HOW MAOA, DECISION-MAKING, AND NEGATIVE PARENTING IMPACT AGGRESSION AND CONDUCT PROBLEMS IN CHILDREN: A GENE BY ENVIRONMENT INTERACTION STUDY

by

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ABSTRACT

Objective: Test if decision-making moderates the relationship between MAOA gene variant and aggression/conduct problems and if low/high MAOA and negative parenting will moderate decision-making and aggression/conduct problems.

Method: Study used archival baseline data from a population of at-risk children. Measures include the buccal swab, the Iowa Gambling Task, the Teacher Report of Proactive and Reactive Aggression, the Behavior Assessment System for Children, and the Alabama Parenting Questionnaire.

Analyses: Base models will test if MAOA variants, decision-making, and negative parenting impacts reactive aggression/conduct problems, and if the interactions between MAOA gene variants x decision-making and negative parenting x decision-making impacts reactive aggression/conduct problems. Moderation models will test if MAOA gene variants and negative parenting impacts decision-making, and if the interaction between MAOA gene variants x negative parenting behaviors impacts decision-making.

Results: There was a significant moderation between MAOA gene variants and ADM when predicting to reactive aggression as well as a significant moderation between MAOA gene variants and ADM when predicting to conduct problems.

Discussion: The moderation effect between affective decision-making and MAOA gene variants predicting to reactive aggression and the moderation effect between low affective decision-making and MAOA gene variants predicting to conduct problems, demonstrates that the higher MAOA allele can act as a protective factor. However, a different relationship exists between

high affective decision-making and MAOA gene variants when predicting to conduct problems. In this moderation, higher affective decision-making can act as a protective factor against the risks associated with having the lower MAOA variant, but children with the higher MAOA variant engage in more conduct problem behavior. The study utilized a sample of aggressive and conduct problem youth, and these children may be more effective at using conduct problem behaviors to achieve their goals, even though they have the affective decision-making abilities to understand the differences between reward and punishment.

DEDICATION

This dissertation is dedicated to all of the people in my life who made this dream a reality. Thank you for being there and thank you for your guidance; it could not have been accomplished without you.

LIST OF ABBREVIATIONS AND SYMBOLS

AA = African American

ADM = Affective Decision-Making

APQ = Alabama Parenting Questionnaire

b = Unstandardized coefficient

ß = Beta

BASC = Behavior Assessment System for Children

BASC-TRS = Behavior Assessment System for Children – Teacher Rating Scale

bp = Base pair

CD = Conduct Disorder

CFI = Comparative Fit Index

CO = Gaining nothing, but 100% probability of losing nothing

CorPun = Corporal Punishment

CP = Conduct Problems

ΔF = Change in F

$\Delta\chi^2$ = Change in Chi-Square

DNA = Deoxyribonucleic acid

EC = Enzyme Commission Number

Est = Estimate

GCP = Group Coping Power

GEE = General Estimating Equations

I^2 = Measure of the percentage of across-study variation attributable to heterogeneity

ICP = Individual Coping Power

IncDis = Inconsistent Discipline

Inv = Involvement

IGT = Iowa Gambling Task

MAO = Monoamine Oxidase (monoamine; oxygen oxidoreductase, deaminating, EC 1.4.3.4)

MAOA = Monoamine Oxidase-A

MAOA-H = Carriers of the high MAOA variant

MAOA-L = Carriers of the low MAOA variant

MAOB = Monoamine Oxidase-B

Mon = Monitoring

mRNA = Messenger ribonucleic acid

N = Number of participants

NPT = Negative Parenting Total Score

ODD = Oppositional Defiant Disorder

p = Probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value

PPT = Positive Parenting Total score

PosPar = Positive Parenting

R^2 = Coefficient of determination

RA = Reactive Aggression

RTS = Reaction Time Score

RMSEA = Root-Mean-Square Error of Approximation

RO = 50:50 gamble between a win and loss

SE = Standard error

SMH = Somatic Marker Hypothesis

SNP = Single-Nucleotide Polymorphisms

SRMR = Standardized root mean square residual

Std = Standard

t = t-test

TRRPA = Teacher Report of Reactive and Proactive Aggression

VNTR = Variable number tandem repeat

x = by

XX = Females

XY = Males

z = Standard normal score

χ^2 = Chi-Square

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To my mentor: thank you so much for all of your patience, the excitement you expressed about this project, and for reigning me in when it became a little overwhelming.

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INTRODUCTION

This study will attempt to determine if genetic variants of Monoamine Oxidase A (MAOA), decision-making abilities, and parenting behaviors are related within a population of children with aggressive and conduct problem behaviors. Specifically, this study has two main aims; to determine whether decision-making abilities act as a moderator between genetic variants of MAOA and aggressive or conduct problem behavior; and to determine whether self-reported ratings of negative parenting behaviors and genetic variants of MAOA will act as a moderator between decision-making abilities and aggressive or conduct problem behavior. Previous research has shown an association between parts of these models; however, no research has examined the entire model. This introduction outline will present an overview of MAOA, reactive aggression, proactive aggression, conduct problems, decision-making, and negative parenting.

Monoamine Oxidase

Monoamine Oxidase (MAO; monoamine; oxygen oxidoreductase, deaminating, EC 1.4.3.4) has two forms; MAOA and MAOB (Johnston 1968; Knoll & Magyar 1972). Both forms of MAO degrade or catalyze neurotransmitters such as serotonin, dopamine, norepinephrine, and epinephrine, which have been linked to aggressive and conduct problem behavior (i.e., serotonin and dopamine) and stress regulation (i.e., norepinephrine and epinephrine; Caspi et al., 2002). MAO catalyzes these neurotransmitters by producing hydrogen peroxide (Shih 1991; Thorpe et al., 1987).

Monoamine Oxidase-A versus Monoamine Oxidase-B

Monoamine Oxidase-A (MAOA) and Monoamine Oxidase-B (MAOB) are structurally different (i.e., made up of different polypeptide units) and therefore not a modification of the same protein (Lan et al., 1989a). However, they may share a common ancestral gene (Grimsby et al., 1991). MAOA is more likely to catalyze or degrade serotonin, norepinephrine, dopamine, and clorgyline (Green & Youdim, 1975; Johnston, 1968; Knoll & Magyar, 1972; Fowler et al., 1982). MAOB is more likely to catalyze phenylethylamine, benzylamine, and deprenyl (Green & Youdim, 1975; Johnston, 1968; Knoll & Magyar, 1972; Fowler et al., 1982).

Monoamine Oxidase-A

Monoamine Oxidase-A (MAOA) is an enzyme encoded on the X chromosome in humans (Caspi et al., 2002). Specifically, the specific encoding region contains a 30-base pair (bp) repeating sequence, in the promoter region of MAOA, which specifies how much MAOA is produced by the brain (Sabol, Hu, & Hamer, 1998; Denney, Koch, & Craig, 1999; Deckert et al., 1999). MAOA has been mapped to the site of Xp11.23 (Lan et al., 1989b; Levy et al., 1989) and is related to orbitofrontal cortex development (Good et al., 2003), which is associated with integrity of the MAOA gene promotor region.

The MAOA upstream variable number tandem repeat (VNTR), is a functional polymorphism that can alter the ability of MAOA to transcribe and produce MAOA, and therefore catalyze neurotransmitters in the brain. MAOA alleles are characterized by the specific number of sequences they contain, and based upon this number they are either classified as producing low or high levels of MAOA, which in turn affects the levels of monoamine neurotransmitters in the brain. 3.5- and 4-repeat sequences are classified as high MAOA activity, and therefore produce lower levels of monoamine neurotransmitters in the brain. 2-, 3-, and 5-

repeat sequences are classified as low MAOA activity, and therefore produce higher levels of monoamine neurotransmitters in the brain (Sabol, Hu, & Hamer, 1998). This presentation of low versus high levels of monoamine can affect how a person responds to their environment.

Studies examining the association between genes and environments have shown that some individuals are more susceptible to negative environments, and have found MAOA underlies a biological stress response system due to its association with neurotransmitters such as norepinephrine and epinephrine (Caspi et al., 2002).

The low activity allele produces lower levels of MAOA, and therefore causes the brain to experience increased levels monoamine neurotransmitters. Due to these increased neurotransmitters, the amygdala and hippocampus are more responsive in reaction to emotional stress, such as maltreatment in childhood, and the regulatory prefrontal regions of the brain are impaired (Caspi et al., 2002; Meyer-Lindenberg et al., 2006). This may predispose an individual toward neural hyperactivity toward threat (Morell, 1993). Another theory suggests that the lower levels of MAOA could cause variations in an individual's responsivity to social environment, without a need for adversity or stressful situations (Belsky & Pluess, 2009).

Caspi et al., examined a group of male children from ages three to twenty-eight to ascertain why some children who were maltreated in early life later developed antisocial behaviors and others did not (2002). They found that maltreated children with higher levels of MAOA (i.e., children who had the allele conferring higher levels of MAOA expression) were less likely to develop antisocial behaviors later in life (b=0.24, SE=0.11, t=2.15, p=0.03). Similar studies have also replicated Caspi and colleagues' findings (e.g., Beach et al., 2010; Fergusson, Boden, Horwood, Miller, & Kennedy, 2011; Fergusson, Boden, Horwood, Miller, & Kennedy, 2012; Ducci et al., 2008; Kim-Cohen et al., 2006), with some exceptions (Huizinga et al., 2006;

Prichard, Jorm, Mackinnon, & Easteal, 2007; Haberstick et al., 2014; Haberstick et al., 2005; Prom-Wormley et al., 2009). Kim-Cohen and colleagues also performed a meta-analysis to examine the gene by environment interaction between MAOA gene variants and early childhood adversity and found no evidence of significant heterogeneity in the high-activity MAOA group across studies (χ^2=0.79, p=0.940, I^2=0.0%), mild to moderate heterogeneity across studies in the low-activity MAOA group (χ^2=6.29, p=0.179, I^2=36.4%). Therefore, the association between early familial adversity and mental health is significantly stronger in the low-activity MAOA versus the high-activity MAOA groups (2006). For an overview of studies examining MAOA gene variants and the subsequently described variables, please see Appendix A.

Reactive Aggression

Reactive aggression, as its name implies, is a more reaction-based aggressive behavior, and is often more emotional in nature (Dodge, 1991; Vitaro & Brendgen, 2011). Reactive aggression is typically an immediate and instinctual reaction of aggressive behavior in response to a perceived threat or a tendency to assign a hostile attribution to the benign actions of others (i.e., hostile attribution bias; Dodge, 1991; Vitaro & Brendgen, 2011; Dodge & Coie, 1987; Hubbard et al., 2001; Lobbestael, Cima, & Arntz, 2013). Individuals who engage in reactive aggressive behaviors typically have poor emotional and behavior regulation, and often experience stronger reactions to hostile and anger-related emotions (Rathert, Fite, Gaertner, & Vitulano, 2011; White, Jarrett, & Ollendick, 2013; White & Turner, 2014; Frick & Morris, 2004; Hubbard et al., 2004; Marsee & Frick, 2007). The poor emotional regulation can develop during early childhood in response to instances of adversity, including, but not limited to, negative parenting behaviors (i.e., corporal punishment or inconsistent discipline), violence or abuse, and inconsistent attachments (Dodge, 1991; Connor et al., 2004; Shields & Cicchetti, 1998).

Berkowitz hypothesized reactive aggression often stems from frustration, especially when the individual responds with anger or hostility (1978). Aversive events can be particularly problematic, as an individual is more likely to respond aggressively due to fear and the fight or flight response (Berkowitz, 1978). Therefore, a child who experiences aversive events early in life may learn to respond instinctually to neutral stimuli with reactive aggression.

MAOA and the Association with Reactive Aggression

In previous literature, lower levels of MAOA expression have been associated with reactive aggression. Mutations within the MAOA gene or absence of the gene entirely, can lead to issues with emotion regulation difficulties and impulsivity in males, including extreme aggressive reactions to emotion-provoking events (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993; Huang et al., 2004). Previous researchers have hypothesized low expression of MAOA is associated with impulsive aggression, which is typically anger related (Meyer-Lindenberg et al., 2006).

Low activity MAOA individuals who have experienced childhood physical abuse are also more at risk for increased impulsive behaviors and reactive opposition later in life (Huang et al., 2004; Jaffe et al., 2005). It has also been found that the low activity MAOA allele is associated with reactive aggression, lower frustration tolerance, and increased irritability in children (Manuck, Flory, Ferrell, Mann, & Muldoon, 2000; Meyer-Lindenberg et al., 2006). For an overview of studies that have examined the relationship between MAOA levels and aggression, please see Appendix B.

Proactive Aggression

Proactive aggression, as compared to reactive aggression, is more goal oriented and instrumental in nature. Individuals may use proactive aggression in an effort to obtain some

reward or achieve a desired outcome (Dodge, 1991; Vitaro & Brendgen, 2011; Crick & Dodge, 1996; Dodge et al., 1997; Marsee & Frick, 2007; Smithmyer, Hubbard, & Simons, 2000). This deliberate and planful use of aggressive behavior is very different from reactive aggression and its emotional drive.

Proactive aggression can develop in early childhood, just as reactive aggression can; however, the developmental pathways are different. Bandura's social learning theory posits that learning does not only occur in the presence of a reinforcement, and therefore, it is possible that early exposure to aggression or violent behavior may be a precursor to later proactive aggressive behaviors (Bandura, 1973; Dodge, 1991).

MAOA and the Association with Proactive Aggression

Previous literature found an association between higher levels of MAOA expression and proactive aggression in violent offenders versus healthy controls, and theorized that exposure to aversive events can lead to emotional insensitivity, but through modelling or social learning theory, the individual learns to engage in proactive aggressive behaviors to obtain some reward (Kolla, Attard, Craig, Blackwood, & Hodgins, 2014). However, other studies have not found an association between proactive aggression and MAOA gene variants (Kuepper, Grant, Wielpuetz, Hennig, 2013; Zhang et al., 2016). Due to the lack of substantial evidence indicating a clear link between MAOA gene variants and proactive aggression, proactive aggression will not be analyzed in the primary analyses; however, it will be examined in the exploratory analyses to determine if such a link exists in the total models. For an overview of studies that have examined the relationship between MAOA levels and aggression, please see Appendix B.

Conduct Problems

As with reactive aggression, researchers believe that one component of conduct problem behavior stems from difficulties regulating emotions (Eisenberg et al., 2001; Frick et al., 2003; Frick, Christian, & Wootton, 1999; Loney et al., 2006; Morris et al., 2002). However, the types of behaviors exhibited are separate and distinct from the behaviors seen in individuals exhibiting reactive aggression.

In the Behavior Assessment System for Children (BASC), conduct problems are considered separately from aggression and the questions asked are designed to determine whether a child or adolescent is exhibiting behaviors related to Oppositional Defiant Disorder (ODD) or Conduct Disorder (CD; American Psychiatric Association, 2013; Reynolds & Kamphaus, 1992). This includes behaviors such as bullying other children, truancy, suspension, breaking others' belongings, stealing while at school, and cheating at school.

MAOA and the Association with Conduct Problems

Researchers have also examined the association between MAOA levels and conduct problem behaviors, and typically found that individuals with lower levels of MAOA were often more at risk for conduct problem behaviors, especially when exposed to aversive environments at a young age.

For example, MAOA gene variants has been shown to moderate the relationship between childhood maltreatment and conduct disorder in Caucasian males (Caspi et al., 2002). There is also a risk for increased conduct problem behavior in African American and Caucasian males when parents engaged in more punitive discipline or corporal punishment (Choe, Shaw, Hyde, & Forbes, 2014; Falk, 2014). For an overview of studies that have examined the relationship between MAOA levels and conduct problems, please see Appendix C.

Decision-Making

Children who engage in aggression or conduct problem behavior are more at risk for developing negative outcomes later in life. As previous studies have shown, individuals with lower levels of MAOA can exhibit deviant behavior, poor impulse control, aggression, and conduct problems (Brunner et al., 1993; Huang et al., 2004; Meyer-Lindenberg et al., 2006; Jaffe et al., 2005; Manuck, Flory, Ferrell, Mann, & Muldoon, 2000; Caspi et al., 2002). However, fewer studies have examined the link between lower MAOA and decision-making processes, especially the link between lower levels of MAOA and affective decision-making processes, in which an individual makes decision based on emotional reactions or somatic cues. For an overview of studies that have examined the relationship between MAOA levels and decision-making, please see Appendix D.

The Iowa Gambling Task (IGT)

Aggressive and conduct problem youth suffer from a number of negative outcomes, which can impact children's healthy development as well as cause broader societal risks and costs. A previous study (Deming et al., 2014) has examined how children with conduct problems respond on the Iowa Gambling Task (IGT; Damásio, 1996, 1998) with findings indicating that they typically perform worse compared to controls. However, very few studies have examined how children who exhibit aggressive behaviors specifically respond on the IGT. Abnormalities in decision-making can be measured with the IGT via two scores--the Affective Decision-Making Score and Reaction Time Score.

Measuring Decision Making. The IGT can produce two scores; the Choice Score or Affective Decision-Making Score and the Reaction Time Score.

Affective Decision-Making Score. The Affective Decision-Making Score is a widely used measure of deficits in decision-making. Abnormalities in affective decision-making have been identified in children with aggressive behavior and conduct problems (Deming et al., 2014; Matthys & Lochman, 2010), and Bechara, Damásio, Tranel, and Anderson created the IGT to test for deficits in affective decision-making through the Somatic Marker Hypothesis (SMH), which postulates that individuals make decisions based on emotional processing (1998). These deficits in emotional processing occur when the individual cannot process the signal sent by the body (Bechara, Damásio, Tranel, & Damásio, 2005).

Advantageous IGT decisions occur when individuals feel that one deck will be disadvantageous compared to another (Jameson, Hinson, & Whitney, 2004), learn from this, and begin to anticipate the potentially disadvantageous decks (Bechara, Damásio, & Damásio, 2000; Damásio, 1998). Deming and colleagues (2014) examined decision-making and found that children who performed better on the IGT, in conjunction with higher levels of skin conductance, showed decreases in conduct problem behavior at follow-up; however, in that study they did not examine the relationship between decision making and aggressive behavior. Schutter, van Bokhoven, Vanderschuren, Lochman, & Matthys reported that adolescents with disruptive behavior disorders and substance dependence made riskier decisions on the IGT (2011). Luman, Sergeant, Knol, & Oosterlaan found that children ages 7 to 12 with Oppositional Defiant Disorder (ODD) preferred the deck with frequent large rewards as well as increasing penalties (2010). Results from these studies show that children with Conduct Disorder (CD) and ODD typically make riskier decisions compared to normal controls.

Reaction Time Score. Aggressive behavior can be present in both CD and ODD, through angry or irritable behavior or through behavior designed intentionally to hurt another person

(American Psychiatric Association, 2013). This overt confrontational behavior can result in a child making poor decisions on the IGT, as they favor the decks associated with larger rewards and larger punishments. This response style might indicate that these children are not responding to punishment (e.g. Matthys & Lochman, 2010). The confrontational style of aggressive behavior may also result from these children behaving impulsively or having poor inhibition, as measured by their Reaction Time Scores when choosing between the decks. A faster reaction time would indicate a greater degree of impulsivity or poor impulse control (Frick et al., 1993). Smith, Xiao, & Bechara (2012) used reaction time as a measurement of children's speed of processing and found that individuals with low Choice Scores would display faster reaction time when choosing disadvantageous decks. Therefore, children with aggression and conduct problems may exhibit more impulsive response styles on the IGT and therefore have faster reaction times (Connor & Doerfler, 2008; Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2005; Smith, Xiao, & Bechara, 2012).

Decision-Making and the Association with MAOA

As previously stated, the IGT is more likely to measure affective or emotional decision-making (i.e., hot decision-making) ability due to the way the task is structured. However, other forms of decision-making (i.e., cold decision-making) also exist, and some have previously been researched in association with low versus high levels of MAOA. It should be noted that findings in both areas of decision-making have been mixed.

For example, individuals with lower MAOA encoding have been shown to have increased deficits on tasks related to executive functioning, such as working memory and attentional control. These individuals also demonstrated reduced frontal brain activation, in areas which support these processes (Fan, Fossella, Sommer, Wu, & Posner, 2003; Meyer-Lindenberg

et al., 2006; Cerasa et al., 2008; Passamonti et al., 2006; Enge, Fleischhauer, Lesch, Reif, & Strobel, 2011).

Cold Decision-Making. Cold decision-making occurs when the individual cognitively understands the task and can use rationale thought, rather than emotional processes, to navigate the decision-making process (Blakemore & Robbins, 2012).

In one study, conducted by Frydman and colleagues, individuals were asked to complete a gambling task with one option (CO) having a non-negative payoff (gaining nothing, but 100% probability of losing nothing) and another option (RO) having a 50:50 gamble between a win and loss (winning $7 and losing $4 were equally likely). Low-MAOA carriers accepted the RO 6.4% more often than High-MAOA carriers (n=64, t=2.49 p=0.015, two-tailed) when the RO had a positive net expected utility (2011).

In another study, conducted by Zhong and colleagues, individuals were asked to rank their preferences for A) 1% chance of winning $200 and zero otherwise, B) 10% chance of winning $20 and zero otherwise, and C) 100% chance of winning $2. Individuals were considered to be making a "long-shot" decision when they ranked their preferences as A, B, and then C. Individuals with the 4-repeat allele (high activity) of MAOA were more likely to make a "long-shot" decision than subjects with 3-repeat allele (low activity; 2009).

Hot Decision-Making. Hot decision-making occurs when the individual does not understand the task cognitively and therefore can only rely on the emotional processes, rather than rational thought, to navigate the decision-making process (Blakemore & Robbins, 2012).

Verdejo-Garcia and colleagues attempted to examine the interaction between MAOA genetic variants and cocaine use on impulsive behaviors. They had subjects complete the IGT, but their results were insignificant (2013). However, they only used the choice score to calculate

decision-making ability and did not consider calculating response time scores. As the response time score may be more in line with what Verdejo-Garcia and colleagues were attempting to measure (i.e., positive urgency and impulsivity), it would prudent to include it in this proposed study (2013).

Decision-Making - Reactive & Proactive Aggression and Conduct Problems

Two previous studies have examined the interaction between decision-making, aggression, and conduct problems. Deming et al., examined the longitudinal effects of callous-unemotional traits, performance on the IGT, and skin conductance levels taken concurrently during the task. Deming and colleagues found that better performance on the IGT and increased skin conductance levels during the IGT predicted decreased conduct problem behaviors later (2014).

In another study, researchers examined whether parental factors could serve as a protective factor for impaired decision-making often observed in aggressive and conduct problem youth. Specifically, the relationship among aggressive and conduct problem children's affective decision-making, positive parenting practices, and children's aggressive and conduct problem behavior was examined longitudinally. Children with aggression and conduct problems displayed affective decision-making deficits on the IGT. Importantly, there was a significant relationship found between parenting monitoring and affective decision-making, indicating that parental monitoring served as a protective factor to help ameliorate poor decision-making in aggressive and conduct problem children (Powe, 2015).

Negative Parenting Behaviors

In theory, negative parent behaviors could act as a moderator for MAOA gene variants, aggression and conduct problem behavior, and decision-making, as the natural behavioral

differences between parenting practices can predict neurobiological changes in human and non-human offspring (Liu et al., 1997; Moffitt, Caspi, & Rutter, 2005).

Negative parenting behaviors could act as a risk factor, essentially compounding upon the risk associated with low MAOA, aggressive and conduct problem behavior, and decision-making deficits. This is especially important in light of the gene by environment interaction found by Caspi et al., where maltreated children with higher levels of MAOA were less likely to develop antisocial behaviors later in life (2002). For an overview of studies examining the association between MAOA and negative parenting practices, please see Appendix E.

Negative Parenting Practices

Negative parenting practices are often defined as being punitive in nature or as causing a more aversive or unpredictable environment in some way. On the Alabama Parenting Questionnaire (APQ), there are two separate scales measuring negative parenting practices; inconsistent discipline and corporal punishment (Shelton, Frick, & Wootton, 1996). Inconsistent discipline attempts to determine if a parent will follow through on a punishment after a threat has been given (i.e., if the parent states they will put the child in time-out, does the child actually go to time-out) and whether the punishment is consistent across similar behaviors (i.e., does the child always receive a 5-minute time-out for fighting with a sibling or does the amount of time vary). Corporal punishment attempts to determine if a parent uses any corporal punishment strategies, such as spanking, which is a known risk factor for aggression, conduct problems, and antisocial behavior, especially if these behaviors are used in early childhood (Dodge & Pettit, 2003; Ford et al., 2000; Dodge, Greenberg, & Malone, 2008).

Aggression and Conduct Problem Behavior. Previous studies have also examined the relationship between negative parenting practices, MAOA gene variants, and aggressive or

conduct problem behavior. Specifically, maternal punitiveness (i.e., corporal punishment and/or harsh discipline) at ages 1.5 and two years of age predicted more antisocial behavior at ages 20 and 15, respectively, among all low MAOA males (Choe, Shaw, Hyde, & Forbes, 2014). Whelan, Kretschmer, and Barker, also found a stronger association between harsh parenting and victimization in low MAOA males, but the relationship was not statistically significant (2014).

Decision-Making. To the author's knowledge, only one study has attempted to examine the relationship between negative parenting practices, MAOA gene variants, and decision-making behavior; however, decision-making was examined in an abstract manner rather than through a specific task. Choe, Shaw, Hyde, and Forbes found that African-American males with low MAOA and punitive mothers at age five had an increased number of juvenile petitions (i.e., exhibited poor decision-making due to increased law-breaking behavior; 2014).

CURRENT STUDY

The aim of this study is to examine how variants of the MAOA gene may moderate the relationship between negative parenting practices and three variables: (1) affective decision-making, (2) childhood reactive aggression, (3) childhood conduct problems, as no previous study has looked directly at these relationships. Specifically, the study is interested in answering the following questions: (1) whether the expression of low versus high MAOA genes in association with more reported negative parenting behaviors will moderate the expression of reactive aggression and conduct problems as reported by the child's teacher and (2) whether the expression of low versus high MAOA variants in association with more reported negative parenting behaviors will moderate the child's affective decision-making score and reaction time score on the IGT. Insufficient evidence did not permit hypotheses related to proactive aggression.

Hypotheses have been sub-divided into base models and moderation models, with further subdivision to include base model hypotheses addressing MAOA gene variants and base model hypotheses addressing negative parenting. Subsequent exploratory analyses will examine six exploratory models and research questions, divided into two sub-groups. One set of exploratory models will attempt to determine whether MAOA gene variants, negative parenting behaviors, and the moderation effect between MAOA gene variants and negative parenting behaviors will affect poor affective decision-making on reactive aggression and conduct problems. The other set of exploratory models will examine the effect of proactive aggression on the model's

moderation effects. Proactive aggression was removed from the base model hypotheses due to the insufficient prior literature's ability to serve as a base for prior hypotheses, and therefore proactive aggression will only be examined in an exploratory manner. This study hypothesizes the following:

MAOA with IGT Models and Hypotheses

1) MAOA Hypotheses
   a) Lower affective decision-making will only be associated with teacher reported reactive aggression in children with the low variant of the MAOA gene (Figure 1)

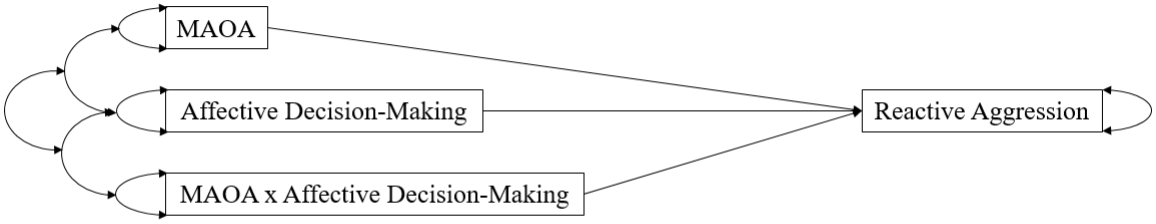

Figure 1
MAOA with IGT Model #1 (Hypothesis 1A)

   b) Faster reaction time scores will only be associated with teacher reported reactive aggression in children with the low variant of the MAOA gene (Figure 2).

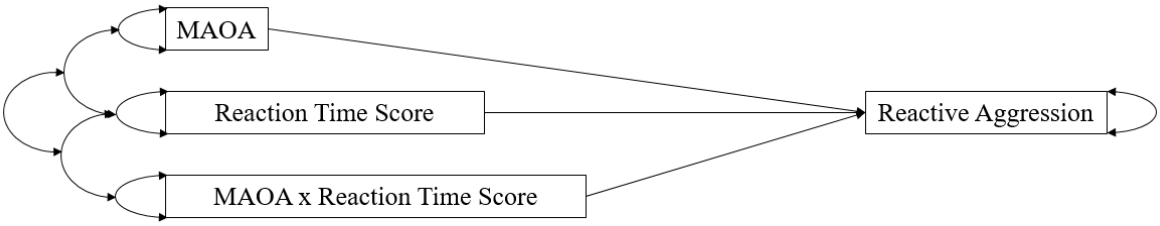

Figure 2
MAOA with IGT Model #2 (Hypothesis 1B)

   c) Lower affective decision-making will only be associated with teacher reported conduct problems in children with the low variant of the MAOA gene (Figure 3).

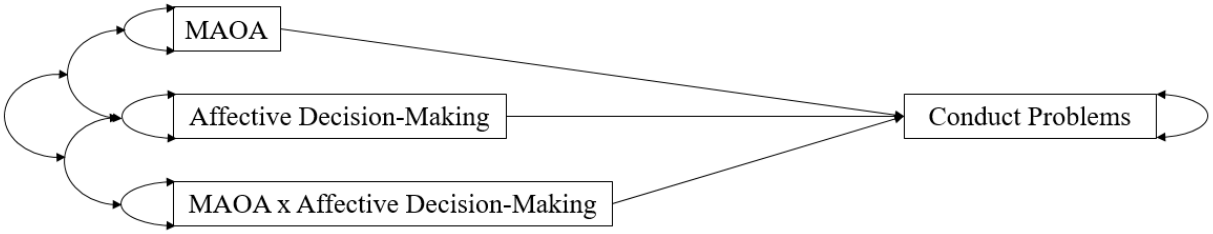

Figure 3
MAOA with IGT Model #3 (Hypothesis 1C)

d) Faster reaction time scores will only be associated with teacher reported conduct problems in children with the low variant of the MAOA gene (Figure 4).

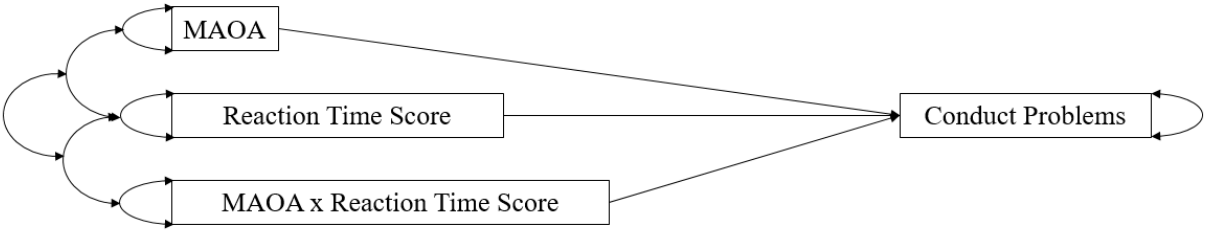

Figure 4
MAOA with IGT Model #4 (Hypothesis 1D)

Negative Parenting with IGT Models and Hypotheses

2) Negative Parenting Hypotheses

a) Lower affective decision-making will only be associated with teacher reported reactive aggression in children whose parents report higher levels of negative parenting behaviors (Figure 5).

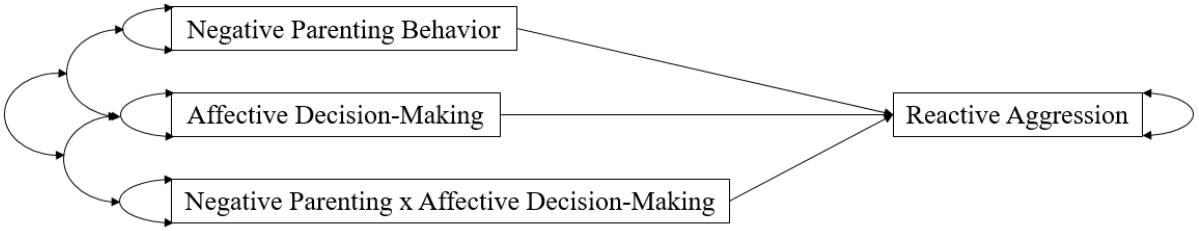

Figure 5
Negative Parenting with IGT Model #5 (Hypothesis 2A)

b) Faster reaction time scores will only be associated with teacher reported reactive aggression in children whose parents report higher levels of negative parenting behaviors (Figure 6).

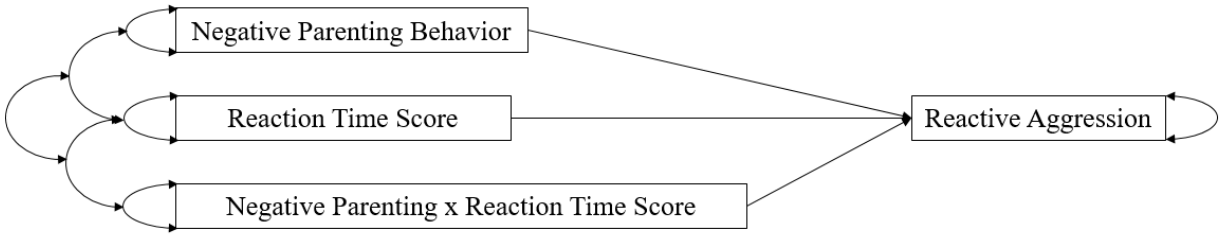

Figure 6
Negative Parenting with IGT Model #6 (Hypothesis 2B)

c) Lower affective decision-making scores will only be associated with teacher reported conduct problem behavior in children whose parents report higher levels of negative parenting behaviors (Figure 7).

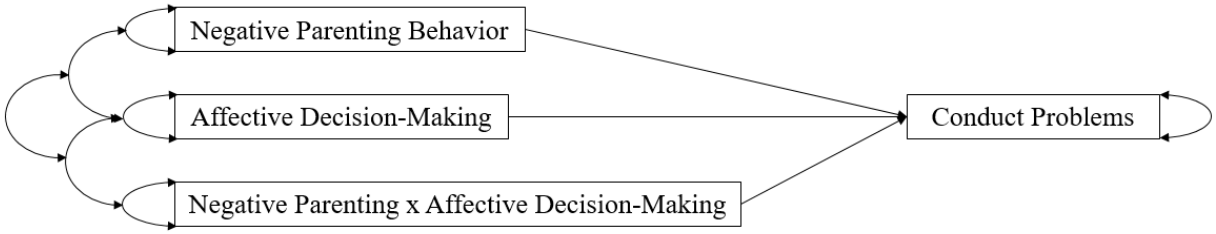

Figure 7
Negative Parenting with IGT Model #7 (Hypothesis 2C)

d) Faster reaction time scores will only be associated with teacher reported conduct problem behavior in children whose parents report higher levels of negative parenting behaviors (Figure 8).

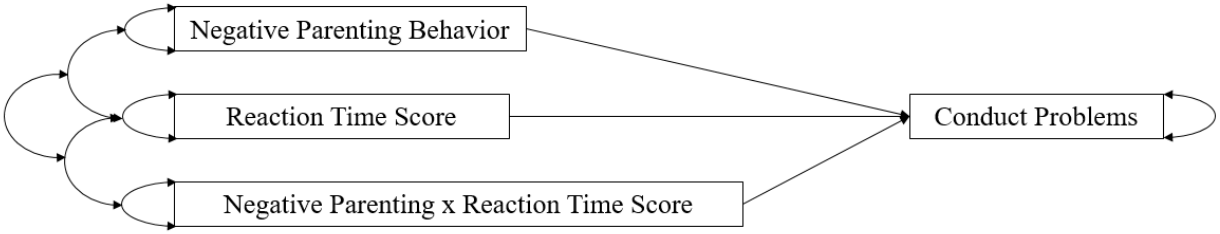

Figure 8
Negative Parenting with IGT Model #8 (Hypothesis 2D)

MAOA with Negative Parenting and IGT Models and Hypotheses

3) Lower affective decision-making scores will only be associated with parent reported negative parenting behaviors in children with the low MAOA variant (Figure 9).

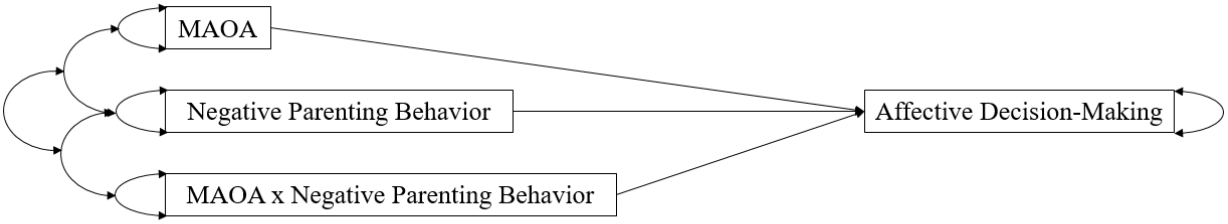

Figure 9
MAOA with Negative Parenting and IGT Model #1 (Hypothesis 3)

4) Faster reaction time scores will only be associated with parent reported negative parenting behaviors in children with the low MAOA variant (Figure 10).

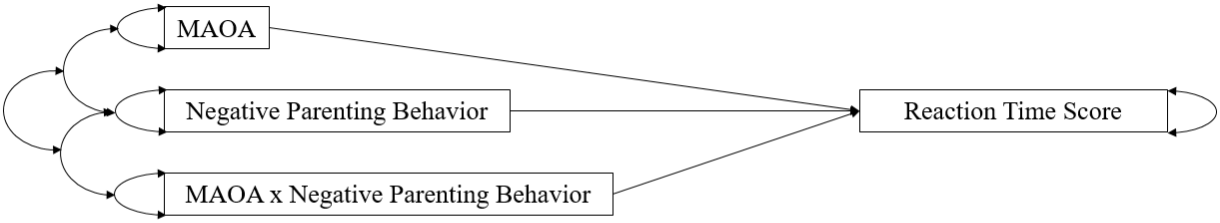

Figure 10
MAOA with Negative Parenting and IGT Model #2 (Hypothesis 4)

METHODS

This study will make use of the baseline sample from the individual versus group Coping Power study. In this study, elementary schools were randomly assigned to either group Coping Power (GCP) or individual Coping Power (ICP). Schools were matched on size, ethnic distribution, and eligibility for free and reduced lunch. There were 20 schools in the full sample (N=360 parent-child pairs), with 10 schools in the GCP condition and 10 schools in the ICP condition.

Participants

Children at each school were screen for at-risk externalizing behavior problems based upon their 4th grade teachers rating. Based upon these teacher ratings, children who fell at or above the cut off score, set at the 25th percentile, were considered eligible for participation. There were three annual cohorts, which resulted in a total sample size of 360 parent-child pairs. Please see Appendix F for sample characteristics.

As MAOA is an X-linked gene, males (XY) can more easily be classified as either low or high MAOA activity, as they are hemizygotes (i.e., carry one copy of the allele). Females (XX) carry two copies of the MAOA allele, and therefore can be homozygotes (i.e., carry two copies of the same allele) or heterozygotes (i.e., carry two different alleles; Whelan, Kretschmer, & Barker, 2014). Females who were homozygotes for the MAOA allele were eligible for this study, while females who were heterozygotes were not eligible.

Procedure and Measures

After informed consents were collected, the baseline measures were completed during the spring semester of the children's 4th grade year. Parents and children were interviewed separately, at their home or the Coping Power facilities, by a trained research blind to the child's condition. Baseline teacher assessments were completed in the spring of 4th grade. Baseline measures include the buccal swab, the Iowa Gambling Task, the Teacher Report of Proactive and Reactive Aggression, the Behavior Assessment System for Children-Teacher Rating Scales (BASC-TRS), and the Alabama Parenting Questionnaire.

Buccal Swab and Genetic Variants of MAOA. DNA was extracted from buccal cells collected from participants, stored in a refrigeration unit, and sent to a lab for genotyping. Participants were genotyped for six single-nucleotide polymorphisms (SNP), including MAOA, which is not defined as a SNP, but rather a genetic variant. The 30 bp VNTR in the promoter region of the MAOA gene was genotyped resulting in five possible fragment sizes that included 291, 321, 336, 351, and 381 bps. 336 (3.5-repeat) and 351 (4-repeat) and 381 (5-repeat) polymorphisms were coded as high activity and 291 (2-repeat) and 321 (3-repeat) were coded as low-activity.

The Iowa Gambling Task. During the administration of the IGT, a computer screen displays four decks of cards, equal in size and similar in appearance. The participant is given $2000 in play money and instructed that the game requires the participant to choose among the four decks until they are instructed to stop. After the participant makes a choice among the decks, the participant receives an amount of money and may be required to "pay" a penalty amount.

The IGT measures affective decision-making by presenting simulated real life decisions. Decks A and B are considered disadvantageous decks, giving the participant a higher reward and higher frequency penalties, creating a net loss. Decks C and D are considered advantageous decks, giving the participant a lower reward and lower frequency penalties, creating a net gain (Bechara, Damásio, Tranel, & Anderson, 1998). The goals of the task are to maximize the amount of money won by choosing from decks C and D, and minimize overall loss by avoiding decks A and B (Bechara, Damásio, Damásio, & Anderson, 1994).

As learning typically increases across the blocks, with Blocks 1 and 2 serving as an experimental phase, and Blocks 3, 4, and 5 serving as a more accurate representation of decision-making abilities, those blocks will be used in the analyses. The Affective Decision-Making score will be computed by adding advantageous deck decisions together and subtracting disadvantageous deck decision ((C+D)-(A+B)) for Trials 41-100 (Visagan, Xiang, & Lamar, 2012; Miu et al., 2012; Gansler, Jerram, Vannorsdall, & Schretlen, 2011; Smith, Xiao, & Bechara, 2012). The Reaction Time Score will be computed for Trials 41-100 (Smith, Xiao, & Bechara, 2012).

Teacher Report of Proactive and Reactive Aggression. The Teacher Report of Proactive and Reactive Aggression (Dodge, Lochman, Hamish, Bates, and Pettit, 1997) is a six-item measure designed to assess whether children are engaging in proactively aggressive behaviors (i.e., “This child uses physical force (or threatens to use physical force) in order to dominate other kids”) or reactively aggressive behaviors (i.e., “When this child has been teased or threatened he/she gets angry easily and strikes back”). Originally used in a study by Dodge and Coie, a later study conducted confirmatory factor analysis on this measure in which the two-factor model was supported ($\Delta\chi^2$ (2) = 42.76, $p < .01$; Fite, Colder, & Pelham Jr., 2006).

Behavior Assessment System for Children. BASC-TRS is a behavior problem checklist that will be completed by the child's individual teacher (Lochman, Boxmeyer, Powell, Qu, Wells, & Windle, 2009). The conduct problems subscale will be the only subscale used from this measure. A covariance structure analysis was conducted by the creators of the BASC, which determined that conduct problems loaded onto externalizing problems, with a factor loading of .92. In addition to this covariance structure analysis, the creators also conducted a principal-axis factor analysis, which indicated externalizing problems was a primary factor, with aggression, hyperactivity, and conduct problems loading onto it significantly. The conduct problems scale was also significantly correlated with other measures including the Achenbach System of Empirically Based Assessment and the previous version of the BASC (Reynolds & Kamphaus, 2004).

Alabama Parenting Questionnaire. The APQ has 42 questions for which the primary caretaker responds how often each item typically occurs in their home environment on a 5-point scale (Shelton, Frick, & Wootton, 1996). Research has shown that the five constructs measured in the APQ can be collapsed into two separate composites: positive parenting composite and a negative parenting composite composed of inconsistent discipline and corporal punishment scales (Frick, Christian, & Wootton, 1999; Frick, Kimonis, Dandreaux, & Farrell, 2003; Shelton et al., 1996). To create the negative parenting composites, the subscales are converted to *z* scores, and then added together within their respective composite group (Barry, Frick, Grafeman, 2008). These composites have been supported by both exploratory factor analysis ($\Delta\chi^2$ (171) = 1921.83, $p < .0001$) and confirmatory factor analysis research ($\Delta\chi^2$ (1, n = 395) = 83.58, $p < .01$; Randolph and Radey, 2011).

Data Analysis

Preliminary data analyses will include the creation of composite scores, the creation of a correlation matrix for IGT scores, Teacher Report of Proactive and Reactive Aggression scores, BASC-TRS scores, and APQ scores, and an assessment of the reliability of each measure.

Exploratory analyses will include six models analyzed through path analysis, designed to measure the interplay between MAOA gene variants, negative parenting behaviors, IGT scores, reactive aggression, proactive aggression, and conduct problems. Subsequent evaluations will examine the model fit for each model, and modify the pathways based upon the modification indices.

The data will primarily be analyzed through path analysis, within two groups, base models and moderation models. Base models will examine the interaction between MAOA gene variants and IGT scores, and the interaction between negative parenting behaviors and IGT scores. Moderation models will examine the interaction between MAOA gene variants and negative parenting behaviors, and the difference between parents who report low or high levels of negative parenting behaviors.

MAOA with IGT and Negative Parenting with IGT: Models and Hypotheses. Eight separate base models have been specified and determined to be identifiable in this study, as seen in Figures 1, 2, 3, 4, 5, 6, 7, and 8. These models will test whether MAOA gene variants, affective decision-making, reaction time scores, and negative parenting have a direct effect on reactive aggression. These models will also test whether the interaction between MAOA gene variants and affective decision-making, MAOA gene variants and reaction time scores, negative parenting behaviors and affective decision-making, and negative parenting behaviors and reaction time score have an effect on reactive aggression.

MAOA with Negative Parenting and IGT: Models and Hypotheses. Two separate models have also been specified and determined to be identifiable in this study, as seen in Figures 9 and 10. These models will test whether MAOA gene variants and negative parenting behaviors will have a direct effect on affective decision-making and reaction time scores. The interaction between MAOA gene variants and negative parenting behaviors and the interaction's effect on affective decision-making and reaction time scores will also be tested.

Exploratory Models. Nine separate exploratory models have been specified and determined to be identifiable in this study, as seen in Figures 11, 12, 13, 14, 15, 16, 17, 18, and 19. The exploratory models have been sub-divided into two groups based upon the purpose of each model. One set will examine the ability of the interaction between negative parenting and MAOA gene variants to moderate the expression of proactive aggression, reactive aggression, and conduct problems in children (Figures 11, 12, and 13).

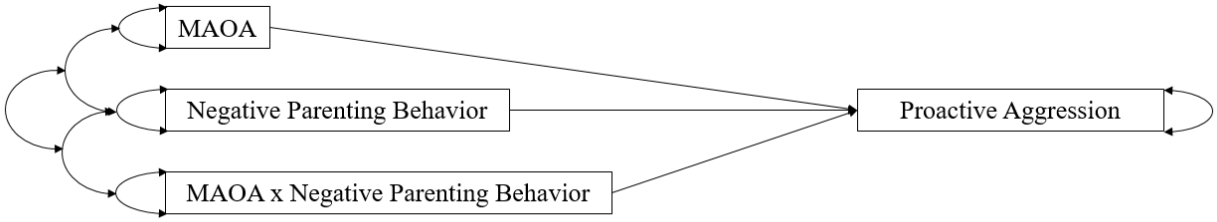

Figure 11
Proactive Moderation Model

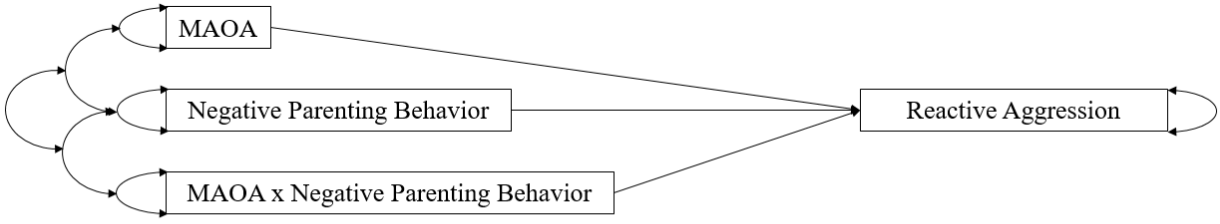

Figure 12
Reactive Moderation Model

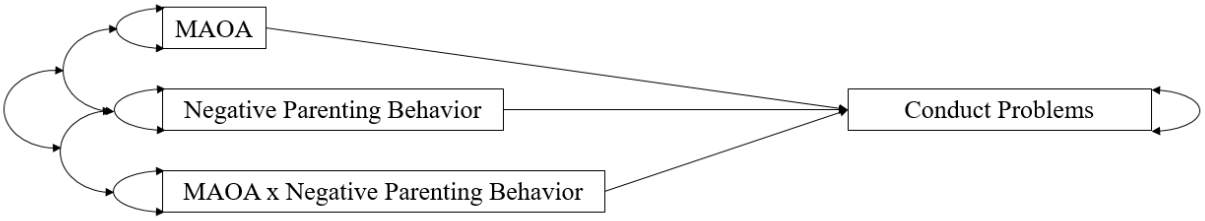

Figure 13
Conduct Problems Moderation Model

Another set will examine the ability of the variables involved in each model to moderate the expression of reactive aggression, conduct problems, and proactive aggression (Figures 14, 15, 16, 17, 18, and 19). Proactive aggression was removed from base model analyses due to insufficient literature, and will only be examine in these exploratory models.

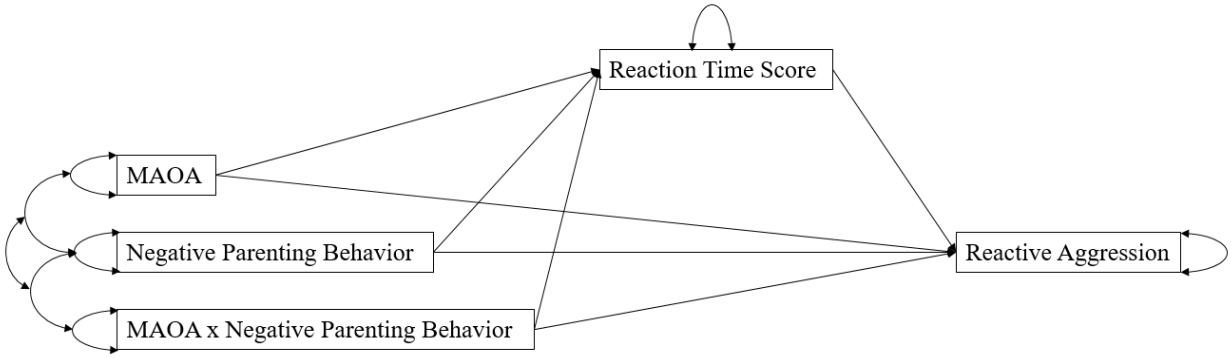

Figure 14
Total Model #1

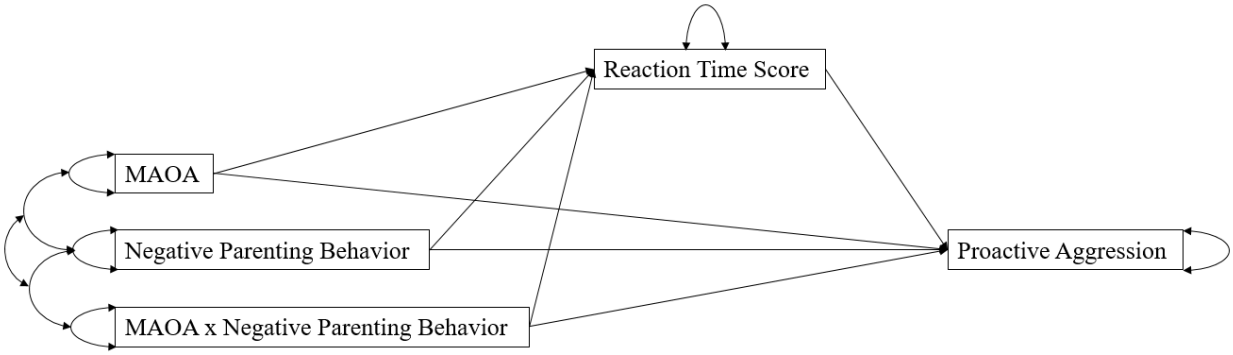

Figure 15
Total Model #2

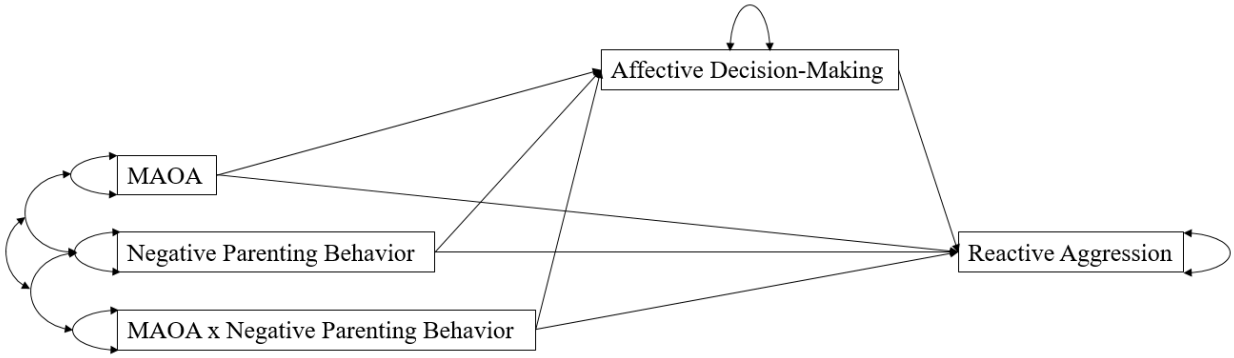

Figure 16
Total Model #3

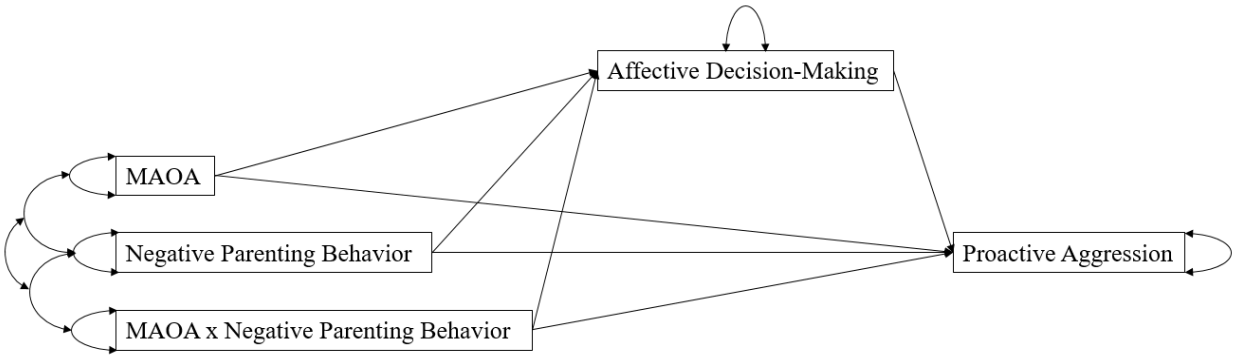

Figure 17
Total Model #4

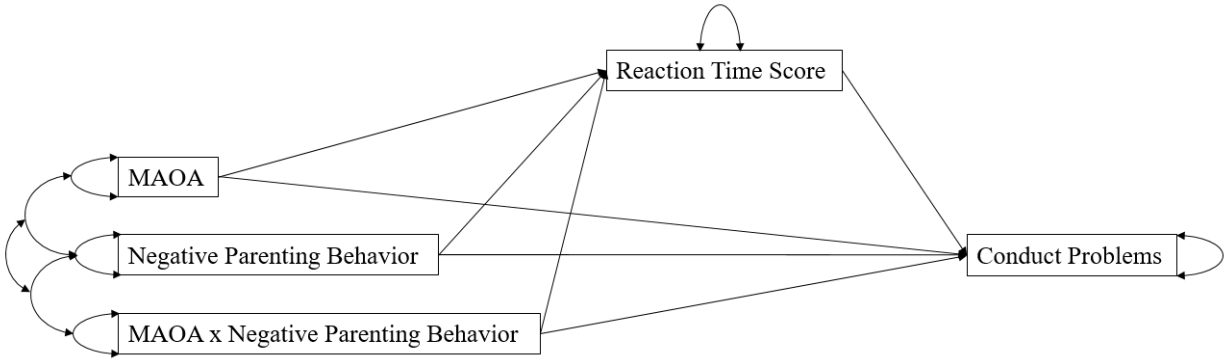

Figure 18
Total Model #5

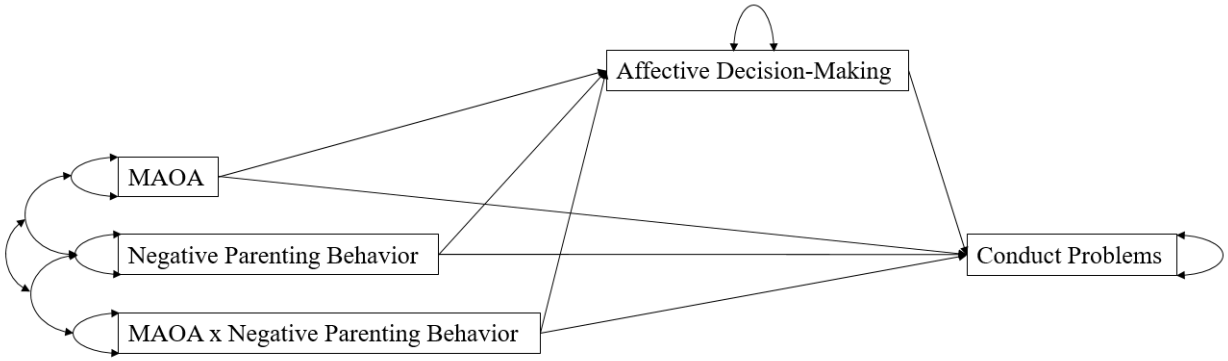

Figure 19
Total Model #6

As previously stated each of these exploratory models will incorporate a different pattern of variables from the study (i.e., choice score, reaction time score, reactive aggression, proactive aggression, conduct problems, and negative parenting behaviors), allowing the researcher to examine the interactions among the variables and the effect the combination of variables will have upon the specified paths. While some paths appear across models, it should be noted that due to the changing variables, the nature of the path will change, and a path, which is significant in one model, may not be significant in another. As such, it is unlikely that similar paths could be compared across models in a meaningful manner.

Sample Size. Typically, in path analysis approaches, larger sample sizes are required to draw accurate conclusions from specified models; however, there is no clear rule that establishes a necessary sample size for analyses (Keith, 2005). Within path analysis, sample size is not a linear function, and as such, different techniques to determine adequate sample size will return different sample size requirements. However, research-based guidelines have been outlined to help researchers determine adequate sample sizes for path analysis. For example, one such guideline suggests that an adequate sample size for path analysis is 100 to 150 participants at minimum (Ding, Veliver, & Harlow, 1995). Others have suggested that a sample size of 100 is small, a sample size between 100 and 200 is medium, and a sample size over 200 is considered

large (Kline, 2005). Therefore, based upon these guidelines, the sample size used in the primary analyses (n = 142) is considered to be a medium sample according to Kline and over the minimum requirement according to Ding and colleagues (2005; 1995).

RESULTS

This data was analyzed using path analysis using Mplus. Preliminary analyses were conducted to ensure no violation of the assumption of normality, linearity, multicollinearity, homoscedasticity, skewness, kurtosis, and outliers. Variables that had overly large variance were converted to z-scores to reduce the variance to one. In some analyses, the models were modified to create a best-fit model, where pathways between variables were added to increase the model's fit indices. Regarding missing data, missing data rates are noted in Table 1. Additionally, this study made use of the Maximum Likelihood estimation method within Mplus.

Table 1
Variable Characteristics

	N	Minimum	Maximum	Mean	Std. Deviation	Skewness		Kurtosis		Missing
						Statistic	SE	Statistic	SE	
Participant Gender	157	1.00	2.00	1.2994	0.45944	0.885	0.194	-1.233	0.385	0
MAOA – High vs. Low	157	0.00	1.00	0.6688	0.47215	-0.724	0.194	-1.495	0.385	0
Zygote Classification	157	1.00	3.00	2.4013	0.91889	-0.885	0.194	-1.233	0.385	0
Conduct Problems	156	-23.36	44.64	0.0000	15.04571	0.838	0.194	0.168	0.386	1
Parental Involvement	143	-2.83	1.90	0.0000	1.0000	-0.319	0.203	-0.281	0.403	14
Positive Parenting	143	-2.62	1.23	0.0000	1.0000	-0.738	0.203	-0.371	0.403	14
Parental Monitoring	143	-0.96	3.60	0.0000	0.9999	1.402	0.203	1.460	0.403	14
Inconsistent Discipline	143	-2.10	2.82	0.0000	1.0000	-0.117	0.203	-0.431	0.403	14
Corporal Punishment	143	-1.82	2.19	0.0000	0.9999	-0.244	0.203	-0.524	0.403	14
APQ Positive Parenting Total Score	143	-5.10	3.13	0.0000	1.83983	-0.616	0.203	-0.263	0.403	14
APQ Negative Parenting Total Score	143	-4.39	5.11	0.0000	1.98569	0.248	0.203	-0.264	0.403	14
TRRPA Proactive Aggression	157	-7.89	4.11	0.0000	2.61303	-0.177	0.194	-0.361	0.385	0
TRRPA Reactive Aggression	157	-5.03	6.97	0.0000	3.24817	0.495	0.194	-0.597	0.385	0

	N	Minimum	Maximum	Mean	Std. Deviation	Skewness		Kurtosis		Missing
						Statistic	SE	Statistic	SE	
IGT Affective Decision-Making	157	-49.465	58.535	-0.00003185	14.2309381	0.244	0.194	4.496	0.385	0
IGT Reaction Time	157	-1.37	5.46	0.0000	1.0000	1.656	0.194	5.165	0.385	0
MAOA by Affective Decision-Making	157	-49.47	58.54	-0.7951	11.53205	0.090	0.194	9.165	0.385	0
MAOA by Reaction Time	157	-1710.07	3862.18	33.8572	983.41128	0.925	0.194	1.514	0.385	0
MAOA by Negative Parenting	148	-4.39	5.11	0.1068	1.64153	0.362	0.199	1.149	0.396	14
Negative Parenting by Affective Decision-Making	143	-144.84	65.05	-2.6370	24.16307	-2.145	0.203	10.904	0.403	14
Negative Parenting by Reaction Time	143	-4.07	3.53	0.0000	1.0000	-0.102	0.203	3.475	0.403	14
Affective Decision Making – High and Low	157	0.00	1.00	0.4395	0.49791	0.246	0.194	-1.965	0.385	0
Valid N (listwise)	142									

Preliminary Analyses

Preliminary analyses began with the creation of composite scores and interaction variables. Variables used in the creation of interaction scores were centered to the mean prior to the creation of interaction scores. A correlation table highlighting relationships among the variables was then created. Please see Table 27 in Appendix G for more information. The Cronbach's alpha was also calculated for each scale (BASC = 0.734, APQ = 0.706, TRRPA = 0.867)

Primary Analyses – MAOA

The following models are concerned with hypothesis one. Due to the models being just-identified (i.e., there are no degrees of freedom), fit statistics are not reported.

MAOA Analyses – Hypothesis 1A. In the first model (1a), paths were drawn from MAOA gene variants, Affective Decision-Making, and an interaction term MAOA gene variants

x Affective Decision-Making to Reactive Aggression. Paths were also drawn between MAOA gene variants, Affective Decision-Making, and MAOA gene variants x Affective Decision-Making. As noted below, there was one significant path from MAOA gene variants by Affective Decision-Making to Reactive Aggression (Estimate = 0.306, $p = 0.021$, n = 157). The variable Affective Decision-Making was converted into a high and low variable, with a median split, to probe the significant interaction effect. A bar graph was created to analyze the impact of MAOA gene variants x Affective Decision-Making on Reactive Aggression, see Figure 20 below.

The bar graph indicates that high affective decision-making, with both lower and higher levels of MAOA, predict to higher levels of reactive aggression, though this pattern is more pronounced for low levels of MAOA. Lower levels of affective decision-making and higher levels of MAOA predict to lower levels of reactive aggression, while lower levels of MAOA and higher levels of affective decision making predict to the highest levels of reactive aggression.

Table 2
MAOA Analysis – Hypothesis 1A

	Estimate	S.E.	Est./S.E.	*p*
Reactive Aggression on				
MAOA	-0.076	0.078	-0.964	0.335
ADM	-0.140	0.135	-1.036	0.300
MxADM	0.306	0.132	2.313	0.021
Affective Decision-Making with				
MAOA	-0.119	0.079	-1.514	0.130
MxADM	0.814	0.027	30.270	0.000
MAOA x ADM with				
MAOA	-0.049	0.080	-0.611	0.541

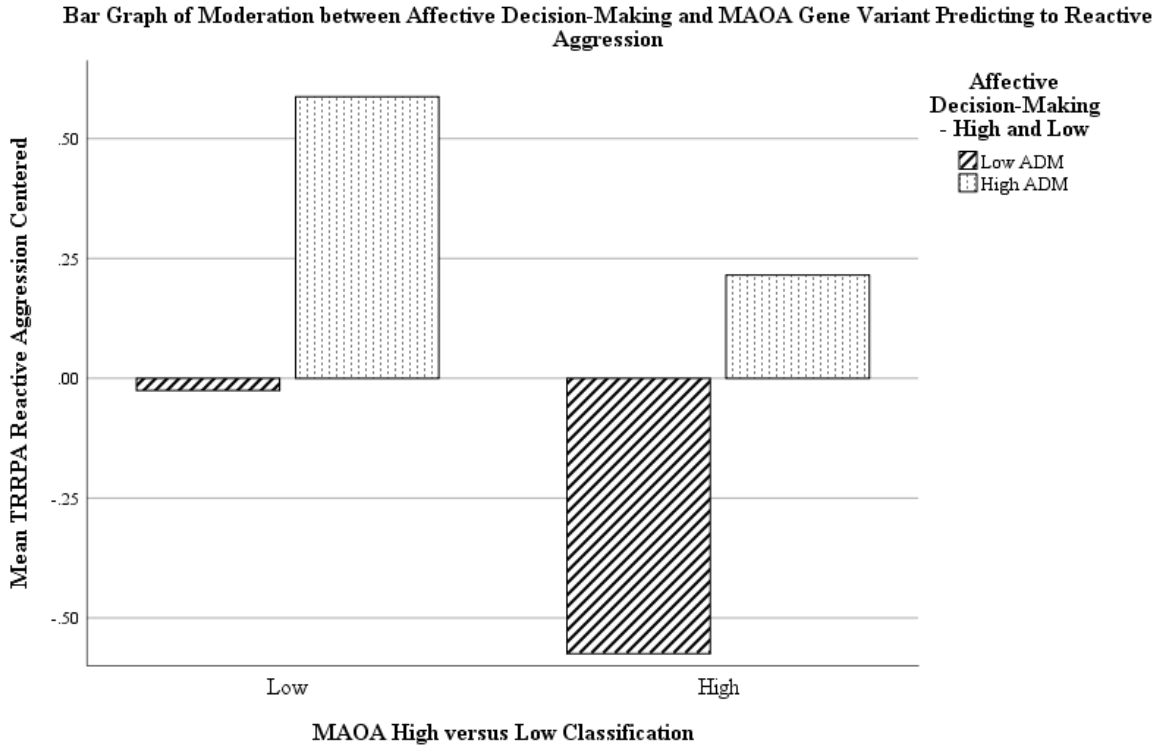

Figure 20
Moderation – MAOA x ADM Predicting to Reactive Aggression

MAOA Analyses – Hypothesis 1B. In the second model (1b), paths were drawn from MAOA gene variants, Reaction Time Score, and the interaction variable, MAOA gene variants x Reaction Time Score to Reactive Aggression. Paths were also drawn between MAOA gene variants, Reaction Time Score, and MAOA gene variants x Reaction Time Score. As noted in the table below, none of the direct paths were significant in this model (n = 157).

Table 3
MAOA Analysis – Hypothesis 1B

	Estimate	SE	Est./SE	*p*
Reactive Aggression on				
MAOA	-0.066	0.079	-0.830	0.406
RTS	-0.169	0.128	-1.322	0.186
MAOA x RTS	0.061	0.128	0.473	0.636
Reaction Time Score with				
MAOA	0.058	0.080	0.729	0.466
MAOA x RTS	0.788	0.030	26.017	0.000
MAOA x RTS with				
MAOA	0.024	0.080	0.305	0.761

MAOA Analyses – Hypothesis 1C. In the third model (1c), paths were drawn from MAOA gene variants, Affective Decision-Making, and an interaction variable, MAOA gene variants x Affective Decision-Making to Conduct Problems. Paths were also drawn between MAOA gene variants, Affective Decision-Making, and MAOA gene variants x Affective Decision-Making. The goodness of fit indices for this model are not reported due to the model being just-identified. The path from MAOA gene variants x Affective Decision-Making and Conduct Problems was also significant (Estimate = 0.276, $p = 0.041$, n = 157). The variable Affective Decision-Making was converted into a high and low variable, with a median split, to probe the significant interaction effect. A bar graph was created to analyze the impact of MAOA gene variants x Affective Decision-Making on Conduct Problems, see Figure 5 below.

This bar graph suggests that low affective decision-making and low levels of MAOA predict to higher levels of teacher reported conduct problems. Low affective decision-making and higher levels of MAOA predict to lower levels of teacher reported conduct problems.

The bar graph indicates that high affective decision-making and lower levels of MAOA predict to lower levels of teacher reported conduct problems. High affective decision-making and higher levels of MAOA predict to higher levels of teacher reported conduct problems.

Table 4
MAOA Analysis – Hypothesis 1C

	Estimate	SE	Est./SE	*p*
Conduct Problems on				
MAOA	-0.017	0.080	-0.217	0.828
ADM	-0.222	0.136	-1.633	0.102
MAOA x ADM	0.276	0.135	2.046	0.041
Affective Decision-Making with				
MAOA	-0.119	0.079	-1.514	0.130
MAOA x ADM	0.814	0.027	30.270	0.000
MAOA x ADM with				
MAOA	-0.049	0.080	-0.611	0.541

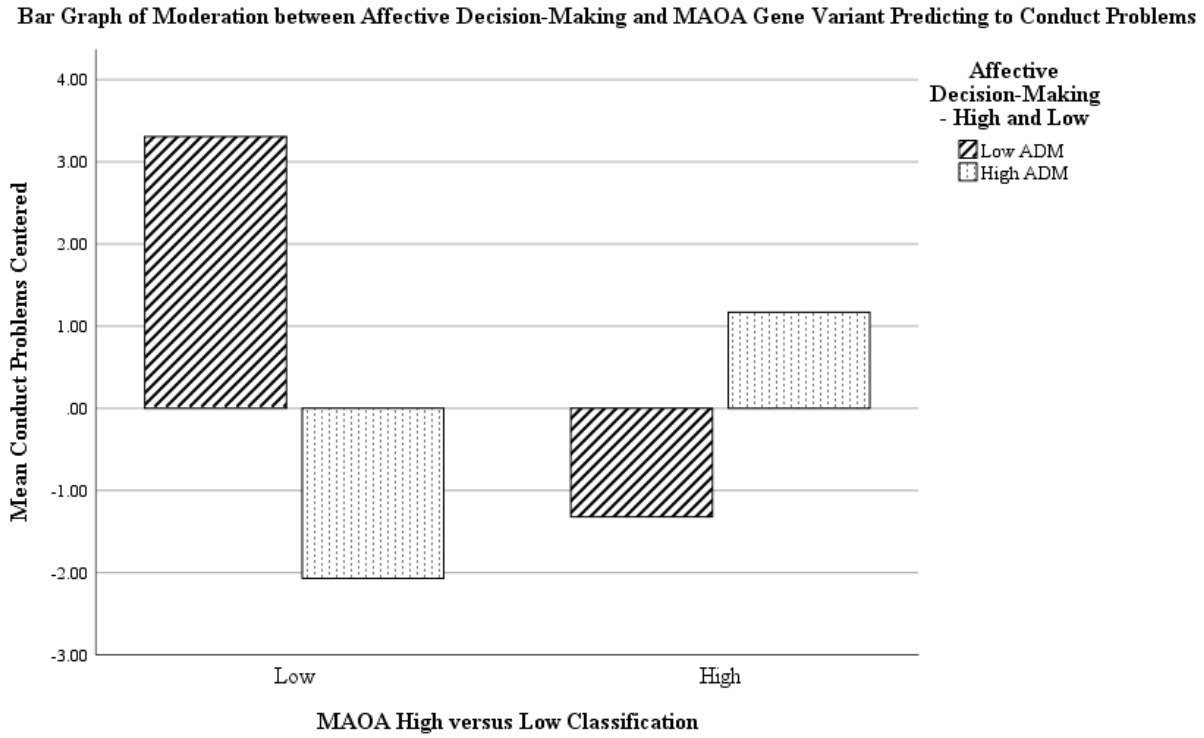

Figure 21
Interaction – MAOA x ADM Predicting to Conduct Problems

MAOA Analyses – Hypothesis 1D. In the fourth model (1d), paths were drawn from MAOA gene variants, Reaction Time Score, and the interaction variable MAOA gene variants x Reaction Time Score to Conduct Problems. As noted in the table below, none of the direct paths were significant (n = 157).

Table 5
MAOA Analysis – Hypothesis 1D

	Estimate	SE	Est./SE	*p*
Conduct Problems on				
MAOA	-0.005	0.080	-0.065	0.948
RTS	-0.023	0.130	-0.177	0.860
MAOA x RTS	0.078	0.129	0.602	0.547
Reaction Time Score with				
MAOA	0.058	0.080	0.729	0.466
MAOA x RTS	0.788	0.030	26.017	0.000
MAOA x RTS with				
MAOA	0.024	0.080	0.305	0.761

Primary Models – Negative Parenting

The following models are concerned with hypothesis two. Due to the models being just-identified, fit statistics are not reported.

Negative Parenting Analyses – Hypothesis 2A. In the fifth model (2a), paths were drawn from Negative Parenting Behavior, Affective Decision-Making, and the interaction variable Negative Parenting x Affective Decision-Making to Reactive Aggression. Additional paths between Affective Decision-Making and Negative Parenting x Affective Decision-Making were also drawn. Modification indices suggested adding paths between Affective Decision-Making and Negative Parenting, as well as Negative Parenting with Negative Parenting x Affective Decision-Making. As noted below, none of the direct paths were significant in this model (n = 157).

Table 6
Negative Parenting Analysis – Hypothesis 2A

	Estimate	SE	Est./SE	*p*
Reactive Aggression on				
NPT	0.031	0.085	0.367	0.714
ADM	0.122	0.079	1.536	0.125
NPT x ADM	0.005	0.085	0.061	0.952
Affective Decision-Making with				
NPT x ADM	-0.089	0.083	-1.065	0.287
NPT	-0.096	0.083	-1.150	0.250
Negative Parenting Total with				
NPT x ADM	-0.059	0.083	-0.702	0.482

Negative Parenting Analyses – Hypothesis 2B. In the sixth model (2b), paths were drawn from Negative Parenting, Reaction Time Score, and the interaction variable Negative Parenting x Reaction Time Score to Reactive Aggression. Additional paths between Reaction Time Score and Negative Parenting x Reaction Time Score were also drawn. Modification indices suggested adding additional paths between Negative Parenting and Reaction Time Score,

and Negative Parenting and Negative Parenting x Reaction Time Score. As noted in the table below, none of the direct paths were significant, (n = 157).

Table 7
Negative Parenting Analysis – Hypothesis 2B

	Estimate	SE	Est./SE	*p*
Reactive Aggression on				
NPT	0.030	0.084	0.351	0.726
RTS	-0.131	0.079	-1.655	0.098
NPT x RTS	-0.046	0.084	-0.549	0.583
Reaction Time Score with				
NPT x RTS	-0.068	0.082	-0.831	0.406
NPT	0.092	0.082	1.128	0.259
Negative Parenting Total with				
NPT x RTS	0.032	0.084	0.384	0.701

Negative Parenting Analyses – Hypothesis 2C. In the seventh model (2c), paths were drawn from Negative Parenting Behavior, Affective Decision-Making, and the interaction variable Negative Parenting x Affective Decision-Making to Conduct Problems. Additional paths between Affective Decision-Making and Negative Parenting x Affective Decision-Making were also drawn. Modification indices suggested adding paths between Affective Decision-Making and Negative Parenting, as well as Negative Parenting with Negative Parenting x Affective Decision-Making. As noted in the table below, none of the direct paths were significant (n = 157).

Table 8
Negative Parenting Analysis – Hypothesis 2C

	Estimate	SE	Est./SE	*p*
Conduct Problems on				
NPT	0.131	0.082	1.607	0.108
ADM	0.024	0.080	0.298	0.765
NPT x ADM	0.070	0.082	0.854	0.393
Affective Decision-Making with				
NPT x ADM	-0.090	0.083	-1.081	0.280
NPT	-0.097	0.083	-1.170	0.242
Negative Parenting Total with				
NPT x ADM	-0.059	0.083	-0.703	0.482

Negative Parenting Analyses – Hypothesis 2D. In the eighth model (2d), paths were drawn from Negative Parenting Behavior, Reaction Time Score, and the interaction variable Negative Parenting x Reaction Time Score to Conduct Problems. Additional paths between Reaction Time Score and Negative Parenting x Reaction Time Score were also drawn. Modification indices suggested adding paths between Reaction Time Score and Negative Parenting, as well as Negative Parenting with Negative Parenting x Reaction Time Score. As noted in the table below, none of the direct paths were significant (n = 157).

Table 9
Negative Parenting Analysis – Hypothesis 2D

	Estimate	SE	Est./SE	*p*
Conduct Problems on				
NPT	0.117	0.082	1.431	0.152
RTS	0.031	0.080	0.388	0.698
NPT x RTS	0.042	0.082	0.518	0.604
Reaction Time Score with				
NPT x RTS	-0.069	0.082	-0.842	0.400
NPT	0.090	0.082	1.100	0.271
Negative Parenting Total with				
NPT x RTS	0.032	0.083	0.384	0.701

Primary Models – Moderation

The following models are concerned with hypothesis three and four. Due to the models being just-identified, fit statistics have not been reported.

Moderation Analyses – Hypothesis 3. In the ninth model (3), paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Affective Decision-Making. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted below, there were no significant direct paths in this model (n = 157).

Table 10
Moderation Analysis – Hypothesis 3

		Estimate	SE	Est./SE	*p*
Affective Decision-Making on					
	MAOA	-0.109	0.080	-1.372	0.170
	NPT	-0.082	0.154	-0.534	0.593
	MAOA x NPT	0.002	0.152	0.012	0.990
MAOA with					
	MAOA x NPT	0.047	0.081	0.583	0.560
Negative Parenting Total with					
	MAOA x NPT	0.840	0.024	34.614	0.000
	MAOA	0.121	0.081	1.500	0.134

Moderation Analyses – Hypothesis 4. In the tenth model (4), paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Reaction Time Score. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted below, there were no significant direct paths in this model (n = 157).

Table 11
Moderation Analysis – Hypothesis 4

		Estimate	SE	Est./SE	*p*
Reaction Time Score on					
	MAOA	0.055	0.080	0.689	0.491
	NPT	-0.030	0.152	-0.198	0.843
	MAOA x NPT	0.139	0.151	0.925	0.355
MAOA with					
	MAOA x NPT	0.046	0.081	0.573	0.566
Negative Parenting Total with					
	MAOA x NPT	0.840	0.024	34.631	0.000
	MAOA	0.120	0.081	1.481	0.139

Exploratory Models – Proactive, Reactive, and Conduct Problems Moderation Models

The following models are exploratory models are not directly linked to any of the hypothesis. Due to the models being just-identified fit statistics have not been reported.

Proactive Moderation Model. In the first exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Proactive Aggression. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 12
Proactive Moderation Model

		Estimate	SE	Est./SE	*p*
Proactive Aggression on					
	MAOA	-0.062	0.081	-0.767	0.443
	NPT	0.061	0.154	0.396	0.692
	MAOA x NPT	-0.018	0.153	-0.116	0.908
MAOA with					
	MAOA x NPT	0.048	0.081	0.598	0.550
Negative Parenting Total with					
	MAOA x NPT	0.841	0.024	34.631	0.000
	MAOA	0.122	0.081	1.511	0.131

Reactive Moderation Model. In the second exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Reactive Aggression. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 13
Reactive Moderation Model

		Estimate	SE	Est./SE	*p*
Reactive Aggression on					
	MAOA	-0.072	0.080	-0.902	0.367
	NPT	-0.051	0.158	-0.323	0.747
	MAOA x NPT	0.097	0.156	0.623	0.533
MAOA with					
	MAOA x NPT	0.049	0.081	0.610	0.542
Negative Parenting Total with					
	MAOA x NPT	0.841	0.024	34.668	0.000
	MAOA	0.121	0.081	1.501	0.133

Conduct Problems Moderation Model. In the third exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Conduct Problems. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 14
Conduct Problems Moderation Model

		Estimate	SE	Est./SE	*p*
Conduct Problems on					
	MAOA	-0.021	0.080	-0.255	0.798
	NPT	0.134	0.152	0.883	0.377
	MAOA x NPT	-0.007	0.151	-0.049	0.961
MAOA with					
	MAOA x NPT	0.049	0.081	0.601	0.548
Negative Parenting Total with					
	MAOA x NPT	0.840	0.024	34.623	0.000
	MAOA	0.123	0.081	1.521	0.128

Total Model #1. In the fourth exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Reaction Time Score. Paths were also drawn from Reaction Time Score,

MAOA gene variants, Negative Parenting Behavior, and the interaction variable to Reactive Aggression. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 15
Total Model #1

		Estimate	SE	Est./SE	*p*
Reactive Aggression on					
	RTS	-0.129	0.079	-1.640	0.101
	MAOA	-0.065	0.080	-0.818	0.413
	NPT	-0.055	0.157	-0.352	0.752
	MAOA x NPT	0.115	0.155	0.739	0.460
Reaction Time Score on					
	MAOA	0.055	0.080	0.686	0.493
	NPT	-0.030	0.152	-0.197	0.844
	MAOA x NPT	0.139	0.151	0.920	0.358
MAOA with					
	MAOA x NPT	0.049	0.081	0.601	0.548
Negative Parenting Total with					
	MAOA x NPT	0.841	0.024	34.665	0.000
	MAOA	0.120	0.081	1.490	0.136

Total Model #2. In the fifth exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Reaction Time Score. Paths were also drawn from Reaction Time Score, MAOA gene variants, Negative Parenting Behavior, and the interaction variable to Proactive Aggression. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 16
Total Model #2

		Estimate	SE	Est./SE	*p*
Proactive Aggression on					
	RTS	-0.111	0.079	-1.397	0.162
	MAOA	-0.055	0.080	-0.692	0.489
	NPT	0.058	0.152	0.382	0.702
	MAOA x NPT	-0.007	0.152	-0.048	0.961
Reaction Time Score on					
	MAOA	0.055	0.080	0.690	0.490
	NPT	-0.032	0.152	-0.211	0.833
	MAOA x NPT	0.139	0.151	0.925	0.355
MAOA with					
	MAOA x NPT	0.047	0.081	0.585	0.558
Negative Parenting Total with					
	MAOA x NPT	0.840	0.024	34.614	0.000
	MAOA	0.121	0.081	1.500	0.134

Total Model #3. In the sixth exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Affective Decision-Making Score. Paths were also drawn from Affective Decision-Making Score, MAOA gene variants, Negative Parenting Behavior, and the interaction variable to Reactive Aggression. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 17
Total Model #3

		Estimate	SE	Est./SE	*p*
Reactive Aggression on					
	ADM	0.115	0.079	1.446	0.148
	MAOA	-0.060	0.080	-0.748	0.454
	NPT	-0.039	0.158	-0.245	0.806
	MAOA x NPT	0.094	0.155	0.606	0.545
Affective Decision-Making on					
	MAOA	-0.109	0.080	-1.372	0.170
	NPT	-0.081	0.154	-0.525	0.600
	MAOA x NPT	0.002	0.152	0.010	0.992
MAOA with					
	MAOA x NPT	0.049	0.081	0.608	0.543
Negative Parenting Total with					
	MAOA x NPT	0.841	0.024	34,643	0.000
	MAOA	0.122	0.081	1.511	0.131

Total Model #4. In the seventh exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Affective Decision-Making Score. Paths were also drawn from Affective Decision-Making Score, MAOA gene variants, Negative Parenting Behavior, and the interaction variable to Proactive Aggression. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 18
Total Model #4

		Estimate	S.E.	Est./S.E.	*p*
Proactive Aggression on					
	ADM	-0.003	0.080	-0.039	0.969
	MAOA	-0.062	0.081	-0.769	0.442
	NPT	0.062	0.154	0.401	0.688
	MAOA x NPT	-0.018	0.153	-0.120	0.905
Affective Decision-Making on					
	MAOA	-0.109	0.080	-1.369	0.171
	NPT	-0.083	0.154	-0.539	0.590
	MAOA x NPT	0.002	0.152	0.016	0.987
MAOA with					
	MAOA x NPT	0.048	0.081	0.593	0.553
Negative Parenting Total with					
	MAOA x NPT	0.840	0.024	34.606	0.000
	MAOA	0.122	0.081	1.517	0.129

Total Model #5. In the eighth exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Reaction Time Score. Paths were also drawn from Reaction Time Score, MAOA gene variants, Negative Parenting Behavior, and the interaction variable to Conduct Problems. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 19
Total Model #5

		Estimate	SE	Est./SE	*p*
Conduct Problems on					
	RTS	0.029	0.080	0.363	0.717
	MAOA	-0.022	0.080	-0.272	0.786
	NPT	0.136	0.152	0.893	0.372
	MAOA x NPT	-0.015	0.152	-0.097	0.923
Reaction Time Score on					
	MAOA	0.055	0.080	0.691	0.489
	NPT	-0.033	0.152	-0.216	0.829
	MAOA x NPT	0.139	0.151	0.924	0.356
MAOA with					
	MAOA x NPT	0.047	0.081	0.586	0.558
Negative Parenting Total with					
	MAOA x NPT	0.840	0.024	34.600	0.000
	MAOA	0.122	0.081	1.507	0.132

Total Model #6. In the ninth exploratory model, paths were drawn from MAOA gene variants, Negative Parenting Behavior, and the interaction variable MAOA gene variants x Negative Parenting to Affective Decision-Making Score. Paths were also drawn from Affective Decision-Making Score, MAOA gene variants, Negative Parenting Behavior, and the interaction variable to Conduct Problems. An additional path between MAOA gene variants and MAOA gene variants x Negative Parenting was also drawn. Modification indices suggested adding paths between Negative Parenting and MAOA gene variants as well as Negative Parenting between MAOA gene variants x Negative Parenting. As noted in the table below, there were no significant direct paths in the model (n = 157).

Table 20
Total Model #6

		Estimate	SE	Est./SE	*p*
Conduct Problems on					
	ADM	0.014	0.080	0.180	0.857
	MAOA	-0.019	0.081	-0.236	0.814
	NPT	0.137	0.152	0.900	0.368
	MAOA x NPT	-0.009	0.151	-0.057	0.955
Affective Decision-Making on					
	MAOA	-0.109	0.080	-1.366	0.172
	NPT	-0.085	0.154	-0.551	0.582
	MAOA x NPT	0.004	0.152	0.026	0.979
MAOA with					
	MAOA x NPT	0.048	0.081	0.596	0.551
Negative Parenting Total with					
	MAOA x NPT	0.840	0.024	34,592	0.000
	MAOA	0.123	0.081	1.527	0.127

DISCUSSION

The results from this study indicated two primary findings. Specifically, this study found two significant moderation effects: one between affective decision-making and MAOA gene variants when predicting to reactive aggression and one between affective decision-making and MAOA gene variants when predicting to conduct problems. Overall, the main aims of the study to a) to determine whether decision-making abilities act as a moderator between genetic variants of MAOA and aggressive or conduct problem behavior and b) to determine whether the interaction between self-reported ratings of negative parent behaviors and genetic variants of MAOA will act as a moderator between decision-making abilities and aggressive or conduct problem behavior, were not supported. Hypotheses 1B, 1D, 2A, 2B, 2C, 2D, 3, and 4 were not supported, as the models that corresponded to these hypotheses had no significant pathways.

Neither children's reaction time nor parenting behaviors were significant moderators. In this study, reaction time was considered a measure of children's impulsive behavior when they are completing the IGT. Other studies have also examined the relationship between MAOA gene variants and impulsive behavior on the IGT with cocaine use, but did not find significant effects (Verdejo-Garcia et al., 2013). This study attempted to further that finding by incorporating the use of a reaction time score; however, results again were not significant.

Parenting behaviors were also not significant. While other studies have found parental maltreatment of children to be a significant moderator in previous studies of MAOA gene variants, this moderation was not seen in this sample. This null finding could be because this

study was measuring parenting behaviors in a broader sense, unlike the specific construct of child maltreatment.

In general, this study's hypotheses were largely not supported. This may be because this sample is composed of children who were screened in for aggressive behavior, which may have limited the ability of the study to examine differences amongst this population. However, significant moderation effects partially supported hypotheses 1A and 1C. The study's significant moderation effects are discussed below.

Moderation between MAOA and Affective Decision-Making – Reactive Aggression

One primary finding of this study was the moderation between low and high levels of MAOA and affective decision-making when predicting to reactive aggression (Hypothesis 1A). Specifically, this moderation indicated that when children have higher affective decision-making scores, and either the high or the low MAOA variant, they are more likely to have teachers report they engage in higher levels of reactive aggression; this pattern is more distinct for children with the lower MAOA variant. When children have lower affective decision-making scores and the higher MAOA variant, they are more likely to have teachers report they engage in lower levels of reactive aggression. However, when children have the lower MAOA variant and have higher affective decision-making scores, they are more likely to have teachers report they engage in higher levels of reactive aggression. Therefore, in the context of poorer affective decision-making, MAOA may be acting as a protective factor and reducing a child's risk associated with poor decision-making.

A protective factor is any environmental variable that can promote a positive developmental outcome by modifying the risks associated with a poor developmental outcome (Herrenkohl et al., 2003; Luthar, Cicchetti, & Becker, 2000; Rutter, 1987; Rutter, 2003; Woolley

& Grogan-Kaylor, 2006). Protective factors can also be defined as variables that act as a buffer against the negative effects associated with risk factors (Rutter, 1987).

Children with lower affective decision-making abilities (i.e., they have chosen primarily from the disadvantageous decks and received more punishment as opposed to reward), who also have the higher MAOA variant are less likely to engage in reactive aggression behaviors as compared to their counterparts (i.e., children with lower affective decision-making and the lower MAOA variant). Therefore, the variant of MAOA these children have is important to consider when looking at these types of behaviors.

Why do different levels of MAOA affect these children in this way? When MAOA is released in the brain, it breaks down other various neurotransmitters by producing hydrogen peroxide (Shih 1991; Thorpe et al., 1987). MAOA typically targets neurotransmitters such as serotonin, norepinephrine, dopamine, and clorgyline (Green & Youdim, 1975; Johnston 1968; Knoll & Magyar, 1972; Fowler et al., 1982). Some of these neurotransmitters, like serotonin, dopamine, and norepinephrine have been linked to aggression, conduct problem behavior, and poor stress regulation (Caspi et al., 2002). Therefore, when a child has the gene that encodes for higher levels of MAOA to be released into the brain, their brain is essentially more efficient at breaking down these neurotransmitters because they have more MAOA and vice versa.

In this moderation, even if a child is engaging in poor decision-making capacity, in that they do not easily recognize the reward versus punishment options presented to them, the higher MAOA variant is essentially protecting them from engaging in more reactive aggression behaviors. Specifically, children with the higher MAOA variant are more efficient at breaking down various neurotransmitters that have been linked to reactive aggression. In this instance, reactive aggression is reaction-based aggressive behavior, and is often considered to be more

emotional in nature. Individuals who engage in reactive aggression typically have poor emotional and behavioral regulation (Dodge, 1991; Vitaro & Brendgen, 2011; Rathert, Fite, Gaertner, & Vitulano, 2011; White, Jarrett, & Ollendick, 2013; White & Turner, 2014; Frick & Morris, 2004; Hubbard et al., 2004; Marsee & Frick, 2007). Reactive aggression can stem from a frustration response, or a response to a perceived threat, but in both cases, reactive aggression tends to be an immediate and instinctual reaction, rather than a pre-planned behavior (Berkowitz, 1978; Dodge, 1991; Vitaro & Brendgen, 2011; Dodge & Coie, 1987; Hubbard et al., 2001; Lobbestael, Cima, & Arntz, 2013). Therefore, if an aggressive child has poor decision-making skills, as the sample in this study came from a sample of children who were screened in for aggressive behavior, they may be more likely to engage in reactive aggression as opposed to other types of aggression, such as conduct problems. However, if they have the higher MAOA variant, they are better able to break down the neurotransmitters associated with reactive aggression behavior. Even though these children made poor decisions on the IGT, they are able to make better decisions when faced with a perceived threat or frustration. Therefore, the higher MAOA variant acts as a protective factor, essentially shielding them from the risks associated with poor affective decision-making and its consequences. A somewhat different pattern exists for conduct problems.

Moderation between MAOA and Affective Decision-Making – Conduct Problems

A second finding of the study was the moderation between low and high levels of MAOA and affective decision-making when predicting to conduct problems (Hypothesis 1C). There were two primary patterns between the two variables that most influenced this moderation effect. First, there was an especially wide variation in the average conduct problem behaviors reported for children with the low MAOA variant compared to the high MAOA variant. Therefore, it may

be that for children with the low MAOA variant, affective decision-making can act as a protective factor and shield a child from the risks associated with the lower variant of MAOA. Second, there is a larger difference between the average conduct problem behaviors reported for children with low affective decision-making scores compared to children with higher affective decision-making scores. As such, children with low affective decision-making scores may be more impacted by their MAOA variant than those with higher affective decision-making scores. Thus, when children have poor affective decision-making, higher levels of MAOA may be acting as a protective factor and reducing a child's risk associated with poor affective decision-making.

The most important implication of this finding is that children with the low MAOA variant who also had higher affective decision-making scores were less likely to engage in conduct problem behaviors according to their teachers in comparison to other children with the low MAOA variant. Children with the lower MAOA variant have less MAOA in their brains, meaning that they are essentially less efficient at breaking down neurotransmitters such as norepinephrine, dopamine, and clorgyline (Green & Youdim, 1975; Johnston 1968; Knoll & Magyar, 1972; Fowler et al., 1982). As such, they may have excess neurotransmitters in their brains, all of which have been associated with aggressive and conduct problem behavior (Caspi et al., 2002). However, if these children have higher affective decision-making abilities, why do they not engage in these aggressive and conduct problem behaviors?

Affective decision-making is considered to be emotional decision-making, or decision-making, which is not typically based on conscious thought. Affective decision-making relies on the body's signals, and uses those signals to interpret whether a decision is good or bad. Children who have better affective decision-making skills are able to accurately interpret their body's cues and choose more advantageously on the IGT (Bechara, Damásio, Tranel, & Anderson, 1998;

Bechara, Damásio, Tranel, & Damásio, 2005). It may be that though these children have excess neurotransmitters associated with aggression and conduct problems behaviors in their brains, due to the fact that they are able to accurately interpret their body's somatic signals, they are able to engage in better affective decision-making.

A second key observation about this moderation effect, as noted above, is that there is greater variation in conduct problems among children with poor affective-decision-making than among children with better affective decision-making, permitting the MAOA variant level to serve as more of a protective factor among these children. Children with poor affective decision-making skills may essentially have difficulties interpreting their body's somatic signals and therefore have difficulties choosing between advantageous decks and disadvantageous decks (Bechara, Damásio, Tranel, & Damásio, 2005; Jameson, Hinson, & Whitney, 2004; Bechara, Damásio, & Damásio, 2000; Damásio, 1998). However, when children with poor affective decision-making skills have the higher MAOA variant, they are less likely to engage in conduct problem behaviors. This may be because when MAOA is released in the brain, it breaks down other various neurotransmitters, such as serotonin, norepinephrine, dopamine, and clorgyline, which have been linked to aggression, conduct problem behavior, and poor stress regulation (Shih 1991; Thorpe et al., 1987; Green & Youdim, 1975; Johnston 1968; Knoll & Magyar, 1972; Fowler et al., 1982; Caspi et al., 2002). As such, when children have the higher MAOA variant, they have higher levels of MAOA in their brains and are more efficient at breaking down neurotransmitters. Therefore, even if the child is having difficulties interpreting their body's somatic signals and is making poor decisions on the IGT, the higher MAOA variant is protecting them from engaging in more conduct problem behaviors. At the same time, having better

affective decision-making abilities also appears to act as a protective factor, this time shielding a child from the risks associated with the lower MAOA variant.

When children have better affective decision-making scores, the relationship between MAOA gene variants, affective decision-making and conduct problems is more limited, and though thus less consequential, does appear counterintuitive. However, the difference between conduct problem behaviors and reactive aggression behaviors may contribute to this difference. Conduct problems are defined as planned acts of disobedient behavior, whereas reactive aggression is usually aggressive behavior that stems from frustration and is not thought out (Reynolds & Kamphaus, 1992; Berkowitz, 1978; Dodge, 1991; Vitaro & Brendgen, 2011; Dodge & Coie, 1987; Hubbard et al., 2001; Lobbestael, Cima, & Arntz, 2013). Therefore, conduct problems are as acts of aggression that are designed to achieve a goal. Even though conduct problems and reactive aggression are different, both have been linked to various neurotransmitters (Caspi et al., 2002). If a child has higher affective decision-making scores and the higher MAOA variant, they are somewhat more likely to engage in conduct problem behaviors. Though this effect is small, this may be related to the population this study recruited from, as this study is composed of a population of at-risk children who were identified as displaying aggressive behavior in the classroom. Even though these children's brains are considered to be more efficient at breaking down the neurotransmitters associated with aggressive behavior, and they have higher affective decision-making, they are still choosing to engage in conduct problem behaviors. Though they may understand the reward versus punishment system presented by the IGT, in the real world, they may choose to engage in conduct problem behaviors as a way to achieve their goals.

Limitations

Though some of the relationships this study initially hypothesized were partially supported by the data, in large part the hypotheses were not supported. While this study did employ a medium sample size, as indicated by Kline (2005), there may have not been enough power to detect smaller effect sizes given that the sample (n = 142) only meets minimum requirements according to other sources (Ding, Velicer, & Harlow, 1995). The study may not have had enough power to accurately test for the interaction terms.

Implications for Clinical Work and Future Directions

Although the current study provides an opportunity to examine how individual differences within at-risk sample can affect outcomes and has implications for targeted clinical work with at-risk children, future studies should aim to recruit more children from both a community sample and more at-risk children. Clinicians may benefit from knowing whether the aggressive child they have as therapy client has the low MAOA variant or the high MAOA variant. If clinicians knew that the child they were seeing for aggressive behavior did have the lower MAOA variant, then that child might need more advanced therapeutic interventions aimed at reducing aggressive or conduct problem behavior. The same could be said for children who are exhibiting poor affective decision-making skills and who have the low MAOA variant.

Future studies may also find that it is beneficial to examine the variables in more detail. Such measures could include parent reports of aggression or conduct problem behavior, teacher and parent reports of children's decision-making abilities, and observational data about parenting behaviors being exhibited in the home. For example, future studies could consider both the teacher's report of conduct problem behavior and the parent's report of conduct problem behavior through use of the BASC teacher and parent rating scales.

Summary

This study had two primary aims. One, to test if decision-making is able to moderate the relationship between low versus high MAOA and aggressive or conduct problem behaviors. Two, to determine if low versus high MAOA and negative parenting behaviors will moderate the relationship between decision-making and aggressive and conduct problem behaviors.

This study made use of archival data from a Group versus Individual Coping Power study. Baseline data was collected prior to the children's 4th grade year. Baseline measures included a buccal swab for genetic data, the Iowa Gambling Task, the Teacher Report of Proactive and Reactive Aggression, the Behavior Assessment System for Children, and the Alabama Parenting Questionnaire.

The study used path analysis to test the data. Base models were created to test if MAOA gene variants, decision-making (affective and reaction time), and negative parenting impacts reactive aggression or conduct problem behaviors. In addition to these main effects, base models also tested whether the moderation between MAOA gene variants and decision-making and the moderation between negative parenting and decision-making impacts reactive aggression or conduct problem behaviors. Moderation models were created to test if MAOA gene variants and negative parenting behaviors impact decision-making. Exploratory models were also created.

While neither aim was supported by the study's finding, the study did find two moderation effects to be significant. This study found that children who had lower affective decision-making scores and the lower MAOA variant displayed higher levels of reactive aggression. Children who had lower affective decision-making scores and the higher MAOA variant displayed lower levels of reactive aggression. This study also found that children with lower levels of affective decision-making scores and the lower MAOA variant displayed higher

levels of conduct problems. Children who had lower affective decision-making scores and the higher MAOA variant displayed lower levels of conduct problems. Children with higher affective decision-making scores and the low MAOA variant displayed lower levels of conduct problems. Finally, children with higher affective decision-making scores and the higher MAOA variant displayed higher levels of conduct problems.

The moderation effect between affective decision-making and MAOA gene variants predicting to reactive aggression and the moderation effect between low affective decision-making and MAOA gene variants predicting to conduct problems also shows that the higher MAOA allele, who therefore have higher levels of MAOA in their brains, can act as a protective factor. In both of these moderations, having higher levels of MAOA in the brain means that a child's brain is more efficient at degrading various neurotransmitters associated with aggression and conduct problem behavior,. Therefore, these children are engaging in less aggressive or conduct problem behavior. However, a different relationship exists between high affective decision-making and MAOA gene variants when predicting to conduct problems. In this moderation, higher affective decision-making can act as a protective factor against the risks associated with having the lower MAOA variant. However, children with the higher MAOA variant engage in more conduct problem behavior. Given that this study recruited from a sample of aggressive and conduct problem youth, it may be that these children are more effective at using conduct problem behaviors to achieve their goals, even though they have the affective decision-making abilities to understand the differences between reward and punishment.

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APPENDICES

Appendix A

Table 21
Characteristics of Studies Examining MAOA

Reference	Sample Size	Age of Sample	Gender of Sample	Race of Sample	Community or Recruited
Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993	N=6	Single family	Male and Female	Dutch (100%)	"Volunteered"
Brunner, Nelen, van Zandvoort, Abeling, van Gennip, Wolters, & ... Van Oost, 1993	N=5	Single family	Male	Dutch (100%)	"Volunteered"
Byrd & Manuck, 2014	N=27 studies, N>18400	Meta-analysis	Male and Female	Meta-analysis	Meta-analysis
Caspi, McClay, Moffitt, Mill, Martin, Craig, & ... Poulton, 2002	N=1037	Assessed at 3, 5, 7, 9, 11, 13, 15, 18, and 21	Male (52%) and Female	Kiwi (i.e., New Zealand; 100%)	Community
Choe, Shaw, Hyde, & Forbes, 2014	N=189	Age 1.5, 2, and 5 for parental behaviors; Age 17 for DNA	Male	Caucasian (56%), African American (44%)	Community
Falk, 2014	N=221	Age 6 to 9	Male (71%) and Female	56% Caucasian, 8% AA, 8% Hispanic/Latino, 28% Mixed/Other	Recruited
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	N=975	Ages 5 and 7	Males	Caucasian (100%)	Recruited
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	N=4 studies	Meta-analysis	Male and Female	Meta-analysis	Meta-analysis
Kinnally, Huang, Haverly, Burke, Galfalvy, Brent, & ... Mann, 2009	N=159	Age 19 to 64 (M=38.6)	Female	Caucasian (61%), African American (22%), Other (11%), Asians (6%)	Recruited
Pickles, Hill, Breen, Quinn, Abbott, Jones, & Sharp, 2013	N=193	Assessed at 29 weeks and 14 months	Male and Female	Caucasian (96.1%), Other (3.9%)	Recruited
Verdejo-García, Albein-Urios, Molina, Ching-López, Martínez-González, & Gutiérrez, 2013	N=124	Age 18 to 50	Male	Caucasian (from Granada, Spain)	Recruited
Whelan, Kretschmer, & Barker, 2014	N=4893	Assessed at 2-4, and 8	Male (N=2506) and Female (N=2387)	Caucasian (100%)	Recruited

Appendix B

Table 22

Analyses and Results of Studies Examining MAOA & Aggression

Reference	Analysis	Analysis Information	Aggression	Type of Aggression
Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993	Skin fibroblast analysis for MAOA levels, coding DNA, and linkage analysis	3 clinically affected males had negligible amounts of MAO. Coded mRNA sequence. C to T mutation. Lod score = 3.55.	Tendency toward aggressive outbursts, typically in response to anger, fear, or frustration	Reactive
Brunner, Nelen, van Zandvoort, Abeling, van Gennip, Wolters, & ... Van Oost, 1993	Linkage analysis and analysis of blood and urine samples	Association between MAO and specific behavior	Tendency toward 1-3 day bursts of aggressive or violent behavior	Reactive
Byrd & Manuck, 2014	Liptak-Stouffer Z score procedure	MAOA genotype moderated an association of early life adversities with later aggressive and antisocial outcomes across all males (p=.0044); Low MAOA increased risk for antisocial behaviors among individuals exposed to maltreatment (p=.00000082)	Moderated aggressive outcomes	Both reactive and proactive
Falk, 2014	Poisson regression and Generalized Estimating Equations (GEE)	MAOA associated with positive reinforcement (B= -1.20, SE=0.48, p=0.01); MAOA-L associated with increased aggressive behavior (B=0.17, SE=0.08, p=0.03); Corporal punishment X MAOA related to growth in aggressive behavior (B= -0.14, SE=0.06, p=0.02); Involvement X MAOA related to growth in ODD symptoms (B= -0.10, SE=0.92, p=0.01); Corporal punishment X MAOA related to growth in aggressive behavior in males (B= -0.14, SE=0.06, p=0.02); Involvement X MAOA related to growth in aggressive behavior (B=0.06, SE=0.03, p=0.04); Negative parenting X MAOA related to growth in rule breaking behavior (B= -0.06, SE=0.03, p=0.03); Positive reinforcement X MAOA related to growth in rule breaking behavior (B= -0.21, SE=0.10, p=0.04)	Controlling for ADHD and initial levels of conduct problems, MAOA-L significantly predicted growth in CBCL aggressive behavior and corporal punishment X MAOA predicted growth in CBCL aggressive behavior in males	Both reactive and proactive
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	Regression	MAOA X physical abuse exposure (b= -0.84, SE=0.40, t=2.09, P=0.037); Weaker interaction in high MAOA males (b=0.61, SE=0.22, t=2.83, P=0.005) compared to low MAOA males (b=1.45, SE=0.33, t=4.40, P<0.001)	High MAOA carriers more likely to have more mental health problems compared to low MAOA carriers (P=0.017)	Both reactive and proactive
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	Measure of heterogeneity through I^2	No evidence of significant heterogeneity in the high-activity MAOA group across studies (χ^2=0.79, P=0.940, I^2=0.0%); Mild to moderate heterogeneity across studies in the low-activity MAOA group (χ^2=6.29, P=0.179, I^2=36.4%)	Mental health problems include ADHD, conduct disorder, ODD, antisocial personality disorder, etc.	Both reactive and proactive
Kinnally, Huang, Haverly, Burke, Galfalvy, Brent, & ... Mann, 2009	ANOVA	Perceived parental care, early stressor, and MAOA interaction predicted impulsivity/aggression scores [t(2,150)=3.17, P=0.048]; Low MAOA with early stressor exhibited lower impulsivity/aggression when higher parental care reported [t(1,12)=2.64, P=0.021]; Similar pattern in low-high groups [t(1,50)=3.86, P=0.0001]	Significant interaction between perceived parental care, early stressor, and MAOA predicted aggression scores in low and low-high groups, but not high groups	Reactive
Pickles, Hill, Breen, Quinn, Abbott, Jones, & Sharp, 2013	Regression and slope analysis	MAOA status X maternal sensitivity on infants distress (weighted standardized coefficient=0.52, F(1, 143)=9.19, p=.003)	Decreasing maternal sensitivity was associated with increasing infant anger proneness only in infants with the low expression MAOA variant	Reactive

Appendix C

Table 23
Analyses and Results of Studies Examining MAOA & Conduct Problems

Reference	Analysis	Analysis Information	Conduct Problems
Byrd & Manuck, 2014	Liptak-Stouffer Z score procedure	MAOA genotype moderated an association of early life adversities with later aggressive and antisocial outcomes across all males (p=.0044); Low MAOA increased risk for antisocial behaviors among individuals exposed to maltreatment (p=.00000082)	Heightened risk for antisocial behavior
Caspi, McClay, Moffitt, Mill, Martin, Craig, & ... Poulton, 2002	Moderated regression analysis	Main effect of maltreatment (b=0.35, SE=0.07, t=4.82, P<0.001); MAOA X maltreatment (b=-0.36, SE=0.14, t=2.53, P=0.01); Interaction showed that the effect of childhood maltreatment was weaker in groups of males with high MAOA (b=0.24, SE=0.11, t=2.15, P=0.03) compared to groups of males with low MAOA (b=0.68, SE=0.12, t=5.54, P<0.001)	G X E significant for conduct disorder P=0.06, violent crime convictions **P**=0.05, self-rated disposition toward violence **P**=0.10, and observer rated antisocial personality disorder **P**=0.04
Choe, Shaw, Hyde, & Forbes, 2014	Multiple group modeling	Significant maternal punitiveness at age 1.5 on AB at age 20 for MAOA-L AA (R^2=.32) and Caucasian (R^2=.20); Significant maternal punitiveness at age 2 on AB at age 15 for MAOA-L AA (R^2=.16) and Caucasian (R^2=.07); Significant alternative caregivers' punitive discipline at age 5 on violent attitudes at age 17 for MAOA-L AA (R^2=.23) and Caucasian (R^2=.25); Significant alternative caregivers' punitive discipline on AB at age 20 for MAOA-L AA (R^2=.30) and Caucasian (R^2=.33); MAOA X mothers' punitive discipline at age 5 that predicted AA men's juvenile petitions (χ^2(2, N=76)=1.55, p=.462, CFI=1.0, RMSEA=.00, SRMR=.08); Mother's punitive discipline predicted more juvenile court petitions among MAOA-L AA men (β=0.41, p=.008) but not among MAOA-H AA men (β= -0.02, p=.913)	Increased juvenile petitions among MAOA-L AA men with punitive mothers at age 5
Falk, 2014	Poisson regression and Generalized Estimating Equations (GEE)	MAOA associated with positive reinforcement (B= -1.20, SE=0.48, p=0.01); MAOA-L associated with increased aggressive behavior (B=0.17, SE=0.08, p=0.03); Corporal punishment X MAOA related to growth in aggressive behavior (B= -0.14, SE=0.06, p=0.02); Involvement X MAOA related to growth in ODD symptoms (B= -0.10, SE=0.92, p=0.01); Corporal punishment X MAOA related to growth in aggressive behavior in males (B= -0.14, SE=0.06, p=0.02); Involvement X MAOA related to growth in aggressive behavior (B=0.06, SE=0.03, p=0.04); Negative parenting X MAOA related to growth in rule breaking behavior (B= -0.06, SE=0.03, p=0.03); Positive reinforcement X MAOA related to growth in rule breaking behavior (B= -0.21, SE=0.10, p=0.04)	Controlling for ADHD and initial levels of conduct problems, interaction between corporal punishment X MAOA predicted growth in CBCL aggressive behavior in males
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	Regression	MAOA X physical abuse exposure (b= -0.84, SE=0.40, t=2.09, P=0.037); Weaker interaction in high MAOA males (b=0.61, SE=0.22, t=2.83, P=0.005) compared to low MAOA males (b=1.45, SE=0.33, t=4.40, P<0.001)	High MAOA carriers more likely to have more mental health problems compared to low MAOA carriers (P=0.017)
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	Measure of heterogeneity through I^2	No evidence of significant heterogeneity in the high-activity MAOA group across studies (χ^2=0.79, P=0.940, I^2=0.0%); Mild to moderate heterogeneity across studies in the low-activity MAOA group (χ^2=6.29, P=0.179, I^2=36.4%)	Mental health problems include ADHD, conduct disorder, ODD, antisocial personality disorder, etc.
Kinnally, Huang, Haverly, Burke, Galfalvy, Brent, & ... Mann, 2009	ANOVA	Perceived parental care, early stressor, and MAOA interaction predicted impulsivity/aggression scores [t(2,150)=3.17, P=0.048]; Low MAOA with early stressor exhibited lower impulsivity/aggression when higher parental care reported [t(1,12)=2.64, P=0.021]; Similar pattern in low-high groups [t(1,50)=3.86, P=0.0001]	Significant interaction between perceived parental care, early stressor, and MAOA predicted aggression scores in low and low-high groups, but not high groups

Appendix D

Table 24
Analyses and Results of Studies Examining MAOA & Decision-Making

Reference	Analysis	Analysis Information	Decision-Making
Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993	Skin fibroblast analysis for MAOA levels, coding DNA, and linkage analysis	3 clinically affected males had negligible amounts of MAO. Coded mRNA sequence. C to T mutation. Lod score = 3.55.	Other types of impulsive behavior included arson, attempted rape, and exhibitionism
Brunner, Nelen, van Zandvoort, Abeling, van Gennip, Wolters, & ... Van Oost, 1993	Linkage analysis and analysis of blood and urine samples	Association between MAO and specific behavior	Impulsive behavior and borderline mental retardation (IQ typically around 85)
Choe, Shaw, Hyde, & Forbes, 2014	Multiple group modeling	Significant maternal punitiveness at age 1.5 on AB at age 20 for MAOA-L AA (R^2=.32) and Caucasian (R^2=.20); Significant maternal punitiveness at age 2 on AB at age 15 for MAOA-L AA (R^2=.16) and Caucasian (R^2=.07); Significant alternative caregivers' punitive discipline at age 5 on violent attitudes at age 17 for MAOA-L AA (R^2=.23) and Caucasian (R^2=.25); Significant alternative caregivers' punitive discipline on AB at age 20 for MAOA-L AA (R^2=.30) and Caucasian (R^2=.33); MAOA X mothers' punitive discipline at age 5 that predicted AA men's juvenile petitions (χ^2(2, N=76)=1.55, p=.462, CFI=1.0, RMSEA=.00, SRMR=.08); Mother's punitive discipline predicted more juvenile court petitions among MAOA-L AA men (β=0.41, p=.008) but not among MAOA-H AA men (β= -0.02, p=.913)	Increased juvenile petitions among MAOA-L AA men with punitive mothers at age 5
Kinnally, Huang, Haverly, Burke, Galfalvy, Brent, & ... Mann, 2009	ANOVA	Perceived parental care, early stressor, and MAOA interaction predicted impulsivity/aggression scores [t(2,150)=3.17, P=0.048]; Low MAOA with early stressor exhibited lower impulsivity/aggression when higher parental care reported [t(1,12)=2.64, P=0.021]; Similar pattern in low-high groups [t(1,50)=3.86, P=0.0001]	Significant interaction between perceived parental care, early stressor, and MAOA predicted impulsivity scores in low and low-high groups, but not high groups
Verdejo-García, Albein-Urios, Molina, Ching-López, Martínez-González, & Gutiérrez, 2013	Hierarchical multiple regression analysis	Predicting to positive urgency MAOA genotype and severity of cocaine use (ΔF=4.42, p<0.05) and MAOA X cocaine severity (ΔF=7.60, p<0.01); Predicting to sensation seeking age (ΔF=7.86, p<0.01) and MAOA X cocaine severity (ΔF=10.22, p<0.01)	Decision-making subscale was not significant (p=0.051); Low activity carriers with greater cocaine severity had higher impulsivity

Appendix E

Table 25
Analyses and Results of Studies Examining MAOA & Parenting Behaviors

Reference	Analysis	Analysis Information	Parenting
Byrd & Manuck, 2014	Liptak-Stouffer Z score procedure	MAOA genotype moderated an association of early life adversities with later aggressive and antisocial outcomes across all males (p=.0044); Low MAOA increased risk for antisocial behaviors among individuals exposed to maltreatment (p=.00000082)	Individuals with low MAOA exposed to maltreatment by parents have a higher risk for antisocial behaviors
Caspi, McClay, Moffitt, Mill, Martin, Craig, & ... Poulton, 2002	Moderated regression analysis	Main effect of maltreatment (b=0.35, SE=0.07, t=4.82, P<0.001); MAOA X maltreatment (b=-0.36, SE=0.14, t=2.53, P=0.01); Interaction showed that the effect of childhood maltreatment was weaker in groups of males with high MAOA (b=0.24, SE=0.11, t=2.15, P=0.03) compared to groups of males with low MAOA (b=0.68, SE=0.12, t=5.54, P<0.001)	Effect of childhood maltreatment weaker in males with high MAOA, compared to males with low MAOA
Choe, Shaw, Hyde, & Forbes, 2014	Multiple group modeling	Significant maternal punitiveness at age 1.5 on AB at age 20 for MAOA-L AA (R^2=.32) and Caucasian (R^2=.20); Significant maternal punitiveness at age 2 on AB at age 15 for MAOA-L AA (R^2=.16) and Caucasian (R^2=.07); Significant alternative caregivers' punitive discipline at age 5 on violent attitudes at age 17 for MAOA-L AA (R^2=.23) and Caucasian (R^2=.25); Significant alternative caregivers' punitive discipline on AB at age 20 for MAOA-L AA (R^2=.30) and Caucasian (R^2=.33); MAOA X mothers' punitive discipline at age 5 that predicted AA men's juvenile petitions (χ^2(2, N=76)=1.55, p=.462, CFI=1.0, RMSEA=.00, SRMR=.08); Mother's punitive discipline predicted more juvenile court petitions among MAOA-L AA men (β=0.41, p=.008) but not among MAOA-H AA men (β= -0.02, p=.913)	Overall, maternal punitiveness at ages 1.5 and 2 predicted more AB at ages 20 and 15, respectively, among all MAOA-L men
Falk, 2014	Poisson regression and Generalized Estimating Equations (GEE)	MAOA associated with positive reinforcement (B= -1.20, SE=0.48, p=0.01); MAOA-L associated with increased aggressive behavior (B=0.17, SE=0.08, p=0.03); Corporal punishment X MAOA related to growth in aggressive behavior (B= -0.14, SE=0.06, p=0.02); Involvement X MAOA related to growth in ODD symptoms (B= -0.10, SE=0.92, p=0.01); Corporal punishment X MAOA related to growth in aggressive behavior in males (B= -0.14, SE=0.06, p=0.02); Involvement X MAOA related to growth in aggressive behavior (B=0.06, SE=0.03, p=0.04); Negative parenting X MAOA related to growth in rule breaking behavior (B= -0.06, SE=0.03, p=0.03); Positive reinforcement X MAOA related to growth in rule breaking behavior (B= -0.21, SE=0.10, p=0.04)	Use of punishment significantly predicted growth in aggressive behavior in children with MAOA-H; use of negative parenting significantly predicted growth in rule breaking behavior in children with MAOA-H
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	Regression	MAOA X physical abuse exposure (b= -0.84, SE=0.40, t=2.09, P=0.037); Weaker interaction in high MAOA males (b=0.61, SE=0.22, t=2.83, P=0.005) compared to low MAOA males (b=1.45, SE=0.33, t=4.40, P<0.001)	Physical abuse exposure
Kim-Cohen, Caspi, Taylor, Williams, Newcombe, Craig, & Moffitt, 2006	Measure of heterogeneity through I^2	No evidence of significant heterogeneity in the high-activity MAOA group across studies (χ^2=0.79, P=0.940, I^2=0.0%); Mild to moderate heterogeneity across studies in the low-activity MAOA group (χ^2=6.29, P=0.179, I^2=36.4%)	Familial adversity can be moderated by MAOA activity
Pickles, Hill, Breen, Quinn, Abbott, Jones, & Sharp, 2013	Regression and slope analysis	MAOA status X maternal sensitivity on infants distress (weighted standardized coefficient=0.52, F(1, 143)=9.19, p=.003)	Decreasing maternal sensitivity was associated with increasing anger proneness only in infants with the low expression MAOA variant

Table 25
Analyses and Results of Studies Examining MAOA & Parenting Behaviors

Reference	Analysis	Analysis Information	Parenting
Whelan, Kretschmer, & Barker, 2014	Linear regression, moderated indirect effect model, simple slope analysis	Harsh parenting X victimization stronger for low MAOA males (β=.14, p<.001)	Stronger association between harsh parenting and victimization in low MAOA males

Appendix F

Table 26

Sample Characteristics with Full Sample (N = 157)		Percent	Frequency
Sex	Males	70.1%	110
	Females	29.9%	47
MAOA	Low Activity	33.1%	52
	High Activity	66.9%	105
Zygote Classification	Homozygote	29.9%	47
	Hemizygote	70.1%	110

Appendix G

Table 27
Correlation Table

	Gender	MAOA	Zygote	CP	Inv	Pos Par	Mon	Inc Dis	Cor Pun	PPT	NPT	Proactive	Reactive	ADM	RTS	MAOA x ADM	MAOA x RT	MAOA x NPT	NPT x ADM	NPT x RTS	ADM – High and Low
Gender	1																				
MAOA	.017	1																			
Zygote	-1**	-.017	1																		
CP	-.214**	-.005	.214**	1																	
Involvement	.066	-.087	-.066	-.115	1																
Positive Parenting	.043	-.101	-.043	-.073	.692**	1															
Monitoring	-.024	.128	.024	.168*	-.241**	-.205*	1														
Inconsistent Discipline	.023	.055	-.023	.047	-.192*	-.072	.320**	1													
Corporal Punishment	.074	.053	-.074	.033	-.050	.026	-.011	.162	1												
Positive Parenting Total	.060	-.102	-.060	-.102	.920**	.920**	-.243**	-.144	-.013	1											
Negative Parenting Total	.037	.119	-.037	.125	-.243**	-.127	.659**	.747**	.580**	-.201*	1										
Proactive	-.256**	-.055	.256**	.267**	.026	.032	.089	.032	-.053	.031	.034	1									
Reactive	-.143	-0.74	.143	.354**	.035	-.014	.089	.061	-.121	.011	.015	.606**	1								
ADM	.208**	-.119	-.208**	.004	.119	.095	-.034	-.060	-.093	.116	-.094	.000	.119	1							
RTS	.180*	.058	-.180*	.038	.120	.098	.095	.027	.064	.118	.094	-.110	-.125	.065	1						
MAOA x ADM	.071	-.049	-.071	.096	.132	.118	.007	-.066	-.096	.136	-.078	.016	.196*	.814**	.077	1					
MAOA x RT	.222**	.024	-.222**	.060	.084	.093	.044	.094	.117	.096	.129	-.132	-.074	.079	.788**	.100	1				
MAOA x NPT	-.008	.048	.008	.105	-.246**	-.180*	.598**	.629**	.450**	-.232**	.845**	.029	.048	-.074	.118	-.087	.150	1			
NPT x ADM	-.037	-.006	.037	.059	-.050	.009	-.104	-.048	.036	-.022	-.059	.023	-.011	-.088	-.037	.069	-.070	-.190*	1		
NPT x RTS	.115	.088	-.115	.043	.038	-.032	.026	-.033	.070	.003	.032	-.010	-.035	-.035	-.068	-.058	.095	.035	-.018	1	
ADM – High and Low	.010	-.113	-.010	.089	.105	.078	-.092	-.068	-.158	.100	-.160	-.003	.171*	.643**	-.063	.509**	-.066	-.124	-.067	-.064	1

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).