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Gene-Environment Interplay for Childhood and Adolescent Antisocial Behavior

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Gene-Environment Interplay for
Childhood and Adolescent Antisocial Behavior

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

by

Avital Elisa Falk

2014
Gene-Environment Interplay for
Childhood and Adolescent Antisocial Behavior

by

Avital Elisa Falk
Doctor of Philosophy in Psychology
University of California, Los Angeles, 2014
Professor Steve S. Lee, Chair

Individual differences in parenting behavior are associated with youth conduct problems (CP), but few studies examine the independent associations of positive and negative parenting with CP, despite their factorial independence. Monoamine oxidase-A (MAOA) genotype and callous-unemotional (CU) traits are also associated with CP and may moderate the association between parenting behavior and CP. This dissertation is based on two independent samples: Sample 1 is a two-year prospective longitudinal study of approximately 221 well-characterized 6-9 year-old youth with and without attention-deficit/hyperactivity disorder (ADHD) whereas Sample 2 consisted of nearly 2,500 adolescents followed prospectively for six years from the National Longitudinal Study of Adolescent Health (Add Health). In each sample, we tested the association of multi-method and multi-dimensional measures of positive and negative parenting factors,
MAOA, and their interaction as predictors of growth in CP (oppositional defiant disorder, conduct disorder, rule-breaking behavior, and aggressive behavior), including evaluation of potential differential susceptibility. We also tested the moderating role of CU traits with respect to the prospective prediction of CP from parenting behavior in Sample 1. Several key results emerged from these inter-related studies: first, overall, MAOA-L youth displayed more significant growth in CP, but a significant parenting x MAOA interaction suggested that children with MAOA-H displayed more growth in CP at higher levels of corporal punishment and at lower levels of parental involvement than MAOA-L youth. We also observed significant interactions where positive reinforcement predicted growth in aggressive behavior, but only in children with high CU traits: no association was observed among children with mean or low CU traits. Overall, these studies suggest that genetic and trait-level factors significantly moderated the association between parenting and CP. Implications for intervention development and delivery are discussed with respect to the development of significant CP.
The dissertation of Avital Elisa Falk is approved.

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2014
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CHAPTER ONE: Introduction

Conduct problems (CP) are a diverse collection of aggressive and rule-breaking acts including violence, theft, and property destruction. Childhood CP, particularly physical aggression, is stable over time, as evidenced by the fact that more than 50% of children with oppositional defiant disorder (ODD) or conduct disorder (CD) retain their diagnosis later in development (Loeber, Burke, & Pardini, 2009; Reid, Webster-Stratton & Baydar, 2004). Negative outcomes associated with early CP include continued conduct problems (i.e., homotypic continuity) but CP also prospectively predicts anxiety, depression, substance problems, and school failure (i.e., multifinality), as well as functional impairment such as accidental injuries (Odgers et al., 2007; Woodward, Fergusson & Horwood, 2002). Given its public health significance, identifying early predictors of CP will facilitate the development and implementation of new interventions.

Individual differences in negative parenting behavior are reliably associated with youth CP (Calkins and Keane, 2009). Although negative parenting behavior, such as inconsistent parenting and harsh discipline, is a risk factor for CP, few studies have examined whether positive parenting behavior, independent of negative parenting behavior, is associated with CP. Positive parenting behavior consists of dimensions such as parental involvement and positive reinforcement and although positive parenting behavior is central to efficacious interventions for CP, studies have largely focused on negative parenting and CP. That is, despite being factorially independent, there are few studies of the independent association of positive and negative parenting behavior in the development of CP (see Chronis et al., 2007 for a key exception).

Behavior genetic studies suggest that variation in CP reflects genetic and environmental influences, including gene x environment interaction (G x E). Parenting behavior is a specific
and biologically plausible environmental condition in the context of G x E given that exposure to individual differences in parenting behavior (and nurturing behavior overall) influences offspring neurobiology, a necessary condition for rigorous G x E studies (Moffitt, 2005). One genetic variant potentially relevant to CP, including through its influence on individual differences in reactivity to parenting behavior, is the 30-base pair variable number tandem repeat (VNTR) polymorphism of the monoamine-oxidase-A gene (MAOA). MAOA has been implicated in aggressive behavior in humans and non-human animals with the low-activity genotype altering neural circuitry relevant to social evaluation and emotion regulation such as human amygdala reactivity, an epicenter of how affective stimuli is appraised (Buckholtz et al., 2008). MAOA knockout mice also exhibit serotonergic changes that mediated observed increases in aggressive behavior (Takahashi, Quadros, de Almeida & Miczek, 2010). Parenting behavior is a plausible environmental factor to study as a moderator of the association of MAOA with psychopathology because naturally-occurring differences in parental nurturing behavior reliably predict changes in offspring neurobiology in humans and non-human animals (Liu et al., 1997; Moffitt, Caspi, & Rutter, 2005; Zhu et al., 2010). Thus, MAOA may affect known differences in children’s behavioral development that are associated with exposure to positive and negative parenting behavior.

Despite advances in identifying G x E underlying psychopathology, there is a frequent assumption of a diathesis stress model where environmental risk/adversity and genetic vulnerability interactively heighten risk for illness. However, differential susceptibility contends that some genetic variants may increase reactivity to environmental conditions overall (i.e., positive and negative) rather than specifically to environmental adversity (Belsky et al., 2009). Thus, if MAOA simultaneously influences sensitivity to positive and negative aspects of the
environment, consistent with differential susceptibility, this may improve traction on the inconsistent G x E findings to date with respect to MAOA and psychopathology. Despite its theoretical promise, there are few direct empirical tests of this hypothesis, including rigorous evaluation of established criteria for differential susceptibility (Belsky & Beaver, 2011). Therefore, a key contribution of my dissertation will be its formal evaluation of differential susceptibility with MAOA, positive and negative parenting behavior, and CP rather than the relatively “agnostic” approach common in most G x E studies.

There are also likely to be complex and synergistic relationships between parenting behavior and other dimensions underlying the development of CP, including interactions with child constitutional factors. One such factor is individual differences in callous-unemotional (CU) traits, defined as the display and experience of diminished empathy and guilt. CU traits positively predict chronic CP and children with elevated CU traits are clinically and empirically distinct from other antisocial children. Although much of the intervention literature for CP focuses on increasing positive parenting practices, there is replicated evidence that parenting practices were stronger predictors of offspring CP among children with low versus high levels of CU traits (Falk and Lee, 2012; Wootton, Frick, Shelton & Silverthorn, 1997). In light of the potential moderating role of CU traits for parenting behavior and CP, there are important implications for intervention research and development given that children with CU traits may respond to differentially to available interventions.

Overall, this dissertation examined the independent and interactive effects of positive and negative parenting behavior, youth MAOA genotype, and CU traits with multi-dimensional measures of CP using diverse research strategies (e.g., prospective longitudinal designs, multi-informant/method measures of key constructs). Furthermore, I tested whether predictions of CP
from positive and negative parenting behavior were separately moderated by CU traits and MAOA. These studies are based on two independent samples with important similarities and complementary characteristics. Sample 1 is from a two-year prospective longitudinal study of approximately 221 intensively ascertained 6-9 year-old youth with and without attention-deficit/hyperactivity disorder (ADHD), and Sample 2 consisted of nearly 2,500 adolescents followed prospectively for six years from the National Longitudinal Study of Adolescent Health (Add Health). Both samples are genetically-informative and developmentally-sensitive, thus improving traction on identifying precursors to significant CP. These studies may also inform intervention development by identifying predictors of CP in children as well as identifying children who may respond differentially to parenting-based interventions. My specific aims were as follows:

Study 1

1. To test the associations of baseline parenting and MAOA with growth in parent-rated Rule Breaking Behavior and Aggressive Behavior, and parent- and teacher-rated oppositional defiant disorder (ODD) and conduct disorder (CD), using data from Sample 1, a prospective longitudinal study of approximately 221 boys with and without ADHD who were evaluated at age 6-9 and again 2 years later.

2. To reproduce the same model, with the addition of interaction terms between parenting behavior and MAOA to examine the moderating effect of MAOA in Sample 1.

Study 1a

1. As with Study 1, our goal was to examine the association of parenting behavior and youth
MAOA genotype with youth CP in Sample 1, but we specifically included structured
diagnostic interviews of ODD and CD to supplement the parent reports.

2. Again, to examine the association of parenting behavior and youth MAOA genotype with
youth CP in Sample 1, but we additionally included observational measures of parenting
behavior to supplement parental self-reports.

3. To test the association of MAOA with of CP in Sample 1 at multiple points in
development by separately examining the association between MAOA and CP at baseline
and at the two-year follow-up.

4. In the presence of significant MAOA x parenting interactions, our goal was to formally
evaluate evidence for differential susceptibility in Sample 1, using established criteria
(Belsky, Bakermans-Kanenburg & van IJzendoorn, 2007) such that MAOA increases
sensitivity to positive and negative aspects of the environment (i.e. positive and negative
parenting behavior).

5. Similar to Study 1, our goal was to test the association of MAOA and parenting with
growth in CP using data from a second sample, Sample 2, which consists
of an unselected sample of approximately 2500 adolescents who were evaluated at age
12-18 years (Wave 1) and 13-19 years (Wave 2). As in Sample 1, we planned to evaluate
each significant interaction for evidence of differential susceptibility.

Study 2

1. To test the associations of baseline parenting behavior and youth CU traits with growth in
parent and teacher rated CP, using data from Sample 1.

2. To reproduce the same model, with the addition of interaction terms between parenting
behavior and CU traits to examine the moderating effect of CU traits in Sample 1.

Study 2a

1. As with Study 2, our goal was to examine the association of baseline positive parenting behavior and youth CU traits with parent and teacher rated CP, using data from Sample 1, but we specifically included observational measures of parenting behavior as well as measure of negative parenting behavior (corporal punishment).

2. Again, to examine the association of baseline positive parenting behavior and youth CU traits with parent and teacher rated CP, using data from Sample 1, but we specifically included structured diagnostic interviews of ODD and CD to supplement the parent reports.
CHAPTER TWO: Interactive association of MAOA genotype and parenting behavior: Prospective prediction of youth conduct problems

Conduct problems (CP), consisting of aggressive/overt and rule breaking/covert acts such as violence, theft, and property destruction (Dishion & Patterson, 2006), are stable as evidenced by the fact that a majority of children with oppositional defiant disorder (ODD) or conduct disorder (CD) are similarly diagnosed later in development (Loeber, Burke, & Pardini, 2009; Reid, Webster-Stratton & Baydar, 2004). Representing specific taxon under the broader construct of CP, ODD and CD uniquely predict functional impairment (e.g., mental health service utilization, accidental injuries), later antisocial personality disorder (i.e., homotypic continuity), as well as anxiety, depression, substance problems, and school failure (i.e., multifinality) (Biederman et al., 2008; Odgers et al., 2007; Pardini & Fite, 2010; Woodward, Fergusson & Horwood, 2002). Given its public health significance, identifying early predictors of CP is necessary to facilitate the development and implementation of new interventions.

Attention-deficit/hyperactivity disorder (ADHD) is critical to the study of CP given that ADHD typically accompanies early-onset CP (Hinshaw, Lahey, & Hart, 1993), which is stable, often intractable to intervention, and associated with numerous risk factors (Chronis et al., 2007; Pettit, Bates, & Dodge, 1997). ADHD and comorbid ODD/CD is particularly pernicious given that early-onset CD youth, which is typically accompanied by ADHD, account for a majority of violence in the United States (Moffitt, Caspi, Rutter & Silva, 2001). In particular, the combination of early ADHD and co-occurring CP may reflect unique etiological influences relative to later-onset CP without ADHD. For example, children with ADHD and CP were more likely to have siblings with both ADHD and CP rather than ADHD alone, suggesting potentially specific etiological influences on ADHD and co-occurring CP (Christiansen, et al., 2008).
Danforth, Connor and Doerfler (2014) observed that among children with ADHD, potent risk factors including genetic variation, neurodevelopmental impairment (e.g., inattention, executive functioning deficits), and negative parent-child interaction may increased risk for CP. Thus, an integrative model of CP must adequately consider the role of early ADHD in fueling significant trajectories of CP (Danforth, et al., 2014).

The 30-base pair variable number tandem repeat (VNTR) polymorphism of the monoamine-oxidase-A (MAOA) gene degrades serotonin, norepinephrine, and dopamine following reuptake from the synaptic cleft and is critical to behavioral regulation (Cases et al., 1995). Alleles at this locus are characterized by differential transcriptional efficiency: the 2 and 3 repeat alleles are considered low activity whereas the 3.5 and 4 repeat alleles are high activity (Sabol, Hu, & Hamer, 1998). MAOA has been implicated in aggression in both human and non-human animals: MAOA knockout mice exhibited blunted defensive and fear-related behavior (Godar, Bortolato, Frau, Dousti, Chen & Shih, 2010) and MAOA was associated with increased aggression in rhesus monkeys (Newman et al., 2005). Although it is unclear what factors mediate the association of MAOA and aggression, MAOA knockout mice exhibited serotoninergic changes that predicted increased aggression (Takahashi, Quadros, de Almeida & Miczek, 2010). In addition to altered neurotransmission, MAOA also influences human amygdala reactivity, an epicenter of how affective stimuli is appraised (Buckholtz, et al., 2008). Despite the biological plausibility of the association of MAOA with aggression and CP more generally, findings have been inconsistent. Men with the low-activity MAOA (MAOA-L) genotype showed heightened amygdala activity and negative emotional reactivity than high-activity (MAOA-H) men (Alia-Klein, et al. 2009) whereas MAOA-H predicted elevated parent- and teacher-rated aggression (Beitchman, Mik, Ehtesham, Douglas & Kennedy, 2004; Vanyukov et al., 2007) Similarly, in a
sample of 975 seven year-old boys, MAOA-H was associated with more mental health problems relative to MAOA-L youth (Kim-Cohen et al., 2006). Yet other studies found that MAOA genotype was unrelated to CP (Caspi et al. 2002; Edwards et al., 2010; Haberstick et al. 2005; Huizinga et al. 2006; Young, et al., 2006). These inconsistent findings suggest that the precise association of MAOA and CP, including possible allelic heterogeneity and moderation by environmental factors, requires further scrutiny.

Negative parenting (i.e., harsh/punitive, inconsistent, poor monitoring) is a reliable predictor of CP across development (Calkins & Keane, 2009; DeKlyen, Speltz & Greenberg, 1998; Campbell, Shaw, & Gilliom, 2000; Hoeve et al., 2009). Negative parenting behavior was the most robust predictor of CP and parenting behavior more generally accounted for 11% of the variance in delinquency in a large meta-analysis (Hoeve et al., 2009). Similarly, negative parenting behavior with 3 year-old children was among the strongest predictors of CP at school entry (Campbell et al. 2000). Although negative parenting behavior consists of separable facets, corporal and harsh punishment strongly predicts CP (Hecker, Hermenau, Isele & Elbert, 2013). Early corporal punishment prospectively predicted parent-rated behavior problems at both 36 months and again in 1st grade (Mulvaney & Mebert 2007). However, predictions of negative outcome (e.g., CP) from harsh physical punishment may reflect other, correlated dimensions of parenting behavior. For example, positive parenting behavior is factorially independent from corporal punishment and negative parenting practices (Gardner, Hutchings, Bywater & Whitaker, 2010). Among 323 African-American 10-12 year-old boys, controlling for other parenting dimensions, including warmth, monitoring, and verbal abuse, corporal punishment uniquely predicted CP in the main effects model (Evans, Simons & Simons, 2012). Therefore, there is considerable evidence that negative parenting behavior is important to the development of CP.
Despite its centrality to interventions for CP and its role in promoting offspring development overall (Dornbusch, Ritter, Leiderman, Roberts & Fraleigh, 1987; Gardner, Ward, Burton & Wilson, 2003; Reid, 1987; Steinberg, 2001), there are relatively few studies of positive parenting and CP. Positive parenting, consisting of praise, warmth, consistency, and involvement (Forgatch & DeGarmo, 1999; Gardner, Sonuga-Barke & Sayal, 1999; Hinshaw et al., 2000), is independent from negative parenting, protects against risk factors for CP, and prevents its persistence (Forgatch & DeGarmo, 1999; Gardner, Hutchings, Bywater & Whitaker, 2010). Among 4 to 6 year-old children, controlling for negative parenting behavior as well as initial levels of ADHD and CD, observed positive parenting behavior inversely predicted adolescent CD (Chronis et al., 2007). Similarly, positive parenting behavior prospectively predicted teacher-ratings of grade 6 adjustment beyond negative parenting (Pettit, Bates, & Dodge, 1997). Thus, although positive parenting predicts CP independent of its association with negative parenting, most studies have ignored this important distinction, particularly within the context of different forms of CP (e.g., delinquency vs. overt violence). The current study examines how parenting prospectively predicts multiple measures of CP, including Aggressive Behavior, Rule Breaking Behavior, ODD and CD from multiple informants.

The association of parenting behavior with offspring CP likely includes dynamic and transactional exchanges with other domains of influence, including potential gene x environment interaction (G x E). Differences in exposure to parenting behavior (and parental nurturing behavior more generally) is a plausible environmental factor for rigorous G x E studies because naturally-occurring individual differences in parental nurturing behavior predict changes in offspring neurobiology that may be genetically influenced. Rat pups reared by genetically unrelated mothers who were exposed to high licking and grooming behavior (LG) exhibited
increased hippocampal expression as well as decreased hypothalamic corticotrophin releasing factor and hypothalamic-pituitary-axis reactivity relative to pups reared by genetically unrelated and low LG mothers (Liu et al., 1997). In another study, compared to controls, pups deprived of maternal care displayed down regulation of dopamine transporters and receptors, which contributed to deficits in spatial learning and memory (Zhu et al., 2010). Furthermore, rat pups exposed to high LG showed altered DNA methylation, but this effect was experimentally reversed when pups were raised by low LG mothers (Weaver et al., 2004). Among humans, a recent review found that the quality of early maternal parenting predicted hypothalamic-pituitary-adrenocortical (HPA) axis hypoactivity and offspring stress reactivity (Barrett & Fleming, 2011). Similarly, in a review of mother-infant human interaction, early experiences of responsive caregiving were central to the regulation of neuroendocrine stress activity (Gunnar & Cheatham, 2003). For example, neglected and maltreated infants showed lower levels of and less fluctuation in cortisol throughout the day, relative to typical infants (Gilles, Berntson, Zipf, & Gunnar, 2000); altered cortisol functioning may persist even after environmental conditions improve (Gunnar & Cheatham, 2003). Given that parental nurturing behavior affects offspring neurobiology, a requirement for rigorous G x E studies (Moffitt, Caspi, & Rutter, 2005), parenting behavior is a strong candidate to moderate genetic influences on CP.

Although there is meta-analytic evidence that MAOA moderates the association of environmental adversity (e.g., harsh punishment) and CP (Byrd & Manuck, 2014), rigorous characterization of ODD/CD (and CP more broadly) requires developmental perspectives. For example, defiant behavior often begins in school-age children, but symptoms often decrease over time whereas status violations and non-aggressive behavior (e.g., lying, truancy) increase across adolescence (Maughan, Rowe, Messer, Goodman & Meltzer, 2004; Nock, Kazdin, Hiripi, &
Kessler, 2007). More importantly, causal influences may also be developmentally-sensitive. Early-onset CD (i.e., prior to age 10) is uniquely associated with a diverse array of risk factors including elevated comorbidity, severe neuropsychological deficits, as well as diminished amygdala reactivity on facial emotion recognition tasks (Fairchild et al., 2009; Passamonti et al., 2010; Roisman, Monahan, Campbell, Steinberg & Cauffman, 2010). Age of onset is also differentially associated with multiple forms of CP: whereas early-onset CP is highly heritable and more exclusively associated with overt physical aggression, rule breaking is evident in both child- and adolescent onset CP (Burt, 2012; Burt & Hopwood, 2010). In fact, aggressive behavior and rule breaking behavior may account for differences in CP trajectories associated with age of onset (Burt, Donnellan, Iacono, & McGue, 2011). Similarly, there is meta-analytic evidence that the heritability of aggression and rule breaking behavior are substantially different (Burt, 2009). Overall, these studies suggest that future G x E studies of CP must consider prospective change of separable dimensions of CP over time. The current study addresses this need directly in addition to rigorously accounting for co-occurring ADHD, a key ingredient of integrated models of emergent CP (Danforth, et al., 2014).

There is persuasive evidence that the association between parenting and CP is moderated by MAOA genotype, and that the low-activity genotype predicts worse outcome in conjunction with negative environments than MAOA-L. Given that parenting behavior consists of separable facets, ranging from positive parenting behavior to physical vs. emotional maltreatment and inconsistent discipline, we examined both positive and negative parenting factors, and respectively controlled for negative and positive parenting factors. To improve traction on the specificity of associations with respect to parenting, MAOA, and their interaction with respect to escalating CP over time, we conducted a two-year prospective longitudinal study of 151 six to
nine year-old boys with and without ADHD. Ascertainment consisted multi-dimensional measures of youth CP (i.e., ODD, CD, aggression, rule breaking) as well as structured interviews and rating scales. We hypothesized that, controlling for levels of baseline positive parenting, corporal punishment would positively predict growth in CP and similarly, that controlling for levels of baseline corporal punishment, positive parenting factors would predict decreases in CP. We also predicted that MAOA-L would be associated with high levels of CP. Finally, we predicted that MAOA genotype would moderate the association between parenting and CP, and that high corporal punishment, and low positive parenting would more strongly predict escalating CP among MAOA-L versus MAOA-H youth (Weder et al. 2009).

Methods

Participants

At baseline (i.e., Wave 1 we recruited 221 ethnically diverse (56% Caucasian, 8% African American, 8% Hispanic/Latino, and 28% Mixed/Other) 6 to 9 year-old children (mean age = 7.4, SD = 1.1, 71% male) with (n = 116) and without (n = 105) ADHD. Families were recruited from mailings to local schools, flyers and advertisements, presentations to self-help groups, and referrals from local mental health service providers. All participants were required to have an IQ greater than 70, to live with one biological parent at least halftime, and to be fluent in English. Exclusion criteria for all participants included a previous autism spectrum, seizure, or neurological disorder that prevented full participation in the study. ADHD diagnostic status was based on a fully structured DSM-IV diagnostic interview. All families completed identical screening and testing procedures. To avoid recruiting youth that may have exaggerated differences relative to ADHD probands, children in the non-ADHD control group were allowed
to meet diagnostic criteria for any mental disorder other than ADHD. This procedure has been used in similar studies of childhood ADHD (Lahey et al., 1998). Approximately two years later, all families were invited to participate in a laboratory-based follow-up assessment (i.e., Wave 2), which consisted of procedures that were highly parallel to Wave 1 (e.g., structured diagnostic interviews, standardized test of youth academic achievement). 88% (n = 195) of the original Wave 1 families participated in the Wave 2 follow-up. We observed no significant attrition between the families retained for Wave 2 relative to those who did not participate (e.g., refusal, unable to be located) with respect to age, sex, or average number of Wave 1 ADHD symptoms.

Wave 1 Procedures

Eligibility was determined after parents completed a telephone screening and eligible families who were interested in participating were mailed rating scales. Families were then invited to our laboratory for in-person assessments. Following parent consent and child assent, parents completed the DISC and other measures related to parenting, child behavior, family functioning, personality, and their own psychopathology. During that same time, children completed standardized test of cognitive ability and academic achievement. Whenever possible, children were assessed without medication. Similarly, parents were asked to complete rating scales based on the child’s unmedicated behavior. All interviewers were blind to the child’s diagnostic status. The Institutional Review Board approved all study procedures.

Wave 2 Procedures:

Approximately two years later, all families were invited to participate in a laboratory-based follow-up assessment (i.e., Wave 2) that consisted of procedures that were highly parallel to Wave 1 (e.g., structured diagnostic interviews). 84% (n = 127) of the original Wave 1 families participated in the Wave 2 follow-up. We observed no significant attrition between the families
retained for Wave 2 relative to those who did not participate (e.g., refusal, unable to be located) with respect to age or average number of Wave 1 ADHD symptoms. Given missing data for individual measures, analyses in this paper are based on N = 124.

Wave 1 Measures:

Genotyping

DNA was extracted from saliva. The 30-bp VNTR polymorphism in the promoter region was genotyped using standard primers, which produced the following fragments: 317 (3-repeats), 347 (4-repeats), and 377 (5-repeats). The 3-repeat allele is associated with less transcriptional efficiency and low MAOA activity (L) compared to the 4-repeat allele, which is associated with greater transcriptional efficiency and high activity (H) (Sabol et al., 1998). We coded MAOA genotypes according to their efficiency (Lawson et al., 2003): the low-activity group (MAOA-L; n = 64, 42%) and the high activity group (MAOA-H; n = 87, 58%) (Sjoberg et al., 2007). Given the rarity of the 5-repeat allele in the population and in this sample (n = 3), it was excluded.

Measures

Alabama Parenting Questionnaire (APQ; Frick, 1991). Parents self-reported the frequency of parenting behaviors across parental involvement (10 items), positive parenting (6 items), inconsistent parenting (6 items), poor monitoring (10 items), and corporal punishment (3 items) dimensions. Because the structure of the APQ has varied from three (Hinshaw et. al. 2000; Hawes & Dadds, 2006) to as many as five factors (Shelton, Frick, & Wootton, 1996), we factor analyzed the APQ (results available upon request). We identified four factors: (1) Positive reinforcement (e.g., “compliments child when s/he does something well” and “tells child you like it when s/he helps out around the house);” (2) Parental involvement (e.g., “talks to child about his/her friends);” (3) Parental monitoring/supervision (e.g., “stays out late in the evening past
the time that he/she is supposed to be home);” and (4) Corporal punishment (e.g., “you slap your child when s/he is misbehaving).” Given the centrality of positive parenting to efficacious interventions, we focused on positive reinforcement; negative parenting defined as corporal punishment. These factors correlated highly with the original factor: $r = .96, p < .01$ for positive reinforcement and $r = .86, p < .01$ for corporal punishment. Thus, we scored the APQ according to these previous studies (Essau, Sasagawa, & Frick, 2006; Shelton et al., 1996). The alpha for positive reinforcement was .78, and the alpha for corporal punishment was .61. There was significant variability for positive reinforcement (range = 18-30; mean = 25.62, SD = 2.89; skewness = -0.28, SE = 0.20) and for corporal punishment (range = 3-8; mean = 4.16, SD = 1.34; skewness = 1.25, SE = 0.20).

**Antisocial Behavior**

*Diagnostic Interview Schedule for Children—Fourth Edition (DISC-IV; Shaffer et al. 2000)*. We administered the DISC-IV, a fully structured interview keyed to DSM-IV diagnostic criteria (i.e., symptom levels, duration, persistence, age of onset, functional impairment), to each parent. Children were diagnosed with ADHD if they met full diagnostic criteria according to the DISC. The DISC-IV is widely used and diagnostic designations from the DISC have shown predictive validity in previous studies of ADHD youth (Lee, Lahey, Owens & Hinshaw, 2008).

*Disruptive Behavior Disorder rating scale (DBD; Pelham, Gnagy, Greenslade, & Milich, 1992).* At Wave 1 and Wave 2, parents completed the 45-item DBD rating scale of child DSM-IV ADHD, ODD, and CD. Ratings ranged from 0 (not at all) to 3 (very much). We utilized the total number of ODD symptoms where symptoms were considered present if rated as 2 or 3. The Cronbach’s alpha was .82 and .71 for Wave 1 and 2, respectively. To maximize variability given the relatively young age of the sample, we summed CD ratings to obtain a total score, yielding a
range from 0-45 (15 total items). The Cronbach’s alpha was .70 and .58 for Wave 1 and 2, respectively. To rigorously predict prospective change in ODD/CD, we controlled for Wave 1 ODD and CD in predictions of Wave 2 ODD and CD, respectively.

*Child Behavior Checklist (CBCL) for ages 6–18 (Achenbach and Rescorla, 2001).* The CBCL is a 113-item rating scale scored on a 0–2 metric. Based on normative data from 3210 6–to 18- year-old children, the CBCL yields developmentally sensitive scales of externalizing and internalizing problems as well as competence and impairment. We specifically focused on the raw score from the Aggressive and Rule Breaking Behavior narrow band scales, as well as total Externalizing Problems, which is a combination of Aggressive and Rule Breaking Behavior. As with the DBD, predictions of Wave 2 CBCL Aggressive and Rule Breaking Behavior, and Externalizing Problems included statistical control of each construct at Wave 1, respectively.

*Data Analytic Procedures*

First, in boys, age was only significantly associated with Wave 2 CBCL Aggressive Behavior and it was therefore controlled for in predictions of Wave 2 CBCL Aggressive Behavior only. Race was significantly associated with Wave 2 CBCL Aggressive Behavior and marginally associated with CBCL Rule-Breaking Behavior; we therefore controlled for race in predictions of those outcomes. In girls, although age was unrelated to all Wave 2 outcomes (and was therefore excluded from analyses), race was controlled in predictions of Wave 2 CBCL Aggressive Behavior, CBCL Rule-Breaking Behavior, and DBD ODD symptoms. Race was coded as Caucasian (57.6%), African American (7.2%), Latino (8.6%) and Other/Mixed (26.6%). Among Caucasians, 37% had MAOA-L whereas MAOA-L was present in 60%, 54.5%, and 44% among African American, Latino, and Other/Mixed participants, respectively, suggesting no significant racial-ethnic differences in MAOA ($\chi^2 = 5.7, df = 3, p = 0.13$).
Second, we controlled for Wave 1 ADHD diagnostic status (i.e., ADHD versus non-ADHD) to account for the association of ADHD with MAOA and CP (Marsh et al., 2008). Third, given their considerable skew, we utilized Poisson regression to evaluate the independent prediction of Wave 2 CP from Wave 1 parenting. We then reproduced the fully saturated models, consisting of the same covariates, parenting measures, and MAOA, but included the parenting x MAOA interactions. However, for teacher-rated measures (i.e. DBD ODD and CD), we used generalized estimating equations (GEE). GEE allowed examination of both parent and teacher reports as a single repeated measures factor (i.e., an “informant” factor), thereby reducing the number of statistical tests and accounting for their inter-correlation. To combat missing teacher data, we employed data from multiple imputations for each Wave 1 and Wave 2, which were conducted using 10 iterations of Markov Chain Monte Carlo in SAS PROC MI (Yuan, 2002). The ten iterations were highly consistent: the mean across the imputations of Wave 1 DBD (0-1 scale) was 1.4 (SD = 0.04) and .50 (SD = 0.03) for ODD and CD, respectively, and for Wave 2 DBD (0-3 scale) was 3.01 (SD = 0.27) and .70 (SD = 0.25) for ODD and CD, respectively. We randomly selected one set of imputed teacher data for each Wave 1 and Wave 2 for analyses. Finally, because MAOA is X-linked and its functional properties have not been discerned in females (Kim-Cohen et al., 2006), we analyzed males and females separately so that results can be compared with previous studies, given that boys are typically focused exclusively (Li & Lee, 2011).

Results

Population Stratification and Gene-Environment Correlation (rGE)

Although population stratification (PS) can spuriously affect genetic association studies
and potentially confound the interpretation of potential G x E effects, MAOA was unrelated to age ($\chi^2 = 0.42, p = .94$), race-ethnicity, ($\chi^2 = 1.93, p = 0.22$) and Wave 1 ADHD diagnostic status ($\chi^2 = 2.32, df = 1, p = .09$) in boys, and MAOA was unrelated to age ($\chi^2 = 7.28, p = 0.06$), race-ethnicity, ($\chi^2 = 5.17, p = 0.52$) and Wave 1 ADHD diagnostic status ($\chi^2 = 0.07, p = .80$) in girls. Passive and evocative gene-environment correlations (rGE) may also confound tests of G x E (Jaffee & Price, 2007). We considered evocative rGE as a potential confound or alternative explanation to G x E given that youth MAOA genotype was significantly related to some parenting constructs: MAOA was significantly associated with positive reinforcement, and was unrelated to parental involvement and corporal punishment ($B = -1.20$, $SE = 0.48$, $p = 0.01$, $B = -0.96$, $SE = 0.74$, $p = 0.20$, and $B = -0.05$, $SE = 0.23$, $p = 0.84$, respectively) in boys. In girls, we excluded evocative rGE as a potential confound or alternative explanation to G x E given that youth MAOA genotype was unrelated to parental involvement, positive reinforcement, and corporal punishment ($B = 1.08$, $SE = 1.18$, $p = 0.36$, $B = 1.20$, $SE = 0.78$, $p = 0.13$, and $B = -0.02$, $SE = 0.32$, $p = 0.95$). Passive rGE was not tested given that many mothers were heterozygous for MAOA, thus limiting its potential interpretation.

**Boys:**

**Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DBD CD symptoms**

Based on the GEE platform, and controlling for Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 involvement and MAOA genotype were each unrelated to growth in DBD CD symptoms ($B < 0.01$, $SE = 0.02$, $p = 0.93$ and $B = -0.19$, $SE = 0.20$, $p = 0.35$, respectively). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in DBD CD symptoms ($B = -0.06$, $SE = 0.05$, $p = 0.20$).
Controlling for Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 positive reinforcement and MAOA genotype were each unrelated to growth in DBD CD symptoms ($B = -0.01, SE = 0.03, p = 0.73$ and $B = -0.22, SE = 0.20, p = 0.27$, respectively). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was once again unrelated to growth in DBD CD symptoms ($B = 0.04, SE = 0.07, p = 0.56$).

Controlling for Wave 1 ADHD status, informant, Wave 1 positive parenting, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 corporal punishment and MAOA genotype were each unrelated to growth in DBD CD symptoms ($B = 0.04, SE = 0.07, p = 0.59$ and $B = -0.19, SE = 0.20, p = 0.34$, respectively). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in DBD CD symptoms ($B = 0.23, SE = 0.15, p = 0.14$).

**Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DBD ODD symptoms**

Similarly using the GEE platform and controlling for Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 involvement and MAOA genotype were each unrelated to growth in DBD ODD symptoms ($B < -0.01, SE = 0.01, p = 0.91$ and $B = 0.12, SE = 0.11, p = 0.30$, respectively). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in DBD ODD symptoms ($B = -0.04, SE = 0.03, p = 0.12$).

Controlling for Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 positive reinforcement and MAOA genotype were each unrelated to growth in DBD ODD symptoms ($B = 0.02, SE = 0.02, p = 0.20$ and $B = 0.12, SE = 0.11, p = 0.27$, respectively). In the fully saturated model, the Wave
positive reinforcement x MAOA genotype interaction was unrelated to growth in DBD ODD symptoms (B = -0.01, SE = 0.04, p = 0.71).

Controlling for Wave 1 ADHD status, informant, Wave 1 positive parenting, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 corporal punishment and MAOA genotype were each unrelated to growth in DBD ODD symptoms (B = 0.04, SE = 0.04, p = 0.34 and B = 0.13, SE = 0.12, p = 0.28, respectively). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in DBD ODD symptoms (B = -0.08, SE = 0.08, p = 0.34).

**Associations of Wave 1 Parenting and MAOA genotype with Wave 2 CBCL Aggressive Behavior**

Using poisson regression, and controlling for race, age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Aggressive Behavior, Wave 1 involvement was unrelated to growth in Aggressive Behavior (B = -0.01, SE = 0.01, p = 0.31), and MAOA-L genotype was significantly associated with greater growth in Aggressive Behavior (B = 0.17, SE = 0.08, p = 0.03) relative to MAOA-H. In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in Aggressive Behavior (B = -0.01, SE = 0.02, p = 0.63).

Controlling for race, age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Aggressive Behavior, Wave 1 positive reinforcement was unrelated to growth in Aggressive Behavior (B < 0.01, SE = 0.01, p = 0.73), and MAOA genotype was marginally associated with growth in Aggressive Behavior (B = 0.14, SE = 0.08, p = 0.07). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was unrelated to growth in Aggressive Behavior (B = 0.03, SE = 0.03, p = 0.23).

Controlling for race, age, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1
Aggressive Behavior, Wave 1 corporal punishment significantly predicted growth in Aggressive Behavior (B = 0.06, SE = 0.03, p = 0.04), and MAOA-L genotype was significantly associated with greater growth in Aggressive Behavior (B = 0.16, SE = 0.08, p = 0.04), relative to MAOA-H. In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was significantly related to growth in Aggressive Behavior (B = -0.14, SE = 0.06, p = 0.02). We followed up by examining simple effects for each genotype, and found that controlling for age, race, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 Aggressive Behavior, in children with MAOA-L, punishment was unrelated to growth in Aggressive Behavior, and in children with MAOA-H, punishment significantly predicted growth in Aggressive Behavior (B = -0.03, SE = 0.05, p = 0.54 and B = 0.09, SE = 0.04, p = 0.02, respectively).

**Associations of Wave 1 Parenting and MAOA genotype with Wave 2 CBCL Rule Breaking Behavior**

Using poisson regression, and controlling for race, age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Rule Breaking Behavior, Wave 1 involvement and MAOA genotype were unrelated to growth in Rule Breaking Behavior (B = -0.02, SE = 0.02, p = 0.29 and B = 0.13, SE = 0.13, p = 0.34, respectively). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in Rule Breaking Behavior (B = -0.01, SE = 0.02, p = 0.42).

Controlling for race, age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Rule Breaking Behavior, Wave 1 positive reinforcement was marginally associated with growth in Rule Breaking Behavior (B = 0.04, SE = 0.02, p = 0.09) and MAOA genotype was unrelated to growth in Rule Breaking Behavior (B = 0.18, SE = 0.14, p = 0.20). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was unrelated
to growth in Rule Breaking Behavior (B = 0.01, SE = 0.05, p = 0.84).

Controlling for race, age, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 Rule Breaking Behavior, Wave 1 corporal punishment and MAOA genotype were unrelated to growth in Rule Breaking Behavior (B = 0.02, SE = 0.05, p = 0.73 and B = 0.14, SE = 0.14, p = 0.31, respectively). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in Rule Breaking Behavior (B = -0.12, SE = 0.11, p = 0.27).

**Girls:**

*Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DBD CD symptoms*

Using the GEE platform and controlling for race, Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 involvement was unrelated to growth in DBD CD symptoms (B = 0.05, SE = 0.05, p = 0.32) and MAOA-L genotype was significantly associated with growth in DBD CD symptoms (B = 1.10, SE = 0.46, p = 0.02). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in DBD CD symptoms (B = 0.14, SE = 0.11, p = 0.21).

Controlling for race, Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 positive reinforcement was unrelated to growth in DBD CD symptoms (B = 0.10, SE = 0.10, p = 0.29) and MAOA-L genotype was significantly associated with greater growth in DBD CD symptoms (B = 1.30, SE = 0.46, p < 0.01), relative to MAOA-H. In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was unrelated to growth in DBD CD symptoms (B = 0.04, SE = 0.19, p = 0.84).
Controlling for race, Wave 1 ADHD status, informant, Wave 1 positive parenting, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 corporal punishment was unrelated to growth in DBD CD symptoms (B = -0.06, SE = 0.17, p = 0.71) and MAOA-L genotype was significantly associated with growth in DBD CD symptoms (B = 1.22, SE = 0.49, p = 0.01). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was significantly associated with growth in DBD CD symptoms (B = -1.32, SE = 0.58, p = 0.02). We followed up by examining simple effects for each genotype, however, given the small sample size of girls in each MAOA-H and MAOA-L, simple effects could not be calculated.

**Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DBD ODD symptoms**

Similarly using the GEE platform, and controlling for race, Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 involvement and MAOA genotype were each unrelated to growth in DBD ODD symptoms (B = 0.03, SE = 0.03, p = 0.24 and B = 0.27, SE = 0.30, p = 0.38, respectively). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was only marginally associated with growth in DBD ODD symptoms (B = 0.08, SE = 0.05, p = 0.09).

Controlling for race, Wave 1 ADHD status, informant, Wave 1 corporal punishment, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 positive reinforcement and MAOA genotype were each unrelated to growth in DBD ODD symptoms (B = 0.04, SE = 0.04, p = 0.34 and B = 0.37, SE = 0.29, p = 0.21, respectively). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was unrelated to growth in DBD ODD symptoms (B = -0.03, SE = 0.06, p = 0.62).

Controlling for race, Wave 1 ADHD status, informant, Wave 1 positive parenting, and
Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 corporal punishment was significantly positively associated with growth in DBD ODD symptoms (B = 0.15, SE = 0.07, p = 0.03). MAOA genotype was unrelated to growth in DBD ODD symptoms (B = 0.36, SE = 0.30, p = 0.22, respectively). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was marginally associated with growth in DBD ODD symptoms (B = -0.27, SE = 0.15, p = 0.07).

Associations of Wave 1 Parenting and MAOA genotype with Wave 2 CBCL Aggressive Behavior

Using poisson regression and controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Aggressive Behavior, Wave 1 involvement was unrelated to growth in Aggressive Behavior (B = -0.02, SE = 0.01, p = 0.17), and MAOA genotype was marginally associated with growth in Aggressive Behavior (B = -0.24, SE = 0.14, p = 0.09). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was significantly related to growth in Aggressive Behavior (B = 0.06, SE = 0.03, p = 0.03).

Examination of simple effects found that controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment and Wave 1 Aggressive Behavior, in children with MAOA-L, involvement was unrelated to growth in CBCL Aggressive Behavior symptoms and in children with MAOA-H, involvement was significantly associated with a decrease in CBCL Aggressive Behavior (B < 0.01, SE = 0.02, p = 0.87 and B = -0.05, SE = 0.02, p = 0.04, respectively).

Controlling for race, age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Aggressive Behavior, Wave 1 positive reinforcement was unrelated to growth in Aggressive Behavior (B < 0.01, SE = 0.02, p = 0.89), and MAOA-H genotype was associated with greater growth in Aggressive Behavior (B = -0.35, SE = 0.14, p = 0.02), relative to MAOA-L. In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was
unrelated to growth in Aggressive Behavior (B = -0.02, SE = 0.05, p = 0.71).

Controlling for race, age, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 Aggressive Behavior, Wave 1 corporal punishment was unrelated to growth in Aggressive Behavior (B = 0.02, SE = 0.05, p = 0.67), and MAOA-H genotype was significantly associated with greater growth in Aggressive Behavior (B = -0.34, SE = 0.15, p = 0.02), relative to MAOA-L. In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was marginally related to growth in Aggressive Behavior (B = -0.17, SE = 0.10, p = 0.10).

Associations of Wave 1 Parenting and MAOA genotype with Wave 2 CBCL Rule Breaking Behavior

Using poisson regression and controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Rule Breaking Behavior, Wave 1 involvement was unrelated to growth in Rule Breaking Behavior (B = -0.01, SE = 0.03, p = 0.65), and MAOA genotype was marginally associated with growth in Rule Breaking Behavior (B = -0.50, SE = 0.27, p = 0.06). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in Rule Breaking Behavior (B = 0.08, SE = 0.06, p = 0.17).

Controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Rule Breaking Behavior, MAOA-H genotype was significantly associated with greater growth in Rule Breaking Behavior (B = -0.69, SE = 0.28, p = 0.02), but Wave 1 positive reinforcement was not (B = 0.04, SE = 0.04, p = 0.34). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype significantly predicted growth in Rule Breaking Behavior (B = -0.21, SE = 0.10, p = 0.04). Examination of simple effects displayed that controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Rule Breaking Behavior, in children with MAOA-L, Wave 1 positive reinforcement was unrelated to growth in CBCL Rule
Breaking Behavior, and in children with MAOA-H, positive reinforcement significantly associated with an increase in CBCL Rule Breaking Behavior ($B = -0.01$, $SE = 0.09$, $p = 0.94$ and $B = 0.15$, $SE = 0.06$, $p = 0.01$, respectively).

Controlling for race, age, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 Rule Breaking Behavior, Wave 1 corporal punishment was unrelated to growth in Rule Breaking Behavior ($B = 0.05$, $SE = 0.10$, $p = 0.65$) and MAOA-H genotype was significantly associated with growth in Rule Breaking Behavior ($B = -0.73$, $SE = 0.29$, $p = 0.01$). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in Rule Breaking Behavior ($B = 0.29$, $SE = 0.23$, $p = 0.21$).

**Discussion**

We tested the association of baseline (i.e., Wave 1) positive and negative parenting, child MAOA genotype, and their interactions with respect to prospective change in multidimensional measures of CP across a two-year period in boys and girls with and without ADHD. Controlling for Wave 1 ADHD status and initial levels of CP, in boys, MAOA-L genotype significantly predicted growth in CBCL Aggressive Behavior, and in girls, MAOA-L genotype significantly predicted growth in DBD CD and ODD. Similarly, in separate analyses of boys and girls, controlling for Wave 1 ADHD status and positive parenting, corporal punishment uniquely predicted growth in CBCL Aggressive Behavior and ODD symptoms in boys and girls, respectively. We additionally observed significant parenting x MAOA interactions with respect to emergent CP. Specifically, the corporal punishment x MAOA and the parental involvement x MAOA interactions predicted growth in CBCL Aggressive Behavior in boys and girls, respectively. Finally, the positive reinforcement x MAOA interaction was associated with
growth in CBCL Rule Breaking Behavior in girls.

Consistent with a recent meta-analysis (Byrd & Manuck, 2014), as main effects, corporal punishment and MAOA-L genotype each uniquely predicted growth in CP in boys. However, contrary to our hypothesis, corporal punishment significantly predicted growth in CP for children with MAOA-H, but not for children with MAOA-L. Thus, whereas MAOA-L directly predicted escalating CP, the effect of MAOA-H was contingent upon the presence of elevated corporal punishment (Figure 2.1). In girls, the parental involvement x MAOA interaction uniquely predicted growth in CBCL Aggressive Behavior and consistent with the corporal punishment x MAOA interaction, contrary to our hypothesis, parental involvement predicted decreased CP for children with MAOA-H, but not for children with MAOA-L (Figure 2.2). Finally, the positive reinforcement x MAOA interaction indicated that positive reinforcement was associated with growth in CBCL Rule Breaking Behavior, but only in girls with MAOA-H.

We found evidence that corporal punishment uniquely predicted escalating CP, even with rigorous control of Wave 1 ADHD and positive parenting behavior whereas previous evidence found that corporal punishment was less consequential than other indicators of negative parenting (e.g., verbal or physical maltreatment; Evans et al. 2012; Ferguson, 2013). MAOA-L also uniquely predicted escalating CP, which is consistent with previous findings, including evidence that MAOA-L adults exhibited the highest levels of aggression and violence (Kuepper, Grant, Wiepuetz & Hennig 2013; Reif, et al. 2007). Alia-Klein et al. (2009) proposed that the association of MAOA-L and CP is contingent upon exposure to environmental adversity; thus, it is important to consider not only if, but how parenting and MAOA interact in contributing to CP by conducting further studies, including studies of MAOA and CP that include meditational models to examine how MAOA relates to CP, which will then allow for a better understanding.
of how specific parenting factors moderate that relationship. Additionally, studies that look at the interaction between MAOA and *positive* environmental influence, such as positive parenting, will speak to whether environmental influence broadly, beyond adversity, is influential. This is supported by the interactive effects in this study, which are consistent across environmental adversity (corporal punishment), as well as positive environmental influence (parental involvement).

Beyond the importance of examining multiple parenting constructs and other domains of environmental influence, future studies must appropriately disentangle key facets of CP given that they are likely sensitive to different underlying processes. Notably, this study found consistent interactive effects that were specific to Aggressive Behavior, which may indicate that there may be differentiated patterns of association depending on the specific type of CP. For example, the interaction of MAOA and parenting might be associated with only reactive aggression, and *CBCL* Aggressive Behavior is comprised of items such as “temper tantrums or hot temper,” “argues a lot” and “disobedient at home/school,” which reflect reactive, overt aggression (Tackett, Krueger, Sawyer, & Graetz, 2003). Alternatively, the CBCL Rule Breaking Behavior reflects proactive, covert behaviors including “drinks alcohol without parents’ approval” and “lying or cheating.” These preliminary findings, which diverged reliably across Aggressive Behavior versus Rule Breaking outcomes, suggest that future studies adopt diverse measures of CP that are sensitive to key subtypes (e.g., proactive and reactive aggression).

The specific pattern underlying the significant G x E effect in the current study converged around increased corporal punishment and decreased parental involvement predicting escalating CBCL Aggressive Behavior among MAOA-H youth, but not MAOA-L youth. Aggressive behavior, and reactive aggression more specifically, may reflect amygdala dysfunction, which
results in an altered response and reaction to threat (Coccaro, McCloskey, Fitzgerald & Phan, 2007). Pingault et al. (2013) contended that MAOA-L genotype alters individual differences in reactions to the social environment, including by increasing perception of and sensitivity to negative experiences via increased amygdala activation. This is consistent with evidence that MAOA-L is specifically associated with amygdala hypersensitivity (Buckholtz, et al., 2008; Eisenberger, Way, Taylor, Welch & Lieberman, 2007). In an fMRI study, in a response to negatively valenced visual scenes, individuals with MAOA-L showed exaggerated amygdala activation, which is associated with threat sensitivity (Buckholtz & Meyer-Lindenberg, 2008), and in a large sample of healthy adults, MAOA-L was related to increased risk for violent behavior, and increased amygdala activity during emotional arousal, as measured by response to angry and fearful faces (Meyer-Lindenberg et al., 2006). Similarly, in 38 college-age males, those with MAOA-L showed increased amygdala activation after being insulted by an experimenter (Denson, Dobson-Stone, Ronay, von Hippel & Schira, 2014). Perhaps reflecting processes such as evocative gene-environment correlation, in which genetically-influenced traits evoke non-random responses from the environment (Knafo & Jaffee, 2013), including through amygdala hypersensitivity, MAOA-L youth may accumulate more adverse experiences (e.g., parents, siblings, peers) over time. Thus, MAOA x parenting interactions may developmentally specific, in that they will be observable only as the adverse experiences are accumulated. This may partly contribute to the more consistent evidence that MAOA-L is associated with poor adolescent and adult outcomes in the presence of environmental adversity. For example, Pingault et al. (2013) followed 436 kindergarten children rated annually during pre-adolescence (age 6-12) and found no significant MAOA x family socioeconomic adversity. They suggested that null childhood findings may not contradict previous evidence of G x E interactions, and posited that,
with regard to MAOA, G x E effects may only emerge in adolescence or adulthood. Supporting this, a prospective longitudinal study into which followed children from age 6 into early adulthood, found a significant MAOA-L x corporal punishment interaction for CP (Edwards et al., 2010). However, earlier in development, amygdala hypersensitivity may contribute to children being more sensitive to threat and reward, including negative and positive parenting factors, which could underlie MAOA-L youth having less growth in CP than MAOA-H youth. Finally, future studies should include formal meditational models following significant G x E findings to explore the exact role that amygdala sensitivity and heightened threat perception play in the development of CP among youth with MAOA-L and MAOA-H genotypes. These studies would be positioned to evaluate whether children with MAOA-L display heightened amygdala activity and increased perception of life events, relative to peers with MAOA-H. This would a possible mediator of the association between MAOA-L and CP.

We emphasize several important limitations. First, corporal punishment was estimated from a single factor, comprised of only three items. There is the potential for multiple facets of negative parenting that may serve as forms of environmental adversity. For example, it is less clear if and how ineffective parenting and inconsistent discipline may interact with MAOA. Second, we used positive reinforcement and parental involvement as measures of positive parenting, however, positive reinforcement specifically may not be a purely positive construct, which then calls into question the direction of association between positive reinforcement and CP. For example, for positive reinforcement to increase prosocial behavior, reinforcement must be contingent upon other related important concepts such as labeled behavior and timely use of praise (Bell and Eyberg, 2002) whereas parents in this study simply self-reported the frequency of positive reinforcement. We therefore urge that the positive reinforcement x MAOA interaction
be interpreted cautiously given that it is unclear what youth behavior parents were reinforcing. Third, although this study was a two-year longitudinal study, the age range of participants (i.e., 6-9 years at baseline and 8-11 years at follow-up), the CP evidenced likely reflected early onset CP, which is known to have unique correlates and causal influences (Chronis et al., 2007); similarly, if putative G x E effects for CP are sensitive to development (e.g., age-dependent), the current study may not have been well-positioned to discern them. Finally, although the sample was drawn from a large and ethnically-diverse metropolitan city, the sample was not nationally representative.

In the well-characterized sample of school-age children, the association of Wave 1 parenting behavior and Wave 2 CP was moderated by offspring MAOA genotype. Specifically, our results suggest a main effect of MAOA genotype, indicating that children with MAOA-L displayed more growth of CP overall, however, in the fully saturated model with the interaction between parenting and MAOA genotype, there were significant interactions that were in opposition to our proposed hypothesis, and indicated that children with MAOA-H displayed more growth in CP at higher levels of Wave 1 corporal punishment and at lower levels of Wave 1 parental involvement, and Wave 1 parenting was less consequential for children with MAOA-L. This finding may reflect the specific types of parenting studied, and may additionally reflect the MAOA-related interactions that are observable during a specific developmental window. Future work should examine small developmental time periods and the change in the interactive effect of MAOA over time. At this developmental window, our findings indicate that while MAOA-L may confer risk overall, children with MAOA-H may be at higher risk for CP in the specific environmental contexts relative to their peers.
CHAPTER THREE: Study 1a: Additional Methods and Results for Study 1

The range of analyses and results, spanning multiple informants and constructs, necessitated that the manuscript above focus on key patterns with the results. However, for the sake of completeness, we included the following results that were not included in the submitted manuscript above, but were proposed in the original dissertation prospectus. There are five sub-sections, and the additional methods, analytic procedures, and results for each sub-section are detailed below. The sub-sections are: (1) additional measures of CP (DISC ODD and CD in addition to the results above, which include DBD ODD and CD and CBCL Aggressive Behavior and Rule Breaking Behavior); (2) additional observed parenting measures (observed negative talk and observed praise, in addition to the results above, which include APQ self-reported parenting), (3) associations between MAOA and CP in each Wave 1 and Wave 2, excluding any measure of parenting, (4) follow-up analyses testing for differential susceptibility, and (5) analyses of parenting x MAOA from a second sample (i.e., Add Health).

(1) Aim 1a: Diagnostic Interview Schedule for Children (DISC-IV) ODD and CD Outcomes

Methods:

Diagnostic Interview Schedule for Children—Fourth Edition (DISC-IV; Shaffer et al. 2000). We administered the DISC-IV, a fully structured interview keyed to DSM-IV diagnostic criteria (i.e., symptom levels, duration, persistence, age of onset, functional impairment), to each parent. The DISC-IV is widely used and diagnostic designations from the DISC have shown predictive validity in previous studies of ADHD youth (Lee, Lahey, Owens & Hinshaw, 2008). We separately analyzed symptom counts of ODD and CD, as symptoms of ODD and CD are only moderately correlated in our sample (r = 0.39, p < .01), and therefore may reflect different
presentations of CP.

Data Analytic Procedures:

First, given that, in boys, age was significantly associated with Wave 2 DISC CD symptoms, we controlled for age in predictions of DISC CD symptoms. Race was unrelated to DISC CD and DISC ODD symptoms in boys and was therefore excluded from analyses. In girls, age was unrelated to DISC CD and DISC ODD symptoms, and was therefore excluded. Race was significantly associated with DISC ODD symptoms in girls, and we therefore controlled for race in prediction of DISC ODD symptoms. Second, we controlled for Wave 1 ADHD diagnostic status (i.e., ADHD versus non-ADHD) to account for the association of ADHD with MAOA and CP (Marsh et al., 2008). Third, given their considerable skew, we utilized Poisson regression to evaluate the independent prediction of Wave 2 CP from Wave 1 parenting. We then reproduced the same models, consisting of the same covariates, parenting measures, and MAOA, but included the parenting x MAOA interactions.

Results:

Boys:

Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DISC CD symptoms

Using poisson regression and controlling for age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 DISC CD symptoms, Wave 1 involvement and MAOA genotype were each unrelated to growth in DISC CD symptoms (B = -0.01, SE = 0.04, p = 0.80 and B = 0.16, SE = 0.35, p = 0.65, respectively). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in DISC CD symptoms (B = -0.08, SE = 0.08, p = 0.31).

Controlling for age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1
DISC CD symptoms, Wave 1 positive reinforcement and MAOA genotype were each unrelated to growth in DISC CD symptoms ($B = -0.05$, $SE = 0.06$, $p = 0.42$ and $B = 0.31$, $SE = 0.35$, $p = 0.37$, respectively). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was unrelated to growth in DISC CD symptoms ($B = -0.10$, $SE = 0.13$, $p = 0.48$).

Controlling for age, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 DISC CD symptoms, Wave 1 corporal punishment and MAOA genotype were each unrelated to growth in DISC CD symptoms ($B = -0.01$, $SE = 0.15$, $p = 0.96$ and $B = 0.15$, $SE = 0.35$, $p = 0.68$, respectively). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in DISC CD symptoms ($B = 0.30$, $SE = 0.30$, $p = 0.32$).

**Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DISC ODD symptoms**

Using poisson regression and controlling for age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 DISC ODD symptoms, Wave 1 involvement and MAOA genotype were each unrelated to growth in DISC ODD symptoms ($B = 0.01$, $SE = 0.02$, $p = 0.55$ and $B = -0.10$, $SE = 0.15$, $p = 0.51$, respectively). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was significantly related to growth in DISC ODD symptoms ($B = -0.10$, $SE = 0.92$, $p = 0.01$). We followed up by examining simple effects for each genotype, and found that controlling for age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 DISC ODD symptoms, Wave 1 involvement, in children with MAOA-L, involvement was only marginally related to a decline in DISC ODD symptoms, and in children with MAOA-H, involvement was only marginally associated with growth in DISC ODD symptoms ($B = -0.06$, $SE = 0.03$, $p = 0.08$ and $B = 0.04$, $SE = 0.02$, $p = 0.08$, respectively).

Controlling for age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1
DISC ODD symptoms, Wave 1 positive reinforcement and MAOA genotype were each unrelated to growth in DISC ODD symptoms ($B = 0.02, SE = 0.03, p = 0.58$ and $B < -0.01, SE = 0.15, p = 0.99$, respectively). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was marginally related to growth in DISC ODD symptoms ($B = -0.10, SE = 0.05, p = 0.07$).

Controlling for age, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 DISC ODD symptoms, Wave 1 corporal punishment and MAOA genotype were each unrelated to growth in DISC ODD symptoms ($B = -0.01, SE = 0.06, p = 0.591$ and $B < -0.09, SE = 0.15, p = 0.55$, respectively). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in DISC ODD symptoms ($B = 0.07, SE = 0.12, p = 0.53$).

**Girls:**

*Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DISC CD symptoms*

Using poisson regression and controlling for Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 DISC CD symptoms, Wave 1 involvement and MAOA genotype were each unrelated to growth in DISC CD symptoms ($B = 0.09, SE = 0.09, p = 0.31$ and $B = -0.71, SE = 0.66, p = 0.28$, respectively). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in DISC CD symptoms ($B = 0.14, SE = 0.17, p = 0.39$).

Controlling for Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 DISC CD symptoms, Wave 1 positive reinforcement was marginally positively associated with growth in DISC CD symptoms ($B = 0.26, SE = 0.15, p = 0.09$), and MAOA genotype was marginally
associated with growth in DISC CD symptoms (B = -1.57, SE = 0.86, p = 0.07). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was unrelated to growth in DISC CD symptoms (B = 0.11, SE = 0.42, p = 0.79).

Controlling for Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 DISC CD symptoms, Wave 1 corporal punishment was unrelated to growth in DISC CD symptoms (B = -0.16, SE = 0.39, p = 0.68) and MAOA-H genotype was marginally associated with greater growth in DISC CD symptoms (B = -1.69, SE = 0.85, p = 0.05), than MAOA-L. In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in DISC CD symptoms (B = -0.95, SE = 0.68, p = 0.16).

**Associations of Wave 1 Parenting and MAOA genotype with Wave 2 DISC ODD symptoms**

Using poisson regression and controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 DISC ODD symptoms, Wave 1 involvement was marginally negatively associated with Wave 2 DISC ODD symptoms (B = -0.05, SE = 0.03, p = 0.08) and MAOA genotype was marginally associated with growth in DISC ODD symptoms (B = 0.55, SE = 0.32, p = 0.08). In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was unrelated to growth in DISC ODD symptoms (B = 0.09, SE = 0.07, p = 0.17).

Controlling for age, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 DISC ODD symptoms, Wave 1 positive reinforcement and MAOA genotype were each unrelated to growth in DISC ODD symptoms (B = 0.03, SE = 0.06, p = 0.60 and B = 0.07, SE = 0.31, p = 0.83, respectively). In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was unrelated to growth in DISC ODD symptoms (B = 0.01, SE = 0.13, p = 0.91).

Controlling for age, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 DISC
ODD symptoms, Wave 1 corporal punishment and MAOA genotype were each unrelated to growth in DISC ODD symptoms (B = -0.10, SE = 0.12, p = 0.42 and B = 0.50, SE = 0.35, p = 0.15, respectively). In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was unrelated to growth in DISC ODD symptoms (B = 0.03, SE = 0.28, p = 0.91).

(2) Aim 1a: Observed Parenting Behavior

Methods:

*Dyadic Parent Child Interaction Coding System (DPICS; Eyberg, Nelson, Duke, & Boggs, 2005).* The DPICS administration involved three standardized instructions, each given to the parent. First, the parent was instructed to follow the child’s lead in play (10 minutes). In the second situation, parents were told that they are responsible for leading play and must get their child to play according to their rules (10 minutes). In the last condition, parents were instructed to tell their children to clean up the toys in the playroom, without parental assistance (5 minutes). Of the three situations, the clean-up situation involved the greatest level of parental control.

Each of the three DPICS situations was coded based on parent verbalizations and child responses, including negative talk, commands, praise and child compliance. These categories are outlined in the DPICS manual (3rd edition) and are described in greater detail therein (see Eyberg, Nelson, Duke, & Boggs, 2005). Examples of parenting responses include *direct commands* (“you must sit down”), *labeled praise* (“you’re a good builder”), *unlabeled praise* (“good job”), and *negative talk* (“you’re being naughty”). Composite categories are commonly reported in the literature (Chronis-Tuscano et al., 2008; Eyberg et al., 2001), including negative parenting (negative talk), positive parenting (unlabeled and labeled praise), total commands (direct and
indirect commands) and child noncompliance (e.g., direct/indirect command non-compliance). For the present study, parenting will be analyzed using these composite categories. Categories will be scored by tallying the frequency counts (e.g., number of times parent used negative talk) of each behavior during 10 second intervals (see Chronis et al., 2007).

The DPICS categories have previously demonstrated good reliability. Bessner, Brestan, and Eyberg (2005) examined inter-rater reliability for the DPICS categories from 30 mother-child control dyads and 30 mother-child dyads (children aged 2 to 6) that were referred for treatment of oppositional defiant disorder (ODD). Pearson’s correlations between raters were highest for verbalizations (r = .63 to .99) and vocalizations (r = .54 to .93), and lowest for physical behaviors (r = .29 to .82). In addition, Cohen’s kappa estimates ranged between .46 and .90 for the parent categories, and between .51 and .87 for the child categories, reflecting moderate to substantial agreement. Psychometric properties of the DPICS have also been examined for school-aged kids. All parent and child categories demonstrated at least moderate reliability (kappa > .5) among parent-child dyads of abused and non-abused children, aged 8 to 12 (Eyberg et al., 2005). In addition, DPICS categories have also demonstrated test-retest reliability. Brinkmeyer (2005; cited in Eyberg et al., 2005) reported significant moderate correlations for the one-week test-retest reliability of the parent critical statement, parent praise, and child negative talk categories in 79 mother-child dyads (children aged 4 to 6) that were clinic-referred. Moderate to substantial inter-rater and test-retest reliability estimates have also been reported in other samples, suggesting that the DPICS categories are quite stable across different populations and age ranges (Brestan, Foote, & Eyberg, 2005; Hakman, Chaffin, Funderburk, Silovsky, 2009; McCabe, Yeh, Lau Argote, and Liang, 2010).

A team of six UCLA undergraduate student coders were trained in the use of the DPICS
manual (Eyberg et al., 2005) until 70% agreement was attained. Coders participated in full day of training, which included reviewing and discussing each coding category, practicing coding videotapes of the parent-child interactions, and going over quizzes. In addition, coders participated in weekly meetings to discuss areas of disagreement, code difficult sections together, and discuss ways to improve reliability. 20% of the tapes were coded by a second coder to assess inter-rater reliability.

Data Analytic Procedures:

Because MAOA is X-linked and its functional properties have not been discerned in females (Kim-Cohen et al., 2006), we analyzed males and females separately so that results can be compared with previous studies, as most previous studies have focused solely on males (Li & Lee, 2011). For boys, age was significantly associated with Wave 2 CBCL Aggressive Behavior and DISC CD symptoms, and we therefore controlled for age in predictions of Wave 2 CBCL Aggressive Behavior and DISC CD symptoms only. Race was significantly associated with Wave 2 CBCL Aggressive Behavior and marginally associated with CBCL Rule Breaking Behavior, and we therefore controlled for race in predictions of Wave 2 CBCL Aggressive Behavior and CBCL Rule Breaking Behavior.

For girls age was not significantly associated with any Wave 2 outcome measure, and therefore was excluded from analyses. Race was significantly associated with Wave 2 CBCL Rule Breaking Behavior, DISC ODD symptoms, and DBD ODD symptoms, and marginally associated with CBCL Aggressive Behavior, and we therefore controlled for race in predictions of Wave 2 CBCL Aggressive Behavior, CBCL Rule Breaking Behavior, DISC ODD symptoms and DBD ODD symptoms.

Second, we controlled for Wave 1 ADHD diagnostic status (i.e., ADHD versus non-
ADHD) to account for the association of ADHD with MAOA and CP (Marsh et al., 2008). Third, given their considerable skew, we utilized Poisson regression to evaluate the prediction of CP from MAOA genotype.

For teacher-rated measures (i.e. DBD ODD and CD), we used GEE, which allowed examination of both parent and teacher reports as a single repeated measures factor (i.e., an “informant” factor), thereby reducing the number of statistical tests and accounting for their inter-correlation. To combat missing teacher data, we employed data from multiple imputations (see details from Study 1 on page 18).

Results:

Associations of Wave 1 Parenting and MAOA with Wave 2 DISC CD symptoms

Using poisson regression, controlling for age, Wave 1 ADHD status, Wave 1 observed negative parenting, and Wave 1 DISC CD symptoms, Wave 1 observed praise and MAOA genotype were each unrelated to growth in DISC CD symptoms (B = -0.19, SE = 0.02, p = 0.33 and B = 0.39, SE = 0.36, p = 0.28, respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was unrelated to growth in DISC CD symptoms (B = 0.01, SE = 0.04, p = 0.85).

Controlling for age, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 DISC CD symptoms, Wave 1 observed negative parenting and MAOA genotype were each unrelated to growth in DISC CD symptoms (B = 0.01, SE = 0.02, p = 0.65 and B = 0.39, SE = 0.36, p = 0.28, respectively). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in DISC CD symptoms (B = -0.01, SE = 0.04, p = 0.74).
**Associations of Wave 1 Parenting and MAOA with Wave 2 DISC ODD symptoms**

Using poisson regression, controlling for age, Wave 1 ADHD status, Wave 1 observed negative parenting, and Wave 1 DISC ODD symptoms, Wave 1 observed praise and MAOA genotype were each unrelated to growth in DISC ODD symptoms ($B = 0.01$, $SE = 0.10$, $p = 0.10$ and $B = 0.09$, $SE = 0.17$, $p = 0.59$, respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was marginally related to growth in DISC ODD symptoms ($B = -0.03$, $SE = 0.02$, $p = 0.07$).

Controlling for age, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 DISC ODD symptoms, Wave 1 negative parenting and MAOA genotype were each unrelated to growth in DISC ODD symptoms ($B = 0.01$, $SE = 0.01$, $p = 0.26$ and $B = 0.09$, $SE = 0.17$, $p = 0.59$, respectively). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in DISC ODD symptoms ($B = -0.03$, $SE = 0.02$, $p = 0.22$).

**Associations of Wave 1 Parenting and MAOA with Wave 2 DBD CD symptoms**

Based on the GEE platform, controlling for Wave 1 ADHD status, informant, Wave 1 observed negative parenting, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 observed praise and MAOA genotype were each unrelated to growth in DBD CD symptoms ($B = 0.02$, $SE = 0.01$, $p = 0.17$ and $B = -0.19$, $SE = 0.23$, $p = 0.42$, respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was marginally related to growth in DBD CD symptoms ($B = -0.04$, $SE = 0.02$, $p = 0.07$).

Controlling for Wave 1 ADHD status, informant, Wave 1 observed praise, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 observed negative parenting and MAOA genotype were each unrelated to growth in DBD CD symptoms ($B = -0.01$, $SE = 0.01$, $p = 0.32$).
and \( B = -0.19, \ SE = 0.23, \ p = 0.42 \), respectively). In the fully saturated model, the Wave 1 observed negative parenting \( \times \) MAOA genotype interaction was marginally related to growth in DBD CD symptoms (\( B = 0.05, \ SE = 0.03, \ p = 0.08 \)).

**Associations of Wave 1 Parenting and MAOA with Wave 2 DBD ODD symptoms**

Again using the GEE platform, controlling for Wave 1 ADHD status, informant, Wave 1 observed negative parenting, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 observed praise and MAOA genotype were each unrelated to growth in DBD ODD symptoms (\( B < 0.01, \ SE = 0.01, \ p = 0.59 \) and \( B = 0.18, \ SE = 0.13, \ p = 0.15 \), respectively). In the fully saturated model, the Wave 1 observed praise \( \times \) MAOA genotype interaction was unrelated to growth in DBD ODD symptoms (\( B < -0.01, \ SE = 0.02, \ p = 0.77 \)).

Controlling for Wave 1 ADHD status, informant, Wave 1 observed praise, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 observed negative parenting and MAOA genotype were each unrelated to growth in DBD ODD symptoms (\( B = 0.01, \ SE = 0.01, \ p = 0.42 \) and \( B = 0.18, \ SE = 0.13, \ p = 0.15 \), respectively). In the fully saturated model, the Wave 1 observed negative parenting \( \times \) MAOA genotype interaction was unrelated to growth in DBD ODD symptoms (\( B < -0.01, \ SE = 0.01, \ p = 0.84 \)).

**Associations of Wave 1 Parenting and MAOA with Wave 2 CBCL Aggressive Behavior**

Using poisson regression and controlling for race, age, Wave 1 ADHD status, Wave 1 observed negative parenting, and Wave 1 Aggressive Behavior, Wave 1 observed praise was unrelated to growth in Aggressive Behavior (\( B < 0.001, \ SE < 0.01, \ p = 0.99 \)), and MAOA-L genotype was significantly associated with greater growth in Aggressive Behavior (\( B = 0.22, \ SE = 0.09, \ p = 0.01 \)), relative to MAOA-H. In the fully saturated model, the Wave 1 observed praise \( \times \) MAOA genotype interaction was unrelated to growth in Aggressive Behavior (\( B = 0.01, \ SE = \)
Controlling for race, age, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 Aggressive Behavior, Wave 1 observed negative parenting was significantly related to growth in Aggressive Behavior (B = 0.02, SE = 0.01, p = 0.01), and MAOA-L genotype was significantly associated with greater growth in Aggressive Behavior (B = 0.22, SE = 0.09, p = 0.01), relative to MAOA-H. In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in Aggressive Behavior (B = <0.01, SE = 0.01, p = 0.72).

Associations of Wave 1 Parenting and MAOA with Wave 2 CBCL Rule Breaking Behavior

Using poisson regression and controlling for race, age, Wave 1 ADHD status, Wave 1 observed negative parenting, and Wave 1 Rule Breaking Behavior, Wave 1 observed praise and MAOA genotype were unrelated to growth in Rule Breaking Behavior (B = 0.01, SE = 0.01, p =0.27 and B = 0.25, SE = 0.16, p = 0.12, respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was unrelated to growth in Rule Breaking Behavior (B = 0.01, SE = 0.02, p = 0.67).

Controlling for race, age, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 Rule Breaking Behavior, Wave 1 observed negative parenting and MAOA genotype were unrelated to growth in Rule Breaking Behavior (B < 0.01, SE = 0.01, p = 0.67 and B = 0.25, SE = 0.16, p = 0.12, respectively). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in Rule Breaking Behavior (B = 0.01, SE = 0.02, p = 0.76).
**Girls:**

**Associations of Wave 1 Parenting and MAOA with Wave 2 DISC CD symptoms**

Using poisson regression and controlling for ADHD status, Wave 1 observed negative parenting, and Wave 1 DISC CD symptoms, Wave 1 observed praise and MAOA genotype were each unrelated to growth in DISC CD symptoms ($B = -0.02$, $SE = 0.06$, $p = 0.76$ and $B = -0.39$, $SE = 0.75$, $p = 0.61$, respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was unrelated to growth in DISC CD symptoms ($B = -0.23$, $SE = 0.14$, $p = 0.10$).

Controlling for Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 DISC CD symptoms, Wave 1 observed negative parenting was marginally associated with Wave 2 DISC CD symptoms ($B = 0.07$, $SE = 0.04$, $p = 0.08$), and MAOA genotype was unrelated to growth in DISC CD symptoms ($B = -0.39$, $SE = 0.75$, $p = 0.61$). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in DISC CD symptoms ($B = -0.03$, $SE = 0.07$, $p = 0.63$).

**Associations of Wave 1 Parenting and MAOA with Wave 2 DISC ODD symptoms**

Using poisson regression and controlling for race, Wave 1 ADHD status, Wave 1 observed negative parenting, and Wave 1 DISC ODD symptoms, Wave 1 observed praise and MAOA genotype were each unrelated to growth in DISC ODD symptoms ($B = 0.01$, $SE = 0.02$, $p = 0.78$ and $B = 0.22$, $SE = 0.30$, $p = 0.47$, respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was unrelated to growth in DISC ODD symptoms ($B = 0.01$, $SE = 0.05$, $p = 0.76$).

Controlling for race, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 DISC ODD symptoms, Wave 1 negative parenting and MAOA genotype were each unrelated to
growth in DISC ODD symptoms (B = -0.02, SE = 0.02, p = 0.25 and B = 0.22, SE = 0.30, p = 0.47, respectively). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in DISC ODD symptoms (B = -0.05, SE = 0.04, p = 0.15).

**Associations of Wave 1 Parenting and MAOA with Wave 2 DBD CD symptoms**

Based on the GEE platform, controlling for race, Wave 1 ADHD status, informant, Wave 1 observed negative parenting, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 observed praise was unrelated to DBD CD symptoms (B = -0.06, SE = 0.04, p = 0.13), and MAOA-L genotype was significantly associated with greater growth in DBD CD symptoms (B = 1.18, SE = 0.53, p = 0.03), relative to MAOA-H. In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was unrelated to growth in DBD CD symptoms (B = -0.11, SE = 0.08, p = 0.16).

Controlling for race, Wave 1 ADHD status, informant, Wave 1 observed praise, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 observed negative parenting was unrelated to DBD CD symptoms (B < -0.01, SE = 0.05, p = 0.95) and MAOA-L genotype was significantly associated with a greater growth in DBD CD symptoms and (B = 1.18, SE = 0.53, p = 0.03), relative to MAOA-H. In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype significantly predicted growth in DBD CD symptoms (B = -0.13, SE = 0.05, p = 0.02). We followed up by examining simple effects for each genotype, but given the limited variability of CD symptoms within girls with MAOA-H, we could not calculate the simple slope for MAOA-H.

**Associations of Wave 1 Parenting and MAOA with Wave 2 DBD ODD symptoms**

Again based on the GEE platform, controlling for race, Wave 1 ADHD status, informant,
Wave 1 observed negative parenting, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 observed praise was significantly associated with a decline in DBD ODD symptoms (B = -0.07, SE = 0.02, p < 0.001) and MAOA genotype was marginally associated with growth in DBD ODD symptoms (B = 0.52, SE = 0.30, p = 0.08). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was unrelated to growth in DBD ODD symptoms (B = -0.01, SE = 0.04, p = 0.90).

Controlling for race, Wave 1 ADHD status, informant, Wave 1 observed praise, and Wave 1 teacher and parent rated DBD ODD symptoms, Wave 1 observed negative parenting was unrelated to growth in DBD ODD symptoms (B = -0.01, SE = 0.02, p = 0.81) and MAOA genotype was marginally associated with growth in DBD ODD symptoms (B = 0.52, SE = 0.30, p = 0.08). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in DBD ODD symptoms (B = -0.03, SE = 0.02, p = 0.15).

**Associations of Wave 1 Parenting and MAOA with Wave 2 CBCL Aggressive Behavior**

Using poisson regression and controlling for race, Wave 1 ADHD status, Wave 1 observed negative parenting, and Wave 1 Aggressive Behavior, Wave 1 observed praise and MAOA genotype were each unrelated to growth in Aggressive Behavior (B = -0.01, SE = 0.01, p = 0.37 and B = -0.18, SE = 0.14, p = 0.20, respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was marginally associated with growth in Aggressive Behavior (B = 0.04, SE = 0.02, p = 0.05).

Controlling for race, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 Aggressive Behavior, Wave 1 observed negative parenting and MAOA genotype were each unrelated to growth in Aggressive Behavior (B = 0.01, SE = 0.01, p = 0.16 and B= -0.18, SE =
0.14, \( p = 0.20 \), respectively). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was unrelated to growth in Aggressive Behavior (B = -0.02, SE = 0.02, \( p = 0.14 \)).

**Associations of Wave 1 Parenting and MAOA with Wave 2 CBCL Rule Breaking Behavior**

Using poisson regression and controlling for race, Wave 1 ADHD status, Wave 1 observed negative parenting, and Wave 1 Rule Breaking Behavior, Wave 1 observed praise and MAOA genotype were each unrelated to growth in Rule Breaking Behavior (B = -0.01, SE = 0.02, \( p = 0.60 \) and B = -0.26, SE = 0.27, \( p = 0.34 \), respectively). In the fully saturated model, the Wave 1 observed praise x MAOA genotype interaction was unrelated to growth in Rule Breaking Behavior (B = 0.01, SE = 0.04, \( p = 0.84 \)).

Controlling for race, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 Rule Breaking Behavior, Wave 1 observed negative parenting and MAOA genotype were unrelated to growth in Rule Breaking Behavior (B = 0.02, SE = 0.01, \( p = 0.17 \) and B = -0.26, SE = 0.27, \( p = 0.34 \), respectively). In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was significantly associated with growth in Rule Breaking Behavior (B = -0.06, SE = 0.03, \( p = 0.03 \)). We followed up by examining simple effects for each genotype, and found that controlling for race, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 Rule Breaking Behavior, in children with MAOA-L, Wave 1 observed negative parenting was unrelated to growth in CBCL Rule Breaking Behavior and in children with MAOA-H, Wave 1 observed negative parenting was significantly associated with an increase in CBCL Rule Breaking Behavior (B < 0.01, SE = 0.02, \( p = 0.97 \) and B = 0.04, SE = 0.02, \( p = 0.02 \), respectively).
(3) Aim 1a: Associations of MAOA and Wave 1 and Wave 2 CP:

Methods:

The methods are the same as described for Study 1 on pages 13-17 and for 1a on page 33.

Data Analytic Procedures:

Because MAOA is X-linked and its functional properties have not been discerned in females (Kim-Cohen et al., 2006), we analyzed males and females separately so that results can be compared with previous studies, as most previous studies have focused solely on males (Li & Lee, 2011). For boys, age and race were unrelated with Wave 1 CP and were therefore excluded from Wave 1 analyses whereas age was significantly associated with Wave 2 CBCL Aggressive Behavior and DISC CD symptoms. Thus, we controlled age in predictions of these Wave 2 outcomes only. Race was significantly associated with Wave 2 CBCL Aggressive Behavior and marginally associated with CBCL Rule-Breaking Behavior, and we therefore controlled for race in predictions of Wave 2 CBCL Aggressive Behavior and CBCL Rule Breaking Behavior.

For girls, age was marginally associated with Wave 1 DBD ODD symptoms, and therefore, we controlled for age in predictions of Wave 1 DBD ODD symptoms. Race was significantly associated with Wave 1 CBCL Rule Breaking Behavior, CBCL Aggressive Behavior and DISC ODD symptoms, and marginally associated with DBD ODD symptoms and we therefore controlled for race in predictions of Wave 1 CBCL Rule Breaking Behavior, CBCL Aggressive Behavior, DISC ODD symptoms and DBD ODD symptoms. Age was not significantly associated with any Wave 2 outcome measure, and therefore was excluded from analyses. Race was significantly associated with Wave 2 CBCL Rule Breaking Behavior, DISC ODD symptoms, and DBD ODD symptoms, and marginally associated with CBCL Aggressive Behavior, and we therefore controlled for race in predictions of Wave 2 CBCL Aggressive
Behavior, CBCL Rule-Breaking Behavior, DISC ODD symptoms and DBD ODD symptoms.

Second, we controlled for Wave 1 ADHD diagnostic status (i.e., ADHD versus non-ADHD) to account for the association of ADHD with MAOA and CP (Marsh et al., 2008). Third, given their considerable skew, we utilized Poisson regression to evaluate the prediction of CP from MAOA genotype.

For teacher-rated measures (i.e. DBD ODD and CD), we used GEE, which allowed examination of both parent and teacher reports as a single repeated measures factor (i.e., an “informant” factor), thereby reducing the number of statistical tests and accounting for their inter-correlation. To combat missing teacher data, we employed data from multiple imputations (see details from Study 1 on page 18).

**Boys:**

Using poisson regression, controlling for Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 DISC CD symptoms ($B = 0.09$, $SE = 0.24$, $p = 0.72$). Similarly, controlling for Wave 2 age and ADHD status, MAOA genotype was unrelated to Wave 2 DISC CD symptoms ($B = 0.42$, $SE = 0.31$, $p = 0.18$).

Controlling for Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 DISC ODD symptoms ($B = 0.03$, $SE = 0.11$, $p = 0.79$). Similarly, controlling for Wave 2 ADHD status, MAOA genotype was unrelated to Wave 2 DISC ODD symptoms ($B = 0.10$, $SE = 0.14$, $p = 0.46$).

Controlling for Wave 1 ADHD status, MAOA genotype was marginally associated with Wave 1 DBD CD symptoms ($B = 0.37$, $SE = 0.29$, $p = 0.05$). Controlling for Wave 2 ADHD status, MAOA genotype was unrelated to Wave 2 DBD CD symptoms ($B = 0.09$, $SE = 0.26$, $p = 0.46$).
Controlling for Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 DBD ODD symptoms \( (B = 0.03, \ SE = 0.17, \ p = 0.85) \). Controlling for Wave 2 ADHD status, MAOA genotype was unrelated to Wave 2 DBD ODD symptoms \( (B = 0.10, \ SE = 0.12, \ p = 0.43) \).

Controlling for Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 Aggressive Behavior \( (B < 0.01, \ SE = 0.06, \ p = 0.95) \). In contrast, controlling for race, Wave 2 age and ADHD status, MAOA-L genotype was significantly associated with higher levels of Wave 2 Aggressive Behavior \( (B = 0.29, \ SE = 0.08, \ p < 0.001) \), relative to MAOA-H.

Using poisson regression, controlling for Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 Rule Breaking Behavior \( (B = -0.06, \ SE = 0.10, \ p = 0.52) \). In contrast, controlling for race, Wave 2 age and ADHD status, MAOA-L genotype was significantly associated with higher levels of Wave 2 Rule Breaking Behavior \( (B = 0.29, \ SE = 0.13, \ p = 0.03) \), relative to MAOA-H.

**Girls:**

Using poisson regression, controlling for Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 DISC CD symptoms \( (B = 0.31, \ SE = 0.38, \ p = 0.41) \). Similarly, controlling for ADHD status, MAOA genotype was unrelated to Wave 2 DISC CD symptoms \( (B = -0.68, \ SE = 0.65, \ p = 0.29) \).

Controlling for race and Wave 1 ADHD status, MAOA genotype was marginally associated with Wave 1 DISC ODD symptoms \( (B = 0.35, \ SE = 0.20, \ p = 0.08) \). In contrast, controlling for race and Wave 2 ADHD status, MAOA genotype was unrelated to Wave 2 DISC ODD symptoms \( (B = 0.35, \ SE = 0.26, \ p = 0.18) \).
Controlling for race, Wave 1 ADHD status, MAOA genotype was significantly associated with Wave 1 DBD CD symptoms ($B = 1.17$, $SE = 0.45$, $p = 0.01$). Controlling for race, Wave 2 ADHD status, MAOA-L genotype was significantly associated with higher levels of Wave 2 DBD CD symptoms ($B = 0.80$, $SE = 0.33$, $p = 0.02$), relative to MAOA-H.

Controlling for race, Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 DBD ODD symptoms ($B = 0.33$, $SE = 0.25$, $p = 0.18$). Controlling for race, Wave 2 ADHD status, MAOA genotype was unrelated to Wave 2 DBD ODD symptoms ($B = 0.29$, $SE = 0.28$, $p = 0.30$).

Controlling for race and Wave 1 ADHD status, MAOA-L genotype was significantly associated with higher levels of Wave 1 Aggressive Behavior ($B = 0.25$, $SE = 0.11$, $p = 0.02$), relative to MAOA-H. In contrast, controlling for race and ADHD status, MAOA genotype unrelated to Wave 2 Aggressive Behavior ($B = -0.01$, $SE = 0.14$, $p = 0.94$).

Controlling for race and Wave 1 ADHD status, MAOA genotype was unrelated to Wave 1 Rule Breaking Behavior ($B = 0.10$, $SE = 0.7$, $p = 0.58$). Similarly, controlling for race and ADHD status, MAOA genotype was unrelated to Wave 2 Rule Breaking Behavior ($B = -0.08$, $SE = 0.25$, $p = 0.74$).

(4) Aim 1a: Tests for differential susceptibility:

In the fully saturated model, the Wave 1 corporal punishment x MAOA genotype interaction was significantly related to growth in Aggressive Behavior in boys ($B = -0.14$, $SE = 0.06$, $p = 0.02$). We followed up by examining simple effects for each genotype, and found that controlling for age, race, Wave 1 ADHD status, Wave 1 positive parenting, and Wave 1 Aggressive Behavior, in children with MAOA-L, punishment was unrelated to growth in
Aggressive Behavior, and in children with MAOA-H, punishment significantly predicted growth in Aggressive Behavior (B = -0.03, SE = 0.05, p = 0.54 and B = 0.09, SE = 0.04, p = 0.02, respectively).

To evaluate for differential susceptibility in the presence of the significant interaction, we compared MAOA-L versus MAOA-H youth at the extreme ends of corporal punishment by calculating predicted values of Aggressive Behavior using the margins command in STATA (Version 12; StataCorp, 2011) in order to test for presence of a true crossover effect. These comparisons were made at each the lowest and highest values for corporal punishment, with all covariates held at the mean with a chi-square test to evaluate the significance of the difference. While children with MAOA-L differed from children with MAOA-H at low levels of corporal punishment ($\chi^2 = 8.98, p < 0.01$), there was no significant difference at high levels of corporal punishment ($\chi^2 = 2.35, p = 0.13$), and thus, the interaction was not suggestive of differential susceptibility.

In the fully saturated model, the Wave 1 involvement x MAOA genotype interaction was significantly related to growth in Aggressive Behavior (B = 0.06, SE = 0.03, p = 0.04). We followed up by examining simple effects for each genotype, and found that controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Aggressive Behavior, in children with MAOA-L, involvement was unrelated to growth in CBCL Aggressive Behavior symptoms and in children with MAOA-H, involvement was significantly associated with a decrease in CBCL Aggressive Behavior (B < 0.01, SE = 0.02, p = 0.87 and B = -0.05, SE = 0.02, p = 0.04, respectively).

To evaluate for differential susceptibility in the presence of the significant interaction, we compared MAOA-L versus MAOA-H youth at the extreme ends of corporal punishment by
calculating predicted values of Aggressive Behavior using the margins command in STATA (Version 12; StataCorp, 2011) in order to test for presence of a true crossover effect. These comparisons were made at each the lowest and highest values for parental involvement, with all covariates held at the mean with a chi-square test to evaluate the significance of the difference. While children with MAOA-L differed from children with MAOA-H at low levels of parental involvement ($\chi^2 = 6.15, p = 0.01$), there was no significant difference at high levels of parental involvement ($\chi^2 = 1.65, p = 0.20$), and thus, the interaction was not suggestive of differential susceptibility.

In the fully saturated model, the Wave 1 observed negative parenting x MAOA genotype interaction was significantly associated with growth in Rule Breaking Behavior ($B = -0.06$, SE = 0.03, $p = 0.03$). We followed up by examining simple effects for each genotype, and found that controlling for race, Wave 1 ADHD status, Wave 1 observed praise, and Wave 1 Rule Breaking Behavior, in children with MAOA-L, Wave 1 observed negative parenting was unrelated to growth in CBCL Rule Breaking Behavior and in children with MAOA-H, Wave 1 observed negative parenting was significantly associated with an increase in Rule Breaking Behavior symptoms ($B < 0.01$, SE = 0.02, $p = 0.97$ and $B = 0.04$, SE = 0.02, $p = 0.02$, respectively).

To evaluate for differential susceptibility in the presence of the significant interaction, we compared MAOA-L versus MAOA-H youth at the extreme ends of observed negative talk by calculating predicted values of Rule Breaking Behavior using the margins command in STATA (Version 12; StataCorp, 2011) in order to test for presence of a true crossover effect. These comparisons were made at each the lowest and highest values for observed negative talk, with all covariates held at the mean with a chi-square test to evaluate the significance of the difference. There was a marginal difference between children with MAOA-L and MAOA-H at high levels
of observed negative talk ($\chi^2 = 3.65, p = 0.06$) and no significant difference at low levels of observed negative talk ($\chi^2 = 0.70, p = 0.40$), and thus, the interaction was not consistent with the criteria necessary for differential susceptibility.

In the fully saturated model, the Wave 1 positive reinforcement x MAOA genotype interaction was significantly related to growth in Rule Breaking Behavior ($B = -0.21, SE = 0.10, p = 0.04$). We followed up by examining simple effects for each genotype, and found that controlling for race, Wave 1 ADHD status, Wave 1 corporal punishment, and Wave 1 Rule Breaking Behavior, in children with MAOA-L, Wave 1 positive reinforcement was unrelated to growth in CBCL Rule Breaking Behavior, and in children with MAOA-H, positive reinforcement significantly associated with an increase in CBCL Rule Breaking Behavior ($B = -0.01, SE = 0.09, p = 0.94$ and $B = 0.15, SE = 0.06, p = 0.01$, respectively).

To evaluate for differential susceptibility in the presence of the significant interaction, we compared MAOA-L versus MAOA-H youth at the extreme ends of positive reinforcement by calculating predicted values of Rule Breaking Behavior using the margins command in STATA (Version 12; StataCorp, 2011) in order to test for presence of a true crossover effect. These comparisons were made at each the lowest and highest values for positive reinforcement, with all covariates held at the mean with a chi-square test to evaluate the significance of the difference. While children with MAOA-L differed from children with MAOA-H at high levels of positive reinforcement ($\chi^2 = 5.43, p = 0.02$), there was no significant difference at low levels of positive reinforcement ($\chi^2 = 0.82, p = 0.37$), and thus, the interaction was not suggestive of differential susceptibility.
(5) Aim 1a: Add Health (Sample 2) Results

Participants:

The National Longitudinal Study of Adolescent Health (Add Health; Harris 2008) ascertained a stratified random sample of youth from U.S. high schools. Details of the study design can be obtained at http://www.cpc.unc.edu/projects/addhealth. 20,745 adolescents were interviewed at Wave 1 (grades 7-12, ages 12-20 years during the 1994-1995 school year; 47.5% male). Wave 2 interviews were conducted with 14,738 adolescents, two years later. Wave 3 includes 15,197 young adults 7-8 years after Wave 1. We analyzed the genetic subsample (obtained from full siblings and twins only), which consists of 2,488 adolescents (48% male). At Wave 1, adolescents with genetic data were slightly younger (15.7 years vs. 15.6 years, respectively) \[F(1,20345) = 5.65, p < .001\] than adolescents without genetic data, but they did not differ significantly with respect to gender \[F(1,20345 = 2.81, p = .09\] or family income \[F(1,20345) = 2.35, p = .13\]. Finally, although the genetic subsample was ethnically diverse (57.5% Caucasian, 14.3% Hispanic, 18.1% African-American, 7.4% Asian, 1.7% Native American, and 0.9% “Other”), it is not a nationally representative sample.

Genotyping

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. Primer sequences for the 30 bp VNTR in the promoter region of the MAOA open reading frame were: forward, 50-ACAGCCTGACCCGTGGAGAAG-30 (fluorescently labeled) and reverse, 50-GAACGTGACGCTCCATTCCCGA-30. PCR products included five possible fragment sizes: 291, 321, 336, 351, and 381bp (2–5 repeats). The 3-repeat allele is associated with less transcriptional efficiency and low MAOA activity (L) compared to the 4-repeat allele, which is associated with
greater transcriptional efficiency and high activity (H) (Sabol et al., 1998). We coded MAOA genotypes according to their efficiency (Lawson et al., 2003): the low-activity group (MAOA-L; n = 719, 28%) the heterozygous group (n = 548, 22%), and the high activity group (MAOA-H; n = 1148, 45%) (Sjoberg et al., 2007). Given the rarity of the 2-, 3.5- and 5-repeat alleles in the population they were excluded.

Measures

CP was ascertained during at-school and in-home interviews that were conducted at Waves 1 and 2. During Wave 1, in the at-school interview, items including alcohol use, truancy, dangerous behavior and physical violence were assessed. During the at-home interview, questionnaires on each fighting/violence and delinquency were administered. Scales of overt and covert aggression were created from items from these interviews, consistent with Lee (2011). At Wave 2, these items were re-administered and will be analyzed separately from Wave 1 given the instability of CP across development, especially during adolescence and early adulthood.

Maltreatment: Youth retrospectively reported during an in-home interview at Wave III the frequency of each of the following events prior to age 12: (1) parents or adult-caregivers not taking care of the respondent’s basic needs (e.g., hygiene, food/clothing), (2) been slapped, hit or kicked by parents or adult care-givers, and (3) been touched in a sexual way, forced to touch someone else in a sexual way, or forced to have sexual relations with a parent or adult caregiver. Following expert recommendations (Haberstick et al., 2005), if an event occurred at least once, it was scored as positive. 64.8% of youth reported no mistreatment history and 35.2% reported at least one episode. Individuals with genotype data did not differ in maltreatment from individuals without those data [F(1,14033) = .04, p = .84].
**Parental Support/Closeness**: Parental Support/Closeness was assessed from an in-home structured interview at Waves I, using thirteen items based on items used in Li, Berk and Lee (2013). The 13 items measure youth’s perceived familial support, including parental closeness and feeling loved and wanted by family members. Respondents rated their perceived level of parent and family support on an ordinal scale from 1 (not at all) to 5 (very much). Items included “How close do you feel to your mother/father?” and “How much do you think she/he cares about you?”

**Data Analytic Procedures:**

Because MAOA is X-linked and its functional properties have not been discerned in females (Kim-Cohen et al., 2006), we analyzed males and females separately so that results can be compared with previous studies, as most previous studies have focused solely on males (Li & Lee, 2011). First, we evaluated age and race-ethnicity as potential covariates. Age was not significantly associated with overt or covert aggression in males or females, and therefore was excluded from all analyses. Given that race was associated with overt aggression in both males and females, we controlled for race in predictions of overt aggression only. We employed a Poisson distribution, given the highly skewed data. We employed generalized estimating equations (GEE; Hardin, 2005) to examine Wave 1 and Wave 2 CP data as a single repeated measures factor, which extends the generalized linear models methodology, as it does not assume independence between Wave 1 and Wave 2 data. GEE reduces the number of statistical tests and accounts for their inter-correlation.

**Results:**

**Boys:**

*Associations of Maltreatment, Parental Support/Closeness and MAOA genotype with Overt*
**Aggression:**

In the model that included youth MAOA genotype and a repeated measure of Wave 1 and Wave 2 overt aggression, controlling for race, youth MAOA genotype was not significantly associated with overt aggression (B = -0.16, SE = 0.15 \( p = 0.29 \)).

To examine the association of MAOA genotype and retrospectively rated maltreatment with overt aggression, we used a repeated measure of Wave 1 and Wave 2 overt aggression. Controlling for race, maltreatment was positively associated with overt aggression (B = 0.08, SE = 0.02, \( p = 0.001 \)) and MAOA genotype was unrelated to overt aggression (B = -0.01, SE = 0.21 \( p = 0.94 \)). In the fully saturated model, the Wave 1 maltreatment x MAOA genotype interaction was unrelated to overt aggression (B = -0.04, SE = 0.05, \( p = 0.45 \)).

To examine the association of MAOA genotype and self-reported perception of parental support/closeness with overt aggression, we used a repeated measure of Wave 1 and Wave 2 overt aggression. Controlling for race, parental support/closeness was unrelated to overt aggression (B = -0.01, SE = 0.02, \( p = 0.62 \)) and MAOA genotype was unrelated to overt aggression (B = 0.20, SE = 0.18 \( p = 0.25 \)). In the fully saturated model, the Wave 1 parental support/closeness x MAOA genotype interaction was unrelated to overt aggression (B = 0.05, SE = 0.05, \( p = 0.36 \)).

**Associations of Parenting and MAOA with Covert Aggression:**

In the model that included youth MAOA genotype and a repeated measure of Wave 1 and Wave 2 covert aggression, MAOA genotype was not significantly associated with a covert aggression, (B = -0.13, SE = 0.10 \( p = 0.21 \)).

To examine the association of MAOA genotype and retrospectively rated maltreatment with covert aggression, we used a repeated measure of Wave 1 and Wave 2 covert aggression.
Maltreatment was positively associated with covert aggression (B = 0.08, SE = 0.02, p < 0.001) and MAOA genotype was unrelated to covert aggression (B = -0.16, SE = 0.11, p = 0.16). In the fully saturated model, the Wave 1 maltreatment x MAOA genotype interaction was unrelated to covert aggression (B = -0.01, SE = 0.03, p = 0.75).

To examine the association of MAOA genotype and self-reported perception of parental support/closeness with overt aggression, we used a repeated measure of Wave 1 and Wave 2 covert aggression, parental support/closeness was unrelated to covert aggression (B = -0.01, SE = 0.02, p = 0.54) and MAOA genotype was marginally associated with covert aggression (B = 0.27, SE = 0.14, p = 0.06). In the fully saturated model, the Wave 1 maltreatment x MAOA genotype interaction was unrelated to covert aggression (B = 0.05, SE = 0.04, p = 0.29).

Girls:

Associations of Maltreatment, Parental Support/Closeness and MAOA with Overt Aggression:

In the model that included youth MAOA genotype and a repeated measure of Wave 1 and Wave 2 overt aggression, controlling for race, youth MAOA genotype was unrelated to overt aggression (B < -0.01, SE = 0.19, p = 0.99).

To examine the association of MAOA genotype and retrospectively rated maltreatment with overt aggression, we used a repeated measure of Wave 1 and Wave 2 overt aggression. Controlling for race, maltreatment was positively associated with overt aggression (B = 0.10, SE = 0.03, p < 0.001) and MAOA genotype was unrelated to overt aggression (B = 0.03, SE = 0.20, p = 0.86). In the fully saturated model, the Wave 1 maltreatment x MAOA genotype interaction was unrelated to overt aggression (B = -0.02, SE = 0.05, p = 0.71).

To examine the association of MAOA genotype and self-reported perception of parental support/closeness with overt aggression, we used a repeated measure of Wave 1 and Wave 2
overt aggression. Controlling for race, parental support/closeness was unrelated to overt aggression (B = 0.00, SE = 0.01, p = 0.99) and MAOA genotype was unrelated to overt aggression (B = 0.21, SE = 0.24, p = 0.37). In the fully saturated model, the Wave 1 parental support/closeness x MAOA genotype interaction was marginally related to overt aggression (B = -0.09, SE = 0.05, p = 0.08).

**Associations of Maltreatment, Parental Support/Closeness and MAOA genotype with Covert Aggression:**

In the model that included youth MAOA genotype and a repeated measure of Wave 1 and Wave 2 covert aggression, MAOA genotype was unrelated to covert aggression (B = -0.04, SE = 0.16, p = 0.83).

To examine the association of MAOA genotype and retrospectively rated maltreatment with covert aggression, we used a repeated measure of Wave 1 and Wave 2 covert aggression. Maltreatment was positively associated with covert aggression (B = 0.07, SE = 0.02, p = 0.002) and MAOA genotype was unrelated to covert aggression (B = 0.01, SE = 0.17, p = 0.95). In the fully saturated model, the Wave 1 maltreatment x MAOA genotype interaction was unrelated to covert aggression (B = -0.06, SE = 0.04, p = 0.17).

To examine the association of MAOA genotype and self-reported perception of parental support/closeness with a covert aggression, we used a repeated measure of Wave 1 and Wave 2 covert aggression. Parental support/closeness was unrelated to covert aggression (B = 0.01, SE = 0.01, p = 0.10) and MAOA genotype was unrelated to covert aggression (B = 0.27, SE = 0.20, p = 0.17). In the fully saturated model, the Wave 1 parental support/closeness x MAOA genotype interaction was unrelated to covert aggression (B = -0.01, SE = 0.02, p = 0.54).
Conduct problems (CP) consist of diverse aggressive and rule-breaking behavior including noncompliance, physical aggression, delinquency, substance problems, and relational aggression (Hinshaw and Lee, 2003). CP are often persistent: parent-rated oppositional behavior was highly stable from age 2 through age 6 in a large Canadian birth cohort (Petitclerc, et al., 2009) and early aggression prospectively predicted adolescent and adult CP in a large community-based sample (Odgers et al., 2008). Similarly, more than 50% of children with oppositional defiant disorder (ODD) or conduct disorder (CD) are similarly diagnosed later in development Reid et al., 2004; Petitclerc et al., 2009). Negative outcomes associated with early CP include continued CP (i.e., homotypic continuity), but also anxiety, depression, substance problems, and school failure (i.e., multifinality), as well as functional impairment (e.g., accidental injuries; Reid et al., 2004; Odgers et al., 2007). Given its public health significance, identifying early predictors of CP is necessary to facilitate the development and implementation of new interventions.

Individual differences in negative parenting behavior (i.e., harsh/punitive, inconsistent, poor monitoring) reliably predict CP across development (DeKlyen, et al., 1998; Robison, et al., 2005; Calkins and Keane, 2009). Parenting behavior accounted for 11% of the variance in delinquency, and negative parenting was the most robust predictor of CP in a large meta-analysis (Hoeve et al., 2009). Similarly, negative parenting with 3 year-old children was among the strongest predictors of CP at school entry (Campbell, et al., 2000). Interestingly, despite its centrality to interventions for CP and its role in promoting offspring development (Dornbusch, et al., 1987; Reid, 1987; Steinberg, 2001; Gardner, et al., 2003), there are relatively few naturalistic
studies of positive parenting and CP. Positive parenting, consisting of praise, warmth, consistency, and involvement (Forgatch and DeGarmo, 1999; Gardner, et al., 1999; Hinshaw et al., 2000), is factorially independent from negative parenting, attenuates predictions of CP from multiple risk factors, and prevents persistent CP (Forgatch and DeGarmo, 1999; Gardner, et al., 2010). Among 4-6 year-old children, controlling for negative parenting behavior and baseline attention-deficit/hyperactivity disorder (ADHD) and CD, observed positive parenting behavior inversely predicted adolescent CD (Chronis et al., 2007). Similarly, beyond negative parenting, positive parenting prospectively predicted teacher-ratings of grade 6 adjustment (Pettit, et al., 1997). Thus, although positive parenting predicts CP independent of its association with negative parenting, most studies have ignored this important distinction, particularly with respect to predictions of different forms of CP (e.g., delinquency vs. physical aggression).

Integration of developmental perspectives is essential to adequately characterize CP. For example, defiant behavior often begins in school-age children, but it often decreases over time whereas status violations and non-aggressive behavior (e.g., lying, truancy) increase reliably across adolescence (Maughan, et al., 2004; Nock, et al., 2007). Similarly, causal influences are also sensitive to development: early-onset CD is associated with numerous risk factors including elevated comorbidity, severe neuropsychological deficits, and diminished amygdala reactivity on recognition tasks of faces conveying emotional states (e.g., disgust, happiness) (Fairchild et al., 2009; Passamonti et al., 2010). Alternatively, adolescent-onset CP is less severe, characterized by fewer risk factors, and is considered more developmentally normative, although outcomes were still worse relative to controls (Roisman, et al., 2010). Thus, prosecution of the origins and consequences of CP necessitates careful consideration of developmental influences.

There are also likely to be synergistic relationships between positive and negative
parenting behavior with child constitutional factors with respect to the development of CP.

Callosum-emotional (CU) traits, defined as individual differences in the propensity to display
and experience empathy and guilt, positively predict growth in CP (Enebrink, et al., 2005; Viding, et al., 2005; Blair, et al., 2006; Moran et al., 2009). Reflecting evidence that there is substantial heterogeneity among children with CD with respect to clinical presentation, etiology, and long-term outcomes (Frick, 2006), the Diagnostic and Statistical Manual of Mental Disorders (DSM 5) includes “presence of CU traits” as a specifier for CD. By virtue of their predictive validity across multiple instruments (Salekin, 2008), CU traits may designate a clinically distinct group of stable aggressive and antisocial children (Frick and Ellis 1999; Frick and White 2008). As mentioned previously, CU traits prospectively predicted 3-year growth in CP and emotional problems (Moran et al., 2009) and psychopathic traits, including CU traits, prospectively predicted 12% of the variance in adolescent delinquency severity, controlling for initial ADHD and ODD/CD (Piatigorsky and Hinshaw, 2004). By virtue of their predictive validity across multiple instruments (Salekin, 2008), CU traits appear to designate a distinct group of persistently aggressive and antisocial children (Frick and Ellis, 1999; Frick and White, 2008; Frick, Ray, Thornton & Kahn, 2013) with potentially unique causal influences. For example, the heritability of CP in children with high CU traits significantly exceeded youth with low CU traits ($h^2 = .81$ and .34, respectively) (Viding et al., 2005; Viding et al., 2013). Consistent with the centrality of affective dysregulation in psychopathic traits (Pardini and Loeber, 2007), youth with high CU traits displayed less amygdala activity in response to fearful faces than controls, despite similar patterns of neural activation in response to angry and neutral facial expressions (Marsh et al. 2008). This evidence approximates nonhuman primates where amygdala dysfunction was significantly associated with deficient fear processing/recognition
Thus, as suggested by its severe trajectory of aggression, elevated heritability, and unique neural correlates, CU should feature prominently in studies of emergent CP overall and aggressive behavior in particular.

CU traits also reveal different patterns of association with parenting behavior with respect to predictions of CP. In a study of 166 participants, CU traits moderated the association of ineffective parenting and CP such that negative parenting was positively associated with CP among children with low levels of CU traits, but CP in youth with elevated was independent of ineffective parenting behavior (Wootton et al. 1997). Oxford, Cavell and Hughes (2003) similarly reported that ineffective parenting positively correlated with peer- and teacher-rated conduct problems, but only for children without significant CU traits. In a study of juvenile offenders, harsh/inconsistent discipline predicted CP exclusively in children with low levels of psychopathic and CU traits (Edens et al. 2008). Further substantiating the unique contribution of CU traits, in a recent study of 57 families enrolled in an internet-based parent management training program, despite equal skill-learning, treatment outcomes were worse among children with high versus low CU traits (Hogstrom, et al., 2013). Nevertheless, a recent review observed that the association of negative parenting with respect to CP among children with high CU traits has not been definitively characterized (Waller et al., 2013). However, studies to date are typically cross-sectional, employ samples with limited generalizability (e.g., juvenile offenders), and infrequently disaggregate different dimensions of CP. Given that CU traits are particularly salient to early expressions of CP (i.e., prior to age 10), longitudinal studies with a narrow age range should be prioritized given that they are less vulnerable to unmeasured developmental influences (e.g., pubertal onset in mixed studies of children and adolescents).

Although effective interventions for CP frequently prioritize reducing negative parenting
practices and increasing positive parenting practices, there is replicated evidence that parenting practices may be more strongly associated with offspring CP among children with low versus high levels of CU traits (Wootton, et al., 1997; Falk and Lee, 2012). The goal of the current study was to examine the independent association of multiple dimensions of positive and negative parenting behavior and CU traits with respect to prospective change in CP, controlling for baseline CP and other potent risk factors for CP (e.g., ADHD). Further, we examined parenting behavior x child CU traits interactions as predictors of growth in CP. We employed a large and ethnically diverse sample of 6-9 year-old children with (n = 116) and without ADHD (n = 105) followed prospectively for 2 years (N = 184; 88% retention). Assessment of key constructs included multiple measures (e.g., self report and observational data) and differentiated CP constructs (e.g., ODD/CD and aggression versus rule breaking behavior).

Methods

Participants:

Participant characteristics are identical to Goal 1 of Study 1 (see pages 13-14).

Measures:

Parenting Behavior:

We used the same parenting measures used in Study 1, described on pages 15-16 and in Study 1a, described on pages 38-40.

Antisocial Behavior

The CP measures are identical to those detailed for Studies 1 and 1a (pages 16-17 and 33).

Data Analytic Procedures

First, we evaluated age, sex, and race-ethnicity as potential covariates: based on their
significant association with key Wave 2 outcomes, we controlled for sex in predictions of DBD ODD symptoms, CBCL Aggressive Behavior and CBCL Rule-Breaking Behavior. We additionally controlled for age for CBCL Aggressive Behavior. Race-ethnicity was excluded given that it was unrelated to all Wave 2 outcomes; similarly, no covariates were related to DBD CD symptoms. Second, to account for the case–control design (i.e., ADHD vs. control) and the association of CU traits and DBD with ADHD (Marsh et al. 2008), we controlled for Wave 1 ADHD diagnostic status in all models. Next, to examine the independent and prospection prediction of Wave 2 ODD, CD, as well as CBCL Aggressive and Rule Breaking Behavior from Wave 1 positive parenting (i.e., positive reinforcement and parental involvement), as well as CU traits, we employed Poisson regression so that all models were robust to the highly skewed Wave 2 CP distributions.

In predictions of Wave 2 parent- and teacher-rated CP from the DBD rating scale, we rigorously employed generalized estimating equations (GEE; Hardin, 2005), which allowed examination of parent and teacher reports as a single repeated measures factor (i.e., an “informant” factor). This strategy conservatively reduced the number of statistical tests and simultaneously accounted for the correlation between parent and teacher ratings of CP. Finally, we reproduced these models, but added the parenting behavior x CU traits interaction terms, separately for Wave 1 positive reinforcement and parental involvement. For Wave 2 missing teacher data, we used the same imputed data described on page 18.

Results

Associations of Wave 1 Parenting Behavior and CU traits with Wave 2 DBD CD symptoms

To capitalize on the identical parent- and teacher-rated DBD rating scale, we
implemented generalized estimating equations (GEE), thus examining Wave 2 CD symptoms as a single construct derived from both informants. All predictions of Wave 2 DBD CD symptoms controlled for Wave 1 ADHD status, Wave 1 parent and teacher rated CD symptoms, and informant.

Controlling for Wave 1 corporal punishment, Wave 1 involvement was unrelated to change in DBD CD symptoms (B = 0.03, SE = 0.02, p = 0.17) and CU traits were positively associated with growth in DBD CD symptoms (B = 0.20, SE = 0.04, p < 0.001). In the fully saturated model, the Wave 1 involvement x CU traits interaction was marginally associated with an increase in DBD CD symptoms (B = -0.02, SE = 0.01, p = 0.05).

Controlling for Wave 1 corporal punishment, Wave 1 positive reinforcement was unrelated to change in DBD CD symptoms (B = 0.02, SE = 0.03, p = 0.63) and CU traits were positively associated with growth in DBD CD symptoms (B = 0.19, SE = 0.04, p < 0.001). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was marginally related to an increase in DBD CD symptoms (B = -0.03, SE = 0.02, p = 0.07).

*Associations of Wave 1 Parenting Behavior and CU traits with DBD ODD symptoms*

For Wave 2 DBD ODD symptoms, we once again utilized GEE, with rigorous control of Wave 1 ADHD status, Wave 1 parent and teacher rated ODD symptoms, child sex, and informant. Controlling for Wave 1 corporal punishment, Wave 1 involvement was unrelated to change in DBD ODD symptoms (B = 0.01, SE = 0.01, p = 0.58), and CU traits were marginally positively associated with growth in DBD ODD symptoms and (B = 0.04, SE = 0.02, p = 0.07). In the fully saturated model, the Wave 1 involvement x CU traits interaction was unrelated to change in DBD ODD symptoms (B < 0.01, SE = 0.01, p = 0.99).

Controlling for Wave 1 corporal punishment, Wave 1 positive reinforcement and CU
traits were each marginally positively related to an increase in DBD ODD symptoms (B = 0.03, SE = 0.02, p = 0.07 and B = 0.04, SE = 0.02, p = 0.06, respectively). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was unrelated to change in DISC ODD symptoms (B = 0.01, SE = 0.04, p = 0.53).

**Associations of Wave 1 Parenting Behavior and CU traits with Wave 2 CBCL Aggressive Behavior**

All models examining Wave 2 CBCL Aggressive Behavior controlled for age, sex, as well as Wave 1 ADHD status and CBCL Aggressive Behavior. Controlling for Wave 1 corporal punishment, Wave 1 involvement predicted decreased CBCL Aggressive Behavior (B = -0.02, SE = 0.01, p = 0.04), and CU traits were unrelated to change in CBCL Aggressive Behavior (B = 0.02, SE = 0.02, p = 0.31). In the fully saturated model, the Wave 1 involvement x CU traits interaction was unrelated to change in CBCL Aggressive Behavior (B = 0.01, SE < 0.01, p = 0.16).

Controlling for Wave 1 corporal punishment, Wave 1 positive reinforcement and CU traits were unrelated to change in CBCL Aggressive Behavior (B < 0.01, SE = 0.01, p = 0.85 and B = 0.02, SE = 0.02, p = 0.38, respectively). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was significantly associated with an increase in CBCL Aggressive Behavior (B = 0.02, SE = 0.01, p < 0.01). To probe the significant interaction, we examined the association of Wave 1 positive reinforcement and CBCL Aggressive Behavior at three levels of CU traits (Aiken and West, 1991). For children with low (<1 SD below the grand mean; CU = 1.03) and mean levels of CU traits (CU = 2.97), positive reinforcement was unrelated to CBCL Aggressive Behavior (B = -0.04, SE = 0.03, p = 0.26 and B = -0.02, SE = 0.02, p = 0.33, respectively); for children with high levels of CU traits (>1 SD above the grand
mean; CU = 4.90), however, positive reinforcement significantly predicted an increase in CBCL Aggressive Behavior from Wave 1 to Wave 2 (B = 0.04, SE = 0.02, p = 0.03).

**Associations of Wave 1 Parenting Behavior and CU traits with Wave 2 Rule Breaking Behavior**

All models examining Wave 2 CBCL Rule Breaking Behavior controlled for gender and Wave 1 ADHD status and Rule Breaking Behavior. Controlling for Wave 1 corporal punishment, Wave 1 involvement was unrelated to CBCL Rule Breaking Behavior (B = -0.02, SE = 0.01, p = 0.19), and CU traits were positively associated with an increase in CBCL Rule Breaking Behavior (B = 0.10, SE = 0.03, p < 0.01). In the fully saturated model, the Wave 1 involvement x CU traits interaction was unrelated to change in CBCL Rule Breaking Behavior (B < -0.01, SE = 0.01, p = 0.44).

Controlling for Wave 1 corporal punishment, Wave 1 both positive reinforcement and CU traits positively predicted an increase in CBCL Rule Breaking Behavior (B = 0.05, SE = 0.02, p = 0.01 and B = 0.12, SE = 0.03, p < 0.01, respectively). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was unrelated to change in CBCL Rule Breaking Behavior (B < 0.01, SE = 0.01, p = 0.68).

**Discussion**

Based on a well-characterized sample of 184 6-9 year old children with and without ADHD, followed prospectively for two years, we examined the independent association of separable dimensions of positive parenting behavior (i.e., positive reinforcement and parental involvement), youth CU traits, and their interaction with respect to multiple dimensions of CP (i.e., ODD, CD, CBCL Rule Breaking and Aggressive Behavior). Several key findings emerged: (1) Controlling for baseline (i.e. Wave 1) ADHD diagnostic status (i.e., ADHD versus non-
ADHD comparison youth), Wave 1 CP, and positive and negative parenting behaviors, Wave 1
CU traits positively predicted growth in DBD ODD and CD symptoms, and CBCL Rule
Breaking Behavior; (2) Controlling for Wave 1 ADHD diagnostic status, Wave 1 CP, and
corporal punishment, positive reinforcement predicted growth in CBCL Rule Breaking Behavior
whereas parental involvement predicted a decrease in CBCL Aggressive Behavior. (3) We
observed significant interactions where positive reinforcement predicted growth in CBCL
Aggressive Behavior, but only in children with high CU traits: no association was observed
among children with mean or low CU traits (Figure 4.1). No other significant interactions were
observed between CU traits and parenting behavior.

These preliminary results suggest that separable facets of CP were sensitive to different
risk factors, as evidenced by the fact that CU traits uniquely moderated predictions of emergent
Aggressive Behavior from positive parenting, but not Rule Breaking Behavior. Aggressive and
Rule Breaking Behavior reflect overt and covert CP, respectively, a critical distinction well-
validated in the literature: for example, among 1,669 six to seventeen year old boys, overt and
covet factors of CD symptoms uniquely predicted CBCL Aggressive and Delinquent (now Rule
Breaking Behavior in the current CBCL) Behavior, respectively (Tackett, et al., 2003). Similarly,
in a meta-analysis of 44 factor studies, CP consisted of separable overt and covert dimensions
(Frick et al., 1993). Given that CBCL Aggressive Behavior items reflect overt actions whereas
Rule Breaking items convey covert behaviors, the Behavioral Inhibition System (BIS) may be
activated with overt CP (e.g., aggression), but not covert CP (e.g., rule breaking). The BIS
produces narrowed attention, inhibition of motor behavior and increased scanning in response to
relevant cues in the environment, and thus, impairment in the BIS would result in diminished
sensitivity to cues for punishment, reward, and novelty (Gray, 1987; O’Brien and Frick, 1996;
Fisher and Blair, 1998; Dadds and Salmon, 2003; Pardini, Lochman & Frick, 2003). Thus, the Aggressive and Rule Breaking Behavior scales (and perhaps overt and covert CP more generally) may reflect different underlying mechanisms that are sensitive to different risk factors and related pathways. Studies that focus solely on DSM-based constructs or other aggregate constructs (e.g., antisocial behavior) may misrepresent the considerable heterogeneity within CP.

CU traits moderated the association of positive reinforcement and growth in CBCL Aggressive Behavior, but not with CBCL Rule Breaking Behavior. A recent review suggested that there are separate mechanisms underlying the development of aggression in children with high levels of CU traits relative to their peers with low or mean levels of CU traits (Munoz and Frick, 2012). For example, children with CU traits exhibit impaired BIS (Frick et al., 2003); if children with elevated CU traits lack sensitivity to cues for reward, these preliminary data suggest that children with elevated CU traits exposed to parental positive reinforcement may exhibit more aggression over time. This finding did not apply to CBCL Rule-Breaking behavior, in which the threat of punishment and cue for reward may be more distal and the BIS may be activated less frequently. Therefore the possible BIS impairment in children with CU traits may not be relevant to Rule Breaking Behavior, which may contribute to the finding that, regardless of level of CU traits, children exhibited comparable levels of Rule Breaking Behavior. This is consistent with a recent review where children with high and low CU traits different with respect to important biomarkers for threat response: for example, reduced amygdala response and decreased functional connectivity between the amygdala and ventromedial prefrontal cortex was observed uniquely in children with elevated CU traits (Herpers, et al., 2013). These preliminary data suggest that CU traits designate an important subgroup of children with CP, although we await future studies that will directly test the role of the BIS. For example, laboratory-based tasks
of the BIS in children with and without high levels of CU traits will allow for mediation models that test the role of the BIS and different types of CP (e.g., Aggression versus Rule Breaking).

Whereas the current study found that positive parenting behavior (i.e., positive reinforcement) predicted growth in CP among youth elevated CU traits, Wootton et al., (1997) found that children with CU traits displayed less variability in CP as a function of ineffective parenting; Falk and Lee (2012) similarly found that children with CU traits were less responsive to positive parenting practices than their low CU traits peers. One potential contributing factor to these divergent patterns is the multidimensional nature of positive parenting behavior. Parental involvement and positive reinforcement, while correlated, reflect separable constructs, as indicated by our factor analysis, which confirmed that, in our sample, parental involvement and positive reinforcement are distinct factors (results available upon request). Parental involvement includes items such as “talks to child about his/her friends,” and indicates an involvement in and knowledge of a child’s daily activities, but no maintenance or change of behaviors. In contrast, positive reinforcement is a behavioral concept, and is a central aspect of parenting interventions for CP, used selectively to increase positive behavior (Gardner et al., 2003; Reid et al., 2004). This distinction between involvement and reinforcement, which respectively reflect knowledge and involvement in child behaviors, and change of child behaviors, may explain why the interaction predicting growth in CP was specific to positive reinforcement. Crucially, the implementation of positive reinforcement must be contingent upon other related important concepts such as labeled behavior and timely use of praise to reinforce prosocial behavior (Bell and Eyberg, 2002). Thus, perhaps in contrast to the type of positive reinforcement central to parenting interventions, the parental self-report of positive reinforcement featured in this study may reflect distinct patterns of youth engagement from effective parent management practices.
That is, positive reinforcement may iatrogenically affect negative child behavior if implemented without regard to these important behavioral principles.

We emphasize several study limitations. First, although we meaningfully differentiated parental involvement and positive reinforcement, as mentioned above, positive reinforcement may betray more complex patterns of association unless careful attention is paid to the timing and context of parenting practices (e.g., the reinforcement may be rating reinforcement of negative behavior). Additionally, although parenting is correlated with CP, this association may reflect shared genetic factors and/or genetic mediation, which may further complicate the relationship between parenting and CP in this study. Third, this was a two-year prospective longitudinal study, but given the age range of participants at baseline (6-9 years) and follow-up (8-11 years), the CP in this sample likely reflect early onset CP, which reflect unique correlates and causal influences including higher stability, comorbidity, treatment resistance, and heritability (Hinshaw, et al., 1993; Moffitt 1993; Silverthorn, et al., 2004; Viding et al., 2005). Finally, while we controlled for sex in predictions of certain CP outcomes, sex differences in CU traits and CP more broadly are routinely reported with regard to levels of CU traits and severity of CP (Forth, Brown, Hart & Hare, 1996; Stickle, Kirpatrick & Bruch, 2009; Stickle, et al., 2012), and beyond that, there are different patterns of associations: CU traits are correlated with both relationally and physically aggressive responses to provocation in girls, but only to physically aggressive responses in boys (Stickle et al., 2012), and within groups of children with high CU traits, girls may have equal or higher levels of CU traits than boys (Stickle et al., 2012; Fanti, 2013). Given the limited number of girls in this sample, we await additional studies that are able to prosecute sex differences in CU traits, CP, and their underlying mechanisms.

Independent of baseline ADHD and negative parenting behavior (i.e., corporal
punishment), positive reinforcement predicted growth in CP over a two-year period, but only in children with high levels of CU traits; among children with low or mean levels of CU traits, their Aggressive Behavior was largely stable across time, even in the presence of increased positive reinforcement.
CHAPTER FIVE: Study 2a: Additional Methods and Results for Study 2

Given that the Study 2a consisted of multiple informants and constructs, the manuscript above selectively reported and interpreted key results and patterns. However, the following section includes analyses that were excluded from the submitted manuscript above, but were proposed in the original dissertation prospectus. There are two sub-sections, and the additional methods, analytic procedures, and results for each sub-section are detailed below. The sub-sections including the following: (1) additional measures of CP (i.e., number of CD and ODD symptoms from the DISC) and (2) additional parenting measures (i.e., observed negative talk, observed praise, and self-reported corporal punishment).

Participants:

Participant characteristics are identical to Goal 1 of Study 1 (see pages 13-14).

Measures:

Parenting Behavior:

The current study used the same parenting measures used in Study 1, described on pages 15-16 and in Study 1a, described on pages 38-40.

Antisocial Behavior

The CP measures are identical to those detailed for Studies 1 and 1a (pages 16-17 and 33).

Data Analytic Procedures

First, we evaluated age, sex, and race-ethnicity as potential covariates: based on their significant association with key Wave 2 outcomes, we controlled for sex in predictions of DBD ODD symptoms, CBCL Aggressive Behavior and CBCL Rule-Breaking Behavior. We additionally controlled for age for CBCL Aggressive Behavior. Race-ethnicity was excluded
given that it was unrelated to all Wave 2 outcomes; similarly, no covariates were related to DBD CD symptoms. Second, to account for the case–control design (i.e., ADHD vs. control) and the association of CU traits and DBD with ADHD (Marsh et al. 2008), we controlled for Wave 1 ADHD diagnostic status in all models. Next, to examine the independent and prospection prediction of Wave 2 ODD, CD, as well as CBCL Aggressive and Rule Breaking Behavior from Wave 1 observed negative talk, observed praise, and self-reported corporal punishment, as well as CU traits, we employed Poisson regression so that all models were robust to the highly skewed Wave 2 CP distributions.

In predictions of Wave 2 parent- and teacher-rated CP from the DBD rating scale, we rigorously employed generalized estimating equations (GEE; Hardin, 2005), which allowed examination of parent and teacher reports as a single repeated measures factor (i.e., an “informant” factor). This strategy conservatively reduced the number of statistical tests and simultaneously accounted for the correlation between parent and teacher ratings of CP. Finally, we reproduced these models, but added the parenting behavior x CU traits interaction terms, separately for Wave 1 observed negative talk, observed praise, and self-reported corporal punishment.

For Wave 2 missing teacher data, we used the same imputed data described on page 18.

(1) Aim 2a: Associations of Wave 1 Parenting Behavior and CU traits with Wave 2 DISC CD symptoms

Briefly, to review, all predictions of Wave 2 DISC CD symptoms controlled for both Wave 1 ADHD status and CD symptoms. We employed poisson regression to examine the association of Wave 1 observed positive parenting behavior (i.e., praise) and CU traits with
respect to Wave 2 CD symptoms, controlling for observed negative parenting. Wave 1 observed praise was unrelated to change in DISC CD symptoms (B = -0.01, SE = 0.02, p = 0.46) whereas CU traits were positively associated with an increase in DISC CD symptoms (B = 0.16, SE = 0.08, p = 0.04). In the fully saturated model, the Wave 1 observed praise x CU traits interaction was unrelated to change in DISC CD symptoms (B = 0.01, SE = 0.01, p = 0.14).

In a highly similar model, controlling for Wave 1 observed praise, Wave 1 observed negative parenting was marginally positively related to an increase in DISC CD symptoms (B = 0.03, SE = 0.02, p = 0.09) and CU traits were positively associated with growth in DISC CD symptoms (B = 0.16, SE = 0.08, p = 0.04). In the fully saturated model, the Wave 1 observed negative parenting x CU traits interaction was unrelated to change in DISC CD symptoms (B = -0.01, SE = 0.01, p = 0.44).

Controlling for Wave 1 corporal punishment, Wave 1 involvement and CU traits were unrelated to change in DISC CD symptoms (B = 0.01, SE = 0.04, p = 0.89 and B = 0.09, SE = 0.08, p = 0.28). In the fully saturated model, the Wave 1 involvement x CU traits interaction was unrelated to change in DISC CD symptoms (B = -0.02, SE = 0.02, p = 0.21).

Controlling for Wave 1 corporal punishment, Wave 1 positive reinforcement was unrelated to change in DISC CD symptoms (B = -0.04, SE = 0.05, p = 0.48) and CU traits were marginally associated with growth in DISC CD symptoms (B = 0.15, SE = 0.07, p = 0.05). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was unrelated to change in DISC CD symptoms (B = -0.02, SE = 0.03, p = 0.51).

Controlling for Wave 1 positive parenting, Wave 1 corporal punishment and CU traits were unrelated to change in DISC CD symptoms (B = 0.03, SE = 0.12, p = 0.78 and B = 0.10, SE = 0.08, p = 0.20). In the fully saturated model, the Wave 1 corporal punishment x CU traits interaction was unrelated to change in DISC CD symptoms (B = -0.01, SE = 0.01, p = 0.44).
was unrelated to change in DISC CD symptoms (B = 0.02, SE = 0.06, p = 0.71).

*Associations of Wave 1 Parenting Behavior and CU traits with DISC ODD symptoms*

Parallel to predictions of Wave 2 CD symptoms, all models examining Wave 2 DISC ODD symptoms controlled for both Wave 1 ADHD status and ODD symptoms. Using poisson regression, controlling for Wave 1 observed negative parenting, Wave 1 observed praise was unrelated to change in DISC ODD symptoms (B = -0.01, SE = 0.02, p = 0.46) and CU traits were unrelated to change in DISC ODD symptoms (B = 0.02, SE = 0.03, p = 0.47). In the fully saturated model, the Wave 1 observed praise x CU traits interaction was unrelated to change in DISC ODD symptoms (B < 0.01, SE < 0.01, p = 0.61).

Controlling for Wave 1 observed praise, Wave 1 observed negative parenting and CU traits were each unrelated to change in DISC ODD symptoms (B < 0.01, SE = 0.01, p = 0.69 and B = 0.02, SE = 0.03, p = 0.47, respectively). In the fully saturated model, the Wave 1 observed negative parenting x CU traits interaction was marginally associated with an increase in DISC ODD symptoms (B = -0.01, SE < 0.01, p = 0.08).

Controlling for Wave 1 corporal punishment, Wave 1 involvement and CU traits were unrelated to change in DISC ODD symptoms (B < -0.01, SE = 0.01, p = 0.77 and B = 0.01, SE = 0.03, p = 0.85, respectively). In the fully saturated model, the Wave 1 involvement x CU traits interaction was unrelated to change in DISC ODD symptoms (B = 0.01, SE = 0.01, p = 0.42).

Controlling for Wave 1 corporal punishment, Wave 1 positive reinforcement and CU traits were each unrelated to change in DISC ODD symptoms (B = -0.03, SE = 0.05, p = 0.54 and B = 0.04, SE = 0.03, p = 0.26, respectively). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was unrelated to change in DISC ODD symptoms (B = -0.02, SE = 0.01, p = 0.22).
Controlling for Wave 1 positive parenting, Wave 1 corporal punishment and CU traits were unrelated to change in DISC ODD symptoms (B = -0.02, SE = 0.05, p = 0.64 and B = 0.03, SE = 0.03, p = 0.45, respectively). In the fully saturated model, the Wave 1 corporal punishment x CU traits was unrelated to change in DISC ODD symptoms (B = -0.04, SE = 0.03, p = 0.14).

(2) Aim 2a: Additional Parenting Measures: Observed Negative Talk, Observed Praise and Self-Reported Corporal Punishment

To capitalize on the identical parent- and teacher-rated DBD rating scale, we implemented generalized estimating equations (GEE), thus examining Wave 2 CD symptoms as a single construct derived from both informants. All predictions of Wave 2 DBD CD symptoms controlled for Wave 1 ADHD status, Wave 1 parent and teacher rated CD symptoms, and informant.

Controlling for Wave 1 observed negative parenting, Wave 1 observed praise was unrelated to change in DBD CD symptoms (B = 0.02, SE = 0.01, p = 0.10) whereas CU traits positively predicted an increase in CD symptoms (B = 0.21, SE = 0.04, p < 0.001). In the fully saturated model, the Wave 1 observed praise x CU traits interaction significantly predicted escalating CD symptoms (B = 0.01, SE < 0.01, p = 0.03). We then examined the association of Wave 1 involvement and DBD CD symptoms at three levels of CU traits (Aiken and West, 1991). For children with low (<1 SD below the grand mean; CU = 1.03), given limited variability within the reduced sample (all but 3 children with low levels of CU traits had a score of 0 for DBD CD symptoms), simple effects could not be calculated. However, at both mean (CU = 2.97) and elevated levels (>1 SD above the grand mean; CU = 4.90), observed praise was unrelated to change in DBD CD (B = 0.01, SE = 0.02, p = 0.41 and B = 0.01, SE = 0.01, p = 0.29,
respectively).

Controlling for Wave 1 observed praise, Wave 1 observed negative parenting was unrelated to change in DBD CD symptoms ($B < 0.01, SE = 0.01, p = 0.67$) and CU traits were positively associated with an increase in DBD CD symptoms ($B = 0.21, SE = 0.04, p < 0.001$). In the fully saturated model, the Wave 1 observed negative parenting x CU traits interaction was marginally related to growth in DBD CD symptoms ($B = -0.01, SE = 0.01, p = 0.08$).

Controlling for Wave 1 positive parenting, and Wave 1 teacher and parent rated DBD CD symptoms, Wave 1 corporal punishment was unrelated to change in DBD CD symptoms ($B = 0.03, SE = 0.07, p = 0.69$) and CU traits were positively associated with an increase in DBD CD symptoms ($B = 0.20, SE = 0.04, p < 0.001$). In the fully saturated model, the Wave 1 corporal punishment x CU traits was unrelated to change in DBD CD symptoms ($B = -0.02, SE = 0.03, p = 0.63$).

For Wave 2 DBD ODD symptoms, we once again utilized GEE, with rigorous control of Wave 1 ADHD status, Wave 1 parent and teacher rated ODD symptoms, child sex, and informant. Controlling for Wave 1 observed negative parenting, Wave 1 observed praise and CU traits were each unrelated to prospective change in DBD ODD symptoms ($B = -0.01, SE = 0.01, p = 0.40$ and $B = 0.03, SE = 0.02, p = 0.32$, respectively). In the fully saturated model, the Wave 1 observed praise x CU traits interaction was unrelated to change in DBD ODD symptoms ($B < -0.01, SE < 0.01, p = 0.65$).

Controlling for Wave 1 observed praise, Wave 1 observed negative parenting and CU traits were each unrelated to change in DBD ODD symptoms ($B = 0.01, SE = 0.01, p = 0.19$ and $B = 0.03, SE = 0.02, p = 0.32$, respectively). In the fully saturated model, the Wave 1 observed negative parenting x CU traits interaction was unrelated to change in DBD ODD symptoms ($B <
Controlling for Wave 1 positive parenting, Wave 1 corporal punishment was unrelated to change in DBD ODD symptoms (B = 0.05, SE = 0.04, p = 0.04), and CU traits were marginally positively associated with growth in DBD ODD symptoms (B = 0.05, SE = 0.02, p = 0.05). In the fully saturated model, the Wave 1 corporal punishment x CU traits was unrelated to change in DBD ODD symptoms (B < 0.01, SE = 0.02, p = 0.89).

Models examining Wave 2 CBCL Aggressive Behavior controlled for age, sex, as well as Wave 1 ADHD status and CBCL Aggressive Behavior. Using poisson regression, controlling for Wave 1 observed negative parenting, Wave 1 observed praise and CU traits were unrelated to change in CBCL Aggressive Behavior (B < -0.01, SE < 0.01, p = 0.24 and B = 0.02, SE = 0.02, p = 0.28, respectively). In the fully saturated model, the Wave 1 observed praise x CU traits interaction was unrelated to change in CBCL Aggressive Behavior (B < 0.01, SE < 0.01, p = 0.82).

Controlling for Wave 1 observed praise, Wave 1 observed negative parenting was positively associated with CBCL Aggressive Behavior (B = 0.01, SE < 0.01, p = 0.01) and CU traits were unrelated to change in CBCL Aggressive Behavior (B = 0.02, SE = 0.02, p = 0.28). In the fully saturated model, the Wave 1 observed negative parenting x CU traits interaction was unrelated to change in CBCL Aggressive Behavior (B < -0.01, SE < 0.01, p = 0.13).

Controlling for Wave 1 positive parenting, Wave 1 corporal punishment and CU traits were unrelated to change in CBCL Aggressive Behavior (B = 0.04, SE = 0.02, p = 0.13 and B = 0.02, SE = 0.02, p = 0.16, respectively). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was unrelated to change in CBCL Aggressive Behavior (B = -0.01, SE = 0.01, p = 0.53).
Predictions of Wave 2 CBCL Rule Breaking Behavior controlled for gender and Wave 1 ADHD status and Rule Breaking Behavior. Using poisson regression, controlling for Wave 1 observed negative parenting, Wave 1 observed praise was unrelated to change in CBCL Rule Breaking Behavior (B = 0.01, SE = 0.01, p = 0.77) and CU traits were positively associated with growth in CBCL Rule Breaking Behavior (B = 0.08, SE = 0.04, p = 0.03). In the fully saturated model, the Wave 1 observed praise x CU traits interaction was unrelated to change in CBCL Rule Breaking Behavior (B = 0.01, SE < 0.01, p = 0.12).

Controlling for Wave 1 observed praise, Wave 1 observed negative parenting was unrelated to change in CBCL Rule Breaking Behavior (B = 0.01, SE = 0.01, p = 0.40) and CU traits were positively associated with growth in CBCL Rule Breaking Behavior (B = 0.08, SE = 0.04, p = 0.03). In the fully saturated model, the Wave 1 observed negative parenting x CU traits interaction was significantly associated with an increase in CBCL Rule Breaking Behavior (B = -0.01, SE < 0.01, p = 0.04). To probe the significant interaction, we examined the association of Wave 1 observed negative parenting and CBCL Rule Breaking Behavior at three levels of CU traits (Aiken and West, 1991), however all simple effects were only marginal.

Controlling for Wave 1 positive parenting, Wave 1 corporal punishment was unrelated to change in CBCL Rule Breaking Behavior (B = 0.02, SE = 0.04, p = 0.61) and CU traits were positively associated with CBCL Rule Breaking Behavior (B = 0.12, SE = 0.03, p < 0.001). In the fully saturated model, the Wave 1 positive reinforcement x CU traits interaction was unrelated to change in CBCL Rule Breaking Behavior (B = -0.01, SE = 0.02, p = 0.64).
CHAPTER SIX: Conclusions

This dissertation focused on the association of individual differences in positive and negative parenting behavior with differentiated dimensions of youth conduct problems (CP), as well as their potential moderation by monoamine oxidase-A (MAOA) genotype and callous-unemotional (CU) traits. Data were drawn from two independent samples, consisting of a two-year prospective longitudinal study of 6-9 year-old youth with and without ADHD (N = 221) and approximately 2,500 adolescents followed prospectively for six years from the National Longitudinal Study of Adolescent Health (Add Health).

Capitalizing on multi-method (e.g., observational) and multidimensional measures of parenting and youth CP, a key conclusion from this dissertation is the complexity associated with rigorous measurement of individual differences in parenting behavior. Despite inclusion of empirically separable facets of parenting behavior (i.e., parental involvement, positive reinforcement, corporal punishment), overall, findings were quite inconsistent, differing according to the specific type of CP (i.e., aggressive behavior vs. rule breaking behavior) and potentially masking more specific patterns of association. For example, contrary to our hypothesis that positive parenting constructs would inversely predict CP, among children with high levels of CU traits, positive reinforcement was associated growth in aggressive behavior over a two year period, underscoring the necessity of more closely examining the parenting measures and what constructs they reflect. As the measures currently stand, they are limited in that they only measure parents’ own perception of their involvement, and do not reflect the perception of the child, or how that perception may influence conduct problems. Further, parent reports of scales of praise and positive reinforcement may not be purely positive constructs, as it is unknown what parents are praising and reinforcing. The studies included here focused on the
independent contribution of each parenting measure, however, future work should focus on how the parenting measures function simultaneously, to better reflect the combination of parenting behaviors that best predicts childhood outcomes (e.g., characterizing parenting according to warmth/control).

Understanding the specific combinations of parenting behavior that best predict decreases in CP will inform intervention development. Current evidence based interventions for CP focus on increasing positive parenting constructs, such as positive reinforcement, and a deeper understanding of how the parenting constructs interact with and inform one another can improve these interventions, and cater them to specific subtypes of children. For example, children with high levels of CU traits might not respond as favorably to increases in positive reinforcement in the absence of other parenting variables, such as consistent discipline and monitoring. Further, children who present with rule breaking behavior and not aggressive behavior may similarly respond differently to parenting variables. If parenting interventions were more tailored (i.e., personalized), both in terms of the parenting behaviors and CP that are targeted, parents might find them more rewarding and effective. Moreover, the longevity of changes in parenting behavior may be enhanced, a longstanding concern given that parenting changes are often ethereal (i.e., parenting behavior reverts to pre-intervention patterns).

Overall, this study included multiple measures of parenting, diverse measures of conduct problems, and multiple moderators, and therefore provides a broad perspective. Findings broadly suggested that there needs to be concerted efforts towards improving the measurement of individual differences in positive and negative parenting, spanning from positive reinforcement and parental involvement to harsh punishment and inconsistent discipline. We found that our findings were very specific to the precise measure of parenting and CP, and we stress the
importance of creating more targeted measures of parenting that more accurately reflect the constructs that relate to different types of CP, so that we can rigorously prosecute the specific parenting practices that reliably predict CP and their growth over time.
Table 1.1. Demographic information for Sample 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>ADHD (n = 116)</th>
<th>Control (n = 105)</th>
<th>F/χ²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td># Male (%)</td>
<td>84 (70.60)</td>
<td>67 (63.82)</td>
<td>2.07</td>
<td>.15</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>7.39 (1.05)</td>
<td>7.26 (1.11)</td>
<td>.60</td>
<td>.44</td>
</tr>
<tr>
<td>% Caucasian</td>
<td>57.12</td>
<td>60.94</td>
<td>0.04</td>
<td>.83</td>
</tr>
<tr>
<td>% ODD diagnosis</td>
<td>47.12</td>
<td>12.45</td>
<td>35.25</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>% CD diagnosis</td>
<td>7.56</td>
<td>0.00</td>
<td>8.49</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>% MAOA-H (males)</td>
<td>58.19</td>
<td>35.82</td>
<td>2.32</td>
<td>.13</td>
</tr>
<tr>
<td>% MAOA-H (females)</td>
<td>53.33</td>
<td>58.33</td>
<td>0.17</td>
<td>.68</td>
</tr>
</tbody>
</table>

Note. Mean age = average age at Wave 1 (range: 6-9); ODD = DISC-IV oppositional defiant disorder; CD = DISC-IV conduct disorder; MAOA-H = high activity allele of monoamine-oxidase-A.
Table 1.2. Demographic information for Sample 2

<table>
<thead>
<tr>
<th>Variable</th>
<th>MAOA-L (n = 719)</th>
<th>MAOA-LH (n = 548)</th>
<th>MAOA-H (n = 1148)</th>
<th>F/ χ²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (n)</td>
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<td>0</td>
<td>676</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>% Caucasian</td>
<td>60.44</td>
<td>--</td>
<td>72.49</td>
<td>21.10</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>15.72 (1.65)</td>
<td>--</td>
<td>15.66 (1.71)</td>
<td>.42</td>
<td>.52</td>
</tr>
<tr>
<td>Mean delinquency (SD)</td>
<td>3.77 (4.88)</td>
<td>--</td>
<td>3.92 (5.23)</td>
<td>.25</td>
<td>.62</td>
</tr>
<tr>
<td>Female (n)</td>
<td>218</td>
<td>548</td>
<td>470</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>15.61 (1.67)</td>
<td>15.65 (1.65)</td>
<td>15.41 (1.62)</td>
<td>2.71</td>
<td>.07</td>
</tr>
<tr>
<td>Mean delinquency (SD)</td>
<td>2.94 (4.02)</td>
<td>3.02 (3.80)</td>
<td>3.08 (4.20)</td>
<td>.09</td>
<td>.91</td>
</tr>
<tr>
<td>% Caucasian</td>
<td>47.71</td>
<td>64.04</td>
<td>18.09</td>
<td>68.01</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

Note. Some data were missing due to genotyping failure; Mean age = average age at Wave 1 (range: 12-20); Mean delinquency score = average delinquency score at Wave 1 (range: 0-39).
Table 2.1. Predicting growth in Aggressive Behavior from corporal punishment and MAOA genotype in boys

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th></th>
<th></th>
<th>Model 2</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE</td>
<td><em>p</em></td>
<td>B</td>
<td>SE</td>
<td><em>p</em></td>
</tr>
<tr>
<td>Race</td>
<td>-0.02</td>
<td>0.02</td>
<td>0.30</td>
<td>-0.02</td>
<td>0.02</td>
<td>0.18</td>
</tr>
<tr>
<td>Age</td>
<td>0.07</td>
<td>0.04</td>
<td>0.05</td>
<td>0.07</td>
<td>0.04</td>
<td>0.06</td>
</tr>
<tr>
<td>ADHD diagnostic status</td>
<td>0.48</td>
<td>0.10</td>
<td>&lt;0.001</td>
<td>0.46</td>
<td>0.10</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Corporal Punishment¹</td>
<td>0.06</td>
<td>0.03</td>
<td>0.18</td>
<td>0.11</td>
<td>0.04</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Positive Parenting¹</td>
<td>-0.03</td>
<td>0.02</td>
<td>0.18</td>
<td>-0.03</td>
<td>0.02</td>
<td>0.17</td>
</tr>
<tr>
<td>MAOA genotype</td>
<td>0.16</td>
<td>0.08</td>
<td>0.04</td>
<td>0.77</td>
<td>0.27</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Corporal Punishment x MAOA</td>
<td>-0.13</td>
<td>0.06</td>
<td>&lt;0.02</td>
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</tbody>
</table>
Table 2.2. Predicting growth in Aggressive Behavior from parental involvement and MAOA genotype in girls

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>0.01</td>
<td>0.03</td>
<td>0.76</td>
</tr>
<tr>
<td>ADHD diagnostic status</td>
<td>0.49</td>
<td>0.18</td>
<td>0.01</td>
</tr>
<tr>
<td>Corporal Punishment(^1)</td>
<td>0.06</td>
<td>0.05</td>
<td>0.21</td>
</tr>
<tr>
<td>Parental Involvement(^1)</td>
<td>-0.02</td>
<td>0.01</td>
<td>0.17</td>
</tr>
<tr>
<td>MAOA genotype</td>
<td>-0.24</td>
<td>0.14</td>
<td>0.09</td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>-0.01</td>
<td>0.03</td>
<td>0.86</td>
</tr>
<tr>
<td>ADHD diagnostic status</td>
<td>0.43</td>
<td>0.18</td>
<td>0.02</td>
</tr>
<tr>
<td>Corporal Punishment(^1)</td>
<td>0.10</td>
<td>0.05</td>
<td>0.06</td>
</tr>
<tr>
<td>Parental Involvement(^1)</td>
<td>-0.05</td>
<td>0.02</td>
<td>0.01</td>
</tr>
<tr>
<td>MAOA genotype</td>
<td>-2.78</td>
<td>1.20</td>
<td>0.02</td>
</tr>
<tr>
<td>Parental Involvement x MAOA</td>
<td>0.06</td>
<td>0.03</td>
<td>0.03</td>
</tr>
</tbody>
</table>
Table 4.1. Predicting growth in Aggressive Behavior from positive reinforcement and CU traits

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>Standard Error</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD diagnostic status</td>
<td>0.53</td>
<td>0.09</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age</td>
<td>0.06</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Harsh parenting(^1)</td>
<td>0.04</td>
<td>0.02</td>
<td>0.08</td>
</tr>
<tr>
<td>Positive Reinforcement(^1)</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>0.85</td>
</tr>
<tr>
<td>CU traits(^2)</td>
<td>0.02</td>
<td>0.02</td>
<td>0.38</td>
</tr>
<tr>
<td>Wave 1 Aggressive Behavior</td>
<td>0.14</td>
<td>0.41</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD diagnostic status</td>
<td>0.52</td>
<td>0.09</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age</td>
<td>0.06</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Harsh parenting(^1)</td>
<td>0.05</td>
<td>0.02</td>
<td>0.04</td>
</tr>
<tr>
<td>Positive Reinforcement(^1)</td>
<td>-0.07</td>
<td>0.02</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>CU traits(^2)</td>
<td>-0.48</td>
<td>0.15</td>
<td>0.001</td>
</tr>
<tr>
<td>Wave 1 Aggressive Behavior</td>
<td>0.06</td>
<td>&lt;0.01</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reinforcement x CU traits</td>
<td>0.02</td>
<td>0.01</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Note. ADHD = attention-deficit/hyperactivity disorder; CU traits = callous unemotional traits.

\(^1\)Self-reported harsh punishment and positive reinforcement from the Alabama Parenting Questionnaire. \(^2\)Sum score of CU traits from the Antisocial Process Screening Device.
Figures:

Figure 2.1 Corporal punishment x MAOA genotype for Aggressive Behavior in boys

Figure 2.2 Parental involvement x MAOA genotype for Aggressive Behavior in girls
Figure 4.1. Interaction of positive reinforcement and CU traits for Aggressive Behavior
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