect effect of 5-HTTLPR on Wave 2 ADHD symptoms through narcissism was significant in girls, but not boys. The point estimates and 95% CIs for the conditional indirect effects appear in Table 3.

We then reproduced the identical model above but examined P+T Wave 2 ADHD symptoms (Figure 2). Adjusting for P+T Wave 2 ODD diagnostic status, race-ethnicity, and family income, without regard to CU traits and narcissism, 5-HTTLPR was unrelated to the number of P+T Wave 2 ADHD symptoms ($B = -0.84$, $SE = 0.63$, $p = .19$). The number of long alleles again was positively associated with CU traits ($B = -0.51$, $SE = 0.21$, $p = .02$) and

narcissism ($B = -0.53$, $SE = 0.24$, $p = .03$). Second, CU traits ($B = 0.76$, $SE = 0.25$, $p < .01$) and narcissism were positively associated with P+T Wave 2 ADHD symptoms ($B = 0.44$, $SE = 0.23$, $p = .05$). Third, there was no direct effect of 5-HTTLPR on Wave 2 ADHD symptoms with CU traits and narcissism included ($B = -0.22$, $SE = 0.62$, $p = .73$). Bootstrapping estimated that the total indirect effect of 5-HTTLPR on the number of Wave 2 ADHD symptoms differed significantly from zero such that CU traits and narcissism significantly mediated the association of 5-HTTLPR and the number of P+T Wave 2 ADHD symptoms. Lastly, sex significantly moderated the indirect effect of narcissism (CI: 0.001 to 1.35), but not CU traits (CI: -0.04 to 1.57), such that narcissism was a significant mediator in girls but not boys (Table 3). Although sex did not moderate the indirect effect of CU traits, posthoc tests indicated that the indirect effect of CU traits was also a significant mediator in girls but not boys.¹

5-HTTLPR and Wave 2 ODD: Mediation by Wave 1 CU Traits and Narcissism

We then tested whether Wave 1 CU traits and narcissism mediated the association of 5-HTTLPR on parent-rated Wave 2 ODD symptoms, controlling for race-ethnicity, family income, and Wave 2 ADHD diagnostic status (Figure 3). Excluding CU traits and narcissism from the model, 5-HTTLPR was unrelated to Wave 2 ODD symptoms ($B = -0.21$, $SE = 0.19$, $p = .26$), whereas the number of 5-HTTLPR long alleles positively predicted CU traits ($B = -0.43$, $SE = 0.19$, $p = .03$) and narcissism ($B = -0.44$, $SE = 0.21$, $p = .04$). Second, narcissism positively predicted the number of Wave 2 ODD symptoms ($B = 0.15$, $SE = 0.07$, $p = .04$), whereas CU traits did not ($B = 0.07$, $SE = 0.08$, $p = .38$). Third, there was no significant direct effect of 5-HTTLPR on the total number of Wave 2 ODD symptoms when CU traits and narcissism were included ($B = -0.12$, $SE = 0.19$, $p = .53$). Bootstrapping revealed the total indirect effect of 5-HTTLPR on Wave 2 ODD symptoms differed from zero, such that narcissism, but not CU traits, significantly mediated the association of 5-HTTLPR and parent-rated Wave 2 ODD symptoms (Table 2). There was no moderation by sex for either the indirect effects of CU traits (CI: -0.04 to 0.18) or narcissism (CI: -0.02 to 0.42 ; Table 3).

We then investigated whether Wave 1 CU traits and narcissism mediated the association of 5-HTTLPR with P+T Wave 2 ODD symptoms, accounting for race-ethnicity, family income, and Wave 2 ADHD diagnostic status (Figure 4). Excluding CU traits and narcissism from the model, 5-HTTLPR was unrelated to Wave 2 ODD symptoms ($B = -0.11$, $SE = 0.18$, $p = .54$), but the number of 5-HTTLPR long alleles was positively associated with CU traits ($B = -0.50$, $SE = 0.21$, $p = .02$) and narcissism ($B = -0.49$, $SE = 0.24$, $p = .04$). Second, CU traits were unrelated to the number of P+T Wave 2 ODD symptoms ($B = 0.04$, $SE = 0.07$, $p = .57$), whereas narcissism was positively associated with ODD ($B = 0.22$, $SE = 0.06$, $p < .01$). Third, there was no direct effect of 5-HTTLPR on Wave 2 ODD symptoms with CU traits and narcissism included ($B = 0.02$, $SE = 0.18$, $p = .92$). Bootstrapping revealed that

¹Given the presence of 35 siblings within the study, we replicated all models with a subset of 157 participants (reflecting one randomly selected sibling). All original significant indirect effects based on the original 174 participants remained significant with the subsample with one sibling per set, with the exception of P+T-rated ADHD symptoms. That is, narcissism also significantly mediated the effect of 5-HTTLPR and P+T ADHD symptoms, consistent with the indirect effects observed for the parent-rated ADHD symptoms outcome.

narcissism, but not CU traits, significantly mediated the effect of 5-HTTLPR on the total number of P+T ODD symptoms at Wave 2 (Table 2). Finally, sex moderated indirect effects of narcissism (CI: 0.02 to 0.50), but not CU traits (CI: -0.09 to 0.19), such that narcissism had a significant indirect effect in girls but not boys (Table 3).

5-HTTLPR and Wave 2 Antisocial Behavior: Mediation by Wave 1 CU Traits and Narcissism

We tested whether Wave 1 CU traits and narcissism mediated the association of 5-HTTLPR with youth self-reported antisocial behavior, controlling for race-ethnicity, family income, and Wave 2 ADHD and ODD diagnostic status. Excluding CU traits and narcissism from the model, 5-HTTLPR was unrelated to Wave 2 antisocial behavior ($B = -0.64$, $SE = 0.77$, $p = .41$), whereas the number of 5-HTTLPR long alleles was positively associated with CU traits ($B = -0.73$, $SE = 0.25$, $p < .01$) and positively and marginally associated with narcissism ($B = -0.55$, $SE = 0.31$, $p = .08$). Second, neither CU traits nor narcissism was associated with Wave 2 antisocial behavior ($B = 0.02$, $SE = 0.33$, $p = .96$ and $B = 0.20$, $SE = 0.27$, $p = .47$, respectively). Third, there was no significant direct effect of 5-HTTLPR on Wave 2 antisocial behavior when CU traits and narcissism were included ($B = -0.52$, $SE = 0.81$, $p = .52$). Bootstrapping revealed the total indirect effect of 5-HTTLPR on Wave 2 antisocial behavior did not differ from zero, suggesting that neither CU traits nor narcissism individually significantly mediated the association of 5-HTTLPR and youth self-reported antisocial behavior (Table 2). Sex did not moderate the indirect effects of CU traits (CI: -1.09 to 0.82) nor narcissism (CI: -0.23 to 1.62; Table 3).²

Discussion

The explanatory factors underlying the significant association of 5-HTTLPR on childhood ADHD, ODD, as well as antisocial behavior are largely unknown. Using temporally-ordered predictors, mediators, and outcomes, we evaluated whether Wave 1 CU traits and narcissism mediated the association of 5-HTTLPR with multi-method and multi-informant ratings of ADHD and ODD symptoms, as well as youth self-reported antisocial behavior two years following baseline. Among five to nine year-old children with and without ADHD, multiple mediation analyses with bootstrapping revealed that the number of long alleles of 5-HTTLPR was positively associated with CU traits across every model and was positively associated with narcissism for parents (and marginally for models involving youth ratings). Wave 1 CU traits were positively associated with both parent and teacher ratings of Wave 2 ADHD (with control of race-ethnicity, family income, comorbidity, and narcissism), but not ODD. Wave 1 narcissism was positively associated with parent and teacher ratings of Wave 2 ADHD and ODD (controlling for race-ethnicity, family income, comorbidity, and CU traits). Wave 1 narcissism uniquely mediated the effect of 5-HTTLPR on parent and P+T rated ADHD symptoms as well as parent and P+T rated Wave 2 ODD symptoms. Wave 1 CU traits uniquely mediated the effect of 5-HTTLPR on parent as well as P+T rated Wave 2 ADHD, whereas neither CU traits nor narcissism mediated the association of 5-HTTLPR

²All five models were replicated using the original seven-item narcissism scale, which included the following items that overlap with ODD symptoms: "Uses or 'cons' others" and "Becomes angry when corrected" relative to the five-item narcissism scale (see details in Method section). Results were highly similar to the models described above when using the original seven-item narcissism scale. Results are available upon request.

with Wave 2 youth-rated antisocial behavior. Lastly, sex significantly moderated mediation of parent and P+T ADHD symptoms and P+T ODD symptoms, such that indirect effects of narcissism were present in girls, but not boys.

These preliminary results suggest that individual differences in narcissism and CU traits are potential intermediate constructs underlying the association of 5-HTTLPR with ADHD and conduct problems. Although CU traits are typically used to estimate psychopathy, its association with conduct problems is often attenuated when narcissism is simultaneously considered (Barry et al., 2007). This study simultaneously accounted for narcissism and CU traits, but additionally controlled for ADHD, race-ethnicity, and family income, perhaps further diminishing the explanatory power of CU traits for oppositional problems. Similarly, given that CU traits are somewhat less stable in young children relative to later in childhood and adolescence (Frick et al., 2003; Hawes & Dadds, 2007), the diminished predictions of oppositionality and antisocial behavior may be a result of the young developmental stage of the current sample. Thus, CU traits may predict later conduct problems as youth mature and CU traits become more stable (Pardini & Loeber, 2008). Moreover, clinical samples often exhibit elevated CU traits, and thus might be more likely to uncover effects of CU traits on conduct problems (Allen et al., 2013). In addition to the importance of developmental perspectives to the assessment of psychopathic traits, this study observed important sex differences in the etiology of ADHD and ODD, such that indirect effects of narcissism were only present in girls. These results are consistent with recent evidence of distinct etiological pathways to antisocial behavior and psychopathy in boys and girls (Isen et al., 2010; Krischer & Sevecke, 2008). Specifically, 5-HTTLPR may be more relevant to ADHD and ODD through psychopathic traits in girls, but less so in boys. Given evidence linking female hormones to serotonergic functioning (Fink et al., 1999; Verona & Vitale, 2006), this pathway may reflect individual differences in their physiological development secondary to hormones. Although sex differences in relation to stress and psychopathy have been observed with cortisol (O'Leary, Taylor, & Eckel, 2010), additional inquiry into sex differences within trajectories to ADHD, ODD, and psychopathic traits from 5-HTTLPR would present potential implications for interventions (e.g., tailoring different interventions for boys and girls). Moreover, specific conduct problem dimensions should be specified (e.g., proactive and reactive aggression), given their different patterns of association (Loeber & Schmaleng, 1985; Muñoz Centifanti et al., 2013). For example, proactive aggression is more strongly associated with psychopathy and family history of substance abuse, whereas reactive aggression is related more strongly with hostile attributional biases and neuroticism (Cima & Raine, 2009). Thus, different forms of conduct problems warrant further examination to improve traction on its considerable heterogeneity.

Consistent with prior studies (Gizer et al., 2009; Nobile et al., 2007), the 5-HTTLPR long allele was associated with psychopathic traits, ADHD, and ODD. Further research is needed, however, to clarify the consequences of variation in 5-HTTLPR, which has been implicated in the development of the structure, function, and connectivity of neural structures and circuitry that influence negative emotionality (e.g., amygdala, perigenual cingulate; Pezawas et al., 2005). In response to an unpredictable acute lab stressor task, women homozygous for the short allele of 5-HTTLPR exhibited activation in a network shown to modulate emotions and mood (i.e., amygdala, thalamus, putamen, and caudate) whereas long allele carriers did

not (Drabant et al., 2012). The long allele was also inversely associated with perceived success at emotion regulation and insula activation, suggesting that carriers of the long allele are less responsive to affective salience (Drabant et al., 2012). These findings suggest that 5-HTTLPR may influence the structure and connectivity of neural circuitry involved in regulation of negative emotions, which are themselves related to psychopathic traits (Pezawas et al., 2005). For example, youth with CU traits exhibited lesser amygdala activation and lower connectivity between the amygdala and ventromedial prefrontal cortex while processing fearful facial expression than youth with ADHD and typically-developing youth (Marsh et al., 2008). Additional research is needed to investigate other intermediate phenotypes that may mediate the association of 5-HTTLPR and psychopathic traits to gain a better understanding of the functional consequences of 5-HTTLPR underlying psychopathic traits. Furthermore, other key constructs (e.g., parent psychopathology) may moderate the development of psychopathic traits among carriers of the long allele. CU traits and narcissism were inversely associated with family income, but only among long allele homozygotes (Sadeh et al., 2010). Thus, exploring these moderators could further refine the multiple pathways from 5-HTTLPR to child attention and conduct problems through psychopathic traits.

A relatively consistent finding from this study was that narcissism plays a key role in the development of attention and oppositionality difficulties and that CU traits are more specifically associated with attention problems. In a key etiological model of psychopathy, Lynam (1996) proposed that youth at risk for “fledgling psychopathy” first exhibit ADHD symptoms before developing conduct problems. Our preliminary results indicate that CU traits are associated with ADHD in middle childhood, but at this developmental stage, they may not yet exhibit elevated antisocial behavior. Moreover, CU traits represent one construct to improve traction on significant ADHD heterogeneity. For example, youth with ADHD and CU traits exhibited lower parasympathetic reactivity and sympathetic activity than youth with ADHD without CU traits (Musser, Galloway-Long, Frick, & Nigg, 2013). Thus, subgroups of youth with ADHD may have unique autonomic correlates of CU traits. If replicated, the current results suggest that targeting CU traits and especially narcissism may potentially interrupt genetic influences on emergent ADHD and conduct problems.

Clinical evidence also highlights that assessing psychopathic traits is relevant given their moderation of treatment response (e.g., psychopathic youth have reduced engagement and less favorable outcomes one year after treatment; O'Neill, Lidz, & Heilbrun, 2003). More recent interventions specifically targeting youth psychopathic traits and inclusion of these modalities may effectively remediate early signs of CU traits and narcissism to minimize the risk of developing attention and conduct problems. Emotional recognition skills improved disruptive behavior in youth with CU traits (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012) and an intensive parenting intervention significantly reduced psychopathic traits at a 20-month follow-up (McDonald, Dodson, Rosenfield, & Jouriles, 2011). Notably, 51% of youth with ADHD, ODD, and CD that received a stimulant optimization protocol (while concurrently receiving family-focused behavioral interventions) experienced reduced aggression, regardless of CU traits levels; CU traits also decreased over the course of treatment, providing preliminary evidence that stimulant pharmacology might be effective in reducing CU traits in ADHD youth (Blader et al., 2013). In addition, youth with ADHD and

conduct problems exhibited reduced response to behavioral therapy if they also had CU traits than if they did not; this discrepancy was reduced, however, when youth were also provided stimulant medication, providing additional evidence for the use of pharmacological interventions for comorbid ADHD and CU traits (Waschbusch, Carrey, Willoughby, King & Andrade, 2007). Future research should test whether narcissism similarly moderates treatment outcome and further refine which treatment modalities are most efficacious in the treatment of psychopathic traits.

In this study, psychopathic traits were unrelated to the development of youth-reported antisocial behavior specifically. This may reflect the modest agreement between informants for children's social, emotional, and behavioral problems (Achenbach, McConaughy, & Howell, 1987). For example, youth and teachers have been indicated to report significantly lower levels of child inattentiveness/hyperactivity and oppositionality relative to parent informants (Loeber, Green, Lahey, & Stouthamer-Loeber, 1991). Accordingly, laboratory measures of youth antisocial behavior are valuable (Hinshaw et al., 1995). Future studies should also examine youth self-reported psychopathic traits to determine if mediational pathways are robust across additional informants, given the incremental validity of youth self-report in studies of psychopathic traits (Jones & Miller, 2012). Lastly, although latent variable modeling could facilitate the identification of latent constructs (e.g., conduct problems, antisocial behavior) from multiple informants (De Los Reyes & Kazdin, 2005), statistical advances do not currently allow for the use of multiple mediation and more powerful bootstrapping procedures within a latent variable modeling framework.

We consider several important study limitations. First, we assumed 5-HTTLPR effects were linear, although alternative models of transmission cannot be ruled out (e.g., dominant). Second, the rs25531 polymorphism is indicated to alter the functionality of the long allele (Greenberg et al., 1999; Nobile et al., 1999); however, it was unavailable in the current study, and thus we cannot ascertain its role within the current study. Third, the internal consistency of CU traits was modest, likely reflecting the relative infrequency of these traits, especially in this relatively young sample. Improved measurement approaches for these critical constructs should be prioritized, including behavioral or in vivo tasks (Kimonis, Frick, Munoz, Aucoin, 2007). Fourth, although we covaried for race-ethnicity, population stratification is a threat in ethnically heterogeneous samples. However, population stratification remains a more significant threat in large samples and samples with highly distinct ancestral subgroups (Hutchison et al., 2004). Lastly, the current study did not account for the role of gene-environment correlations and interactions that are also likely to contribute to the development of ADHD, ODD, and antisocial behavior.

The current study provides preliminary evidence of CU traits and narcissism as two intermediate constructs underlying the association of 5-HTTLPR with clinically-significant attention and conduct problems. Specifically, the number of long alleles of 5-HTTLPR was positively associated with CU traits and narcissism, and narcissism was positively associated with attention and conduct problems two years later, whereas CU traits were positively associated with attention problems two years later. Notably, narcissism significantly mediated the effect of 5-HTTLPR on parent-rated and a composite measure of parent- and teacher-rated Wave 2 attention and conduct problems, whereas CU traits uniquely mediated

the effect of 5-HTTLPR on multi-informant rated attention problems two years later. Child sex moderated these indirect effects where significant mediation was observed only in girls. If narcissism and CU traits are implicated as intermediate phenotypes in future studies, they may constitute logical targets for intervention to potentially prevent the development of highly consequential externalizing behavior.

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Figure 1.
Multiple Mediator Model of Parent-Rated ADHD Symptoms by Psychopathic Traits and 5-HTTLPR (Beta Coefficients)
† p .10. * p .05. ** p .01

Figure 2.
Multiple Mediator Model of Parent- and Teacher-Rated ADHD Symptoms by Psychopathic
Traits and 5-HTTLPR (Beta Coefficients
* p .05. ** p .01.

Figure 3.
Multiple Mediator Model of Parent-Rated ODD Symptoms by Psychopathic Traits and 5-HTTLPR (Beta Coefficients)
* p .05. ** p .01.

Figure 4. Multiple Mediator Model of Parent- and Teacher-Rated ODD Symptoms by Psychopathic Traits and 5-HTTLPR (Beta Coefficients)
* p .05. ** p .01.

Table 1

Descriptive Statistics for Demographic Variables

Variable	<i>M (SD) or % of Sample</i>
Age (<i>SD</i>)	7.34 (1.09)
% Males	69.1
% Caucasian	52.1
% Household income < \$70,000	31
WISC-IV FSIQ (<i>SD</i>)	106.22 (15.16)
% Wave 1 ADHD diagnosis	52.4
% Wave 1 ODD diagnosis	28.4
% Wave 2 ADHD diagnosis	38.3
% Wave 2 ODD diagnosis	19
% Wave 2 CD diagnosis	2

Note. SD = standard deviation; WISC-IV FSIQ = Wechsler Intelligence Scale for Children, Fourth Edition, Full Scale IQ; ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder; CD = conduct disorder.

Table 2

Mediation by Callous-Unemotional Traits and Narcissism on 5-HTTLPR with ADHD Symptoms and ODD Symptoms

	Point est.	SE	95% BCa bootstrap CI	
			Lower	Upper
Parent-Rated ADHD Symptoms				
CU Traits	-.29	.20	-.81	-.02
Narcissism	-.17	.13	-.56	-.01
TOTAL	-.47	.22	-.97	-.09
Parent-Teacher Composite of ADHD Symptoms				
CU Traits	-.39	.21	-.88	-.04
Narcissism	-.23	.16	-.66	-.04
TOTAL	-.62	.28	-1.22	-.15
Parent-Rated ODD Symptoms				
CU Traits	-.03	.04	-.19	.02
Narcissism	-.06	.05	-.21	-.005
TOTAL	-.09	.06	-.26	-.004
Parent-Teacher Composite of ODD Symptoms				
CU Traits	-.02	.04	-.15	.04
Narcissism	-.11	.07	-.28	-.01
TOTAL	-.13	.07	-.31	-.02
Youth-Reported Antisocial Behavior				
CU Traits	-.01	.30	-.57	.67
Narcissism	-.11	.20	-.76	.14
TOTAL	-.12	.27	-.65	.46

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

Table 3

Moderated Mediation of Callous-Unemotional Traits and Narcissism on 5-HTTLPR with ADHD Symptoms and ODD Symptoms by Sex

	Conditional Indirect Effect in Males				Conditional Indirect Effect in Females			
	95% BC bootstrap CI				95% BC bootstrap CI			
	Point est.	SE	Lower	Upper	Point est.	SE	Lower	Upper
Parent-Rated ADHD Symptoms								
CU Traits	-.25	.20	-.83	.02	-.37	.31	-1.18	.07
Narcissism	-.06	.11	-.38	.08	-.42	.27	-1.17	-.03
Parent-Teacher Composite of ADHD Symptoms								
CU Traits	-.21	.22	-.79	.14	-.74	.36	-1.59	-.17
Narcissism	-.08	.14	-.50	.10	-.56	.36	-1.46	-.01
Parent-Rated ODD Symptoms								
CU Traits	-.02	.04	-.16	.02	-.04	.06	-.23	.04
Narcissism	-.02	.04	-.13	.03	-.13	.11	-.42	.01
Parent-Teacher Composite of ODD Symptoms								
CU Traits	-.004	.03	-.09	.04	-.02	.08	-.19	.14
Narcissism	-.03	.05	-.14	.06	-.23	.12	-.52	-.06
Youth-Reported Antisocial Behavior								
CU Traits	-.02	.19	-.48	.32	-.07	.56	-1.01	1.26
Narcissism	-.02	.13	-.41	.17	-.33	.43	-1.51	.26

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