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Antecedents of Borderline Personality Disorder and Antisocial Personality Disorder: An Examination of Gene X Environment Interactions

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ANTECEDENTS OF BORDERLINE PERSONALITY DISORDER AND ANTISOCIAL PERSONALITY DISORDER: AN EXAMINATION OF GENE X ENVIRONMENT INTERACTIONS

by

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ABSTRACT

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Amy Medina

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Current thinking suggests that genotypes associated with impulse-control disorders and negative emotionality, such as monoamine oxidase-a (MAOA), interact with negative early environmental factors like childhood maltreatment and develop into the disorders know as Borderline Personality Disorder (BPD) and Antisocial Personality Disorder (ASPD). Using existing data from a prospective cohort design study of the consequences of child abuse and neglect, participants (N = 896 represent individuals with documented histories of child abuse and neglect and a matched comparison group that were followed up into adulthood and interviewed. A subsample of 631 participants gave permission for DNA extraction and analyses during a follow-up medical status exam. The final sample used in this study consisted of 592 participants, as we restricted analyses to White, non-Hispanic and Black, non-Hispanic individuals. Official reports of child maltreatment were collected from court records during the years 1967 to 1971, while retrospective self-reports were collected at both the 1st and 2nd interviews. BPD, ASPD, BPD symptoms, and ASPD symptoms were measured with a structured interview of DSM-III-R BPD criteria. MAOA genotype was coded into two unique variables; DC1, which represented males with one 3-repeat and females with two 3-repeats (low MAOA activity) compared to all other genotypes and DC2 represented the heterozygous females with the 3,4-genotype compared to all other genotypes. Multiple logistic and ordinary least squares (OLS) were conducted to

analyze the main effect of each independent variable and any interactions in the prediction of one of the six dichotomous dependent variables. All analyses controlled for age, sex, and race. We hypothesized that childhood maltreatment will predict increased risk for BPD, BPD symptoms, ASPD, ASPD symptoms, impulsivity, and suicide attempts. However, similar to Widom, Czaja, & Paris (2009), we expected that males but not females, would show an association between childhood abuse and neglect and BPD. We also did not expect that there will be any differences in these relationships by race (e.g., Whites & Blacks). However, in regard to MAOA genotype, based on a previous publication using this data (Widom & Brzustowicz, 2006), we hypothesized that MAOA genotype would moderate the relationship between childhood maltreatment and our six dependent variables. The results of the present study showed that childhood maltreatment predicted ASPD diagnosis, ASPD symptoms, a lifetime history of suicide attempts, and impulsivity. Unexpectedly, we did not observe a relationship between BPD diagnosis and childhood maltreatment, although there was a relationship between childhood abuse and neglect and number of BPD symptoms. Several differences by race and sex, which indicate that there may be other environmental and contextual factors that may be influential in the development of these disorders in disadvantaged groups. Furthermore, we only observed one significant 3-way interaction suggesting that the heterozygous MAOA genotype (3-,4-) was protective for Black females with a history of childhood maltreatment. Due to the limited nature of MAOA genotype studies in Black females, it is difficult to put these results into context and future research is needed to better understand the impact of MAOA genotype in this population. Overall, our results the significant relationship between childhood maltreatment and personality psychopathology in adulthood.

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DEDICATION

I would like to dedicate this dissertation to my clients and patients at Downstate Medical Center, Queens TASC, Kingsboro Psychiatric Center, the Youth Development Clinic, Jersey City Community Solutions, Rutgers University Behavioral Healthcare, Edna Mahan Correctional Facility for Women (EMFCW), the Adult Diagnostic and Treatment Center (ADTC), and Northern State Prison (NSP). Thank you for everything. I will always carry a piece of you in my heart.

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CHAPTER ONE

Introduction

This dissertation focuses on the role of gene-by-environment interactions in the development of Borderline Personality Disorder (BPD) and Antisocial Personality Disorder (ASPD) with a specific focus on monoamine genotypes (e.g., monoamine oxidase A or MAOA) and their influence on a person's risk of developing BPD and ASPD. The dissertation begins with an overview of the features of BPD and ASPD, the modified course experienced by these individuals, and prevalence of the disorder. I will then provide a brief overview of a leading theory of the two disorders (e.g., a Biosocial Model) and the state of the empirical literature examining genetic and environmental factors associated with an increased risk of developing BPD and ASPD. After describing the existing gaps in the BPD and ASPD literature, I provide a description of the hypotheses, methods, and limitations of the study design. Finally, I will provide an overview or the results and subsequently discuss these findings in the context of the existing literature, ending with future directions for research.

Background

Approximately 9-15% of the U.S. population will meet diagnostic criteria for at least one personality disorder in their lifetime, making personality disorders one of the most prevalent forms of mental illness in America (APA, 2013; Lenzenweger, Lane, Loranger, & Kessler, 2007; Torgersen, Kringlen, & Cramer, 2001; Trull, Jahng, Tomko, Wood, & Sher, 2010). Borderline Personality Disorder (BPD) is characterized by distinct affective, behavioral, cognitive, and interpersonal deficits (APA, 2013; Linehan, 1993). The core features of BPD include a persistent pattern of interpersonal instability, poor self-image, and dysregulated affect in combination with

marked impulsivity and sometimes psychotic-like symptoms of dissociation and paranoid ideation (APA, 2013; Linehan, 1993). This pattern of personality is chronic and typically first appears in early adolescence or early adulthood (APA, 2013; Linehan, 1993). Prevalence rates of BPD range from 0.5% to 5.9% of the general population in the United States (APA, 2013; Grant et al., 2008; Lenzenweger et al., 2007), and are typically over-represented in clinical settings (Zimmerman & Mattia, 1999).

BPD has been shown to cause significant functional impairment for individuals diagnosed with the disorder (Grant et al., 2008; Lenzenweger et al., 2007; Trull et al., 2010), most notable the increased risk of early death through suicide (Black, Blum, Pfohl, & Hale, 2004; Tomko, Trull, Wood, & Sher, 2014; Yen et al., 2003). Rates of suicide in individuals with BPD are around 10% (Oldham, 2006; Yen et al., 2003), with some estimating that approximately 75% of individuals with BPD will attempt suicide in their lifetime (Black et al., 2004). Evidence suggests that individuals with BPD report significantly lower quality of life on global and specific domains including subjective well-being, contact with friends, and social support during periods of illness (Cramer et al., 2006). Overall, individuals with BPD experience functional deficits in multiple domains and rate themselves as more impaired compared to others, indicating they are subjectively suffering because of their mental health problems.

Trull et al. (2010) found that rates of any PD were significantly higher in men (10%) than women (8%), although the prevalence of BPD was slightly higher in women (3%) than men (2.4%). Other studies have shown no differences in the rates of borderline personality disorder in men and women (Grant et al., 2008; Widom, Czaja, & Paris, 2009). In a longitudinal study of abused and neglected children, Widom and colleagues (2009) reported that sex was not a predictive factor of BPD, contrary to the suggestions that BPD is much more common in women.

Using the same data as Trull et al. (2014) with a lower threshold for diagnosing a personality disorder (e.g., only one symptom needed to cause distress or impairment), Grant and colleagues (2008) did not find a significant difference in rates of BPD in men (5.6%) and women (6.2%). However, Grant et al. (2008) did find that women with BPD had significantly higher total scores on a measure of disability, indicating greater levels of functional impairment compared to men. It may be that women present in clinical settings more frequently due to the higher degree of functional impairment they experience from their borderline symptomatology or that women are more likely than their male counterparts to seek help at all, and therefore over-represent the type of individual found in treatment settings (Linehan, 1993). It could also be the case that men with BPD experience other secondary consequences of the disorder such as early death by suicide or incarceration (Comtois & Carmel, 2016; Linehan, 1993), and therefore males may be underrepresented in community-based studies because they do not consider incarcerated members of society.

The differences in prevalence rates of BPD in men and women could also be the result of the diagnostic criteria themselves with some evidence suggesting that specific symptomology is more common in one gender more than the other (Zanarini et al., 2011). Zanarini and colleagues (2011) found that adult women and girls had significantly higher rates of mood reactivity and chronic emptiness compared to men (Zanarini et al., 2011). In contrast, adult men and boys were more likely to report impulsivity and had greater versatility in the types of impulsive behavior they engaged in compared to women and girls, not including self-harm (Zanarini et al., 2011). One possible explanation for this difference in symptom presentation is that men and women with BPD present with slightly different symptom patterns, which makes them “more” or “less” likely to be diagnosed by mental health professionals due to gender stereotypes. Results from

two studies (Benson, Donnellan, & Morey, 2017; Sharp et al., 2014) found that DSM criteria for uncontrolled anger and impulsivity are more frequently rated as positive or present for men than women. However, Benson and colleagues (2017) argue that the observed gender differentiation for these two criteria is not that anger or impulsivity are more easily diagnosed in men, but they are less common in women, unlike other BPD criteria. Importantly, Benson, Donnellan, and Morey (2017) and Zanarini et al. (2011) both found evidence that chronic symptoms of emptiness were more "easily" or more frequently identified in women. Benson and colleagues (2017) proposed that feelings of emptiness might not be a characteristic observed in men with BPD, but it could be that they experience some form of boredom found in other "male-dominated" personality disorders (e.g., ASPD, psychopathy). Taken together, it remains unclear whether the gender differences observed in BPD are due to differences in sample selection or the operationalization of BPD.

Rates of ASPD in the general population have been estimated to be between 0.2 and 3.3%, with rates approaching 50-70% in severe substance abuse populations and in criminal justice settings like jail and prison (APA, 2013). ASPD is characterized as a disorder centered around the disregard of the rights of other people and typically emerges in adolescence (APA, 2013). This disregard manifests itself into individuals engaging in criminal behavior, being deceitful, showing impulsivity and aggression, limited capacity of remorse, recklessness, and irresponsible behavior (APA, 2013). In the current version of the DSM, in order to meet criteria for a diagnosis of ASPD there must also be evidence of conduct disorder before age 15 (APA, 2013). Studies of quality-of-life show ASPD is associated with higher rates of unemployment, poorer physical health, greater financial dependency, and less social support (Goldstein, Dawson, Smith, & Grant, 2012), which is similar to findings for BPD. ASPD is often viewed as a male-

specific disorder; however, there is increasing evidence that this is not the case; with studies not observing differences between males and females in rates of ASPD (Goldstein & Grant, 2009). It is also becoming increasingly clear that there is significant co-occurrence of these two disorders. Grant et al. (2008) found 21% of individuals with a lifetime diagnosis of ASPD also met criteria for BPD. In one study of adult's court-mandated to inpatient substance abuse treatment, more women met criteria for ASPD and BPD (13.5%) compared to men (7.6%; Chun et al., 2017). Tomko and colleagues (2004) found that both men and women with BPD from a community sample were significantly more likely to have ASPD compared to controls and those with other disorders. When BPD is accompanied by adult antisocial behavior, individuals show poorer outcomes (Freestone, Howard, Coid, & Ullerich, 2013; Goldstein et al., 2012). Specifically, they show greater levels of homelessness, financial issues, trouble with police, violent behavior, and dangerous substance abuse compared to individuals with just BPD or adult antisocial behavior (Freestone et al., 2013; Goldstein et al., 2012). Chun and colleagues (2017) used factor analysis to examine the overlapping characteristics of BPD and ASPD. They observed significant evidence supporting a general disorder comprised of the symptoms for both disorders. This "general factor" was characterized by impulsivity and disinhibition with some features of interpersonal dysfunction and emotional dysregulation. In sum, it appears that ASPD shows similar negative effects on an individual's functioning, that are only exacerbated when these two disorders are observed together.

Biosocial Theory of Borderline Personality Disorder (BPD)

Linehan (1993) described BPD as "a disorder of emotion dysregulation", in which a biological vulnerability or predisposition to emotion dysregulation works in conjunction with an invalidating or abusive environment. According to Linehan's (1993) Biosocial Model of

Borderline Personality Disorder, there are three defining characteristics that explain BPD's development: 1. An inherited genetic/biological emotional vulnerability – characterized as being easily activated and aroused by emotions, an intense physiological reaction when experiencing emotions, and a slow return to baseline; 2. An invalidating environment – including childhood maltreatment (e.g., sexual abuse, physical abuse, neglect, etc.); and 3. Chronic emotional dysregulation – exhibited as an inability to understand, label, and regulate one's emotional responses, and rejection of one's emotional experience that leads to increased anxiety and distress. Individuals develop BPD through the interaction between their temperamental emotional vulnerability and invalidating environment, resulting in poor emotion regulation skills and poor distress tolerance. She described this as "poorness of fit" in that the capabilities and characteristics of the child do not or cannot adapt to the demands and expectations of their environment.

Linehan (1993) argued that it was this emotional predisposition that leaves individuals with BPD vulnerable in invalidating environments, which can include family systems that do not allow a child to learn how to tolerate their emotional experience or validate private emotional experiences of the child. These environments are not only characterized by abuse or maltreatment, but also include home environments that do not allow children to express themselves or their private experiences without being punished, trivialized, or dismissed. The child learns that emotional reactions are inappropriate and only to be experienced as internal events, although at times, extreme emotional reactions are occasionally effective in getting caregivers' attention.

Crowell, Beauchaine, and Linehan (2009) updated and modified Linehan's (1993) biosocial theory in light of recent advances in psychobiological research, with the addition of

trait impulsivity as the primary predisposition in the most severe presentations of BPD. These authors expanded upon Linehan's theory with a five-point biosocial model of BPD. This model posits that the development of BPD occurs out of a biological vulnerability for poor impulse control, an environment that promotes the development of emotional lability in an emotionally vulnerable child, and the transaction between biology and environmental risk factors that encourage emotional and behavioral dysregulation. It is the coalescence of these factors during mid-to-late adolescence, which in turn impact other areas of developmental functioning such as interpersonal relationships and overall social functioning.

Biosocial Theory of Antisocial Personality Disorder (ASPD) and Borderline Personality Disorder (BPD)

Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp (2009) proposed a model for antisocial personality and borderline personality development suggesting that both disorders are the result of a temperamental vulnerability to trait impulsivity associated with dysfunctional serotonergic and dopaminergic functioning that interacts with a high-risk family environment which promotes and reinforces emotional lability. Impulsivity appears to be a relatively heritable trait (Beauchaine & Neuhaus, 2008), which is expressed to a greater degree in children who are exposed to early environmental risk factors (Crowell et al., 2009). These authors also suggested that emotional lability and dysregulated anger could co-occur with trait impulsivity as these features share a tendency to be sensitive to early exposure to risky environments (Beauchaine, Gatzke-Kopp, & Mead, 2007; Meier, Slutske, Arndt, & Cadoret, 2008; Stepp, Lazarus, & Byrd, 2016). Similar to Crowell et al. (2009), they suggest that there is an interaction between the child's trait impulsivity and the parents coercive and invalidating reaction to the child that results in the reinforcement of maladaptive behaviors. In support of this model, studies have shown that

adolescent girls expressing poor self-control (or impulsivity) and strong negative emotionality are especially vulnerable to BPD in the context of harsh parenting (Hallquist, Hipwell, & Stepp, 2015). Another important aspect of Beauchaine and colleagues' (2009) model is the association with antisocial peers that begins in adolescence. The assumption is that an emotionally dysregulated and impulsive child begins to develop oppositional behaviors, which over time can lead to poor interpersonal interactions both at home and at school. This leads to the child seeking out social connections with other dysregulated and deviant peers. Furthermore, environmental factors such as neighborhood violence and access to illicit substance only create more opportunities for deviant behaviors.

Beauchaine et al. (2009) suggest that emotionally dysregulated and impulsive boys develop characteristics associated with antisocial personality disorder, while dysregulated girls develop borderline personality disorder. Research has shown some support for this assertion. A study by Lyons-Ruth and colleagues (2007), found similar underlying genetic components related to the expression impulsive and self-damaging phenotype related BPD and ASPD traits in a sample of young adults. However, individuals with borderline traits were more likely to be women and showed higher rates of two or more types of impulsive self-damaging behavior and intense or unstable relationships. In contrast, individuals with antisocial traits were more likely to be men and showed higher rates of repeatedly engaging in illegal activities, aggression, and reckless disregard for the safety of themselves or other people. Taken together, leading theories of BPD suggest that biological, social, and psychological factors such as child temperament, parenting practices, peers, and other negative environments (e.g., child maltreatment, neighborhood violence) all play a role in the development of the disorder. Furthermore, there is some assertation that BPD develops due to socialization differences between men and women

that lead women to be more likely to develop or be diagnosed with traits associated with BPD, compared to men, which are more likely to develop or be diagnosed with traits associated with ASPD.

Childhood Maltreatment and Personality Disorders

Despite being cited as one of the strongest predictors of BPD, the evidence suggesting that most individuals who have experienced some form of child maltreatment develop BPD has been mixed. A study by Afifi and colleagues (2011) examined the prevalence of childhood maltreatment and personality disorders in participants of the second wave of the NESARC study. They examined several types of childhood maltreatment including sexual abuse, physical abuse, emotional abuse, physical neglect (what they described as classic neglect), and emotional neglect. All forms of neglect and abuse, except emotional neglect, were associated with an increased likelihood of having a Cluster B personality disorder. This result held when accounting for covariates like mood disorders, anxiety disorders, substance use disorders, parental substance abuse, age, gender, marital status, and ethnicity. Importantly, this study also shows that the majority of individuals (>60%) that report experiencing childhood maltreatment were not diagnosed with a personality disorder (Afifi et al., 2011).

The relationship between BPD and childhood sexual abuse is one that has been extensively discussed within the BPD literature. In a recent systematic review by Ferreira, Pereira, Benevides, and Melo (2018), they investigated whether a childhood history of sexual abuse (CSA) was associated with a diagnosis of BPD, the clinical presentation of BPD traits, and prognosis. Although most studies did find a relationship between CSA and BPD, the majority of studies were cross-sectional, with samples that were primarily female (from 66% to 100%), from inpatient populations with 17 studies only using hospitalized individuals in their samples. There

were two studies that did not find a relationship between CSA and BPD. A cross-sectional study of outpatients with personality disorders by Bierer et al. (2003) did not find an association between CSA and BPD. However, it is worth noting that only approximately 35% of the Bierer et al. (2003) sample was female, which is markedly lower than any other study. A prospective study, Widom, Czaja, and Paris (2009) did not find an association between CSA and BPD; but found that childhood neglect and physical abuse predicted BPD in middle adulthood. Importantly, Widom et al. (2009) used official reports of childhood maltreatment, which is in contrast with the overwhelming majority of studies of CSA and BPD that use retrospective self-report measures. It is possible that sample selection and the way in which childhood trauma is assessed have a significant impact on the strength of the association between CSA and BPD.

In regard to ASPD, Luntz and Widom (1994) was one of the first studies to show a prospective relationship between childhood maltreatment and ASPD in adulthood. This association was even present when controlling for demographic factors, socioeconomic status, as well as official records of criminal history. MacMillan et al. (2001) found that retrospective reports of physical and sexual abuse in women was associated with increased risk of antisocial behaviors. For males, only retrospective reports of childhood physical abuse showed a significant relationship with antisocial behavior (MacMillan et al., 2001). This link likely starts to show in later childhood and adolescence, with a comprehensive meta-analysis showing that both general and violent juvenile offending was associated with a history of childhood maltreatment, including physical and sexual abuse and neglect (Braga, Gonçalves, Basto-Pereira, & Maia, 2017). This association also appear to be stable over time, with a large longitudinal study showing childhood maltreatment was associated with increased risk of antisocial behavior up until age 50 (Esposti, Pereira, Humphreys, Sales, & Bowes, 2020). Despite women showing less

antisocial behaviors overall compared to males, evidence suggests that there is still a link between childhood maltreatment and antisocial behavior regardless of sex (Afifi, Fortier, Sareen, & Taillieu, 2019).

Genetic Vulnerabilities and Personality Disorders

Genetic studies provide a unique insight into the possible biological and temperamental underpinnings of psychological disorders with advances in technology provided a steady stream of innovative methods to understand the genotypes associated with particular forms of psychopathology. A major focus of the genetic literature on BPD and similar impulse-control disorders, such as antisocial personality disorder (ASPD) and conduct disorder, has been the monoamine neurotransmitters. Monoamines share similar cellular characteristics and have influence over widespread areas of the brain, making them important components in the initiation and disinhibition of many processes and behaviors (Carlson & Birkett, 2016). MAO, specifically monoamine oxidase A (MAOA) is responsible for the destruction of excess monoamine neurotransmitters including dopamine, norepinephrine, and serotonin, after it is released into the synapse. MAO renders a neurotransmitter into an inactive substance that is no longer capable of stimulating postsynaptic receptors.

According to Dick (2011), there are two general models of how genes influence the development of psychopathology and psychological traits. As discussed earlier, the Diathesis-Stress model is one such model, which suggests that a disease or disorder manifests when a person has genetic vulnerabilities that co-occur with a negative or unhealthy environment. However, according to the Diathesis-Stress model, individuals with these vulnerable genes in a positive environment are no more likely than people without these genetic predispositions to develop that disease or disorder. In contrast, the second model refers to a Gene X Environment

(GxE) interaction, which suggests that a genetic predisposition for a disease or disorder can be adaptive or even protective against developing a disorder in a positive or healthy environment, but a negative or unhealthy environment places an individual at increased risk of developing a disease or disorder. Overall, the general distinction between the two models is that Gene X Environment interactions are based on the premise that genes have plasticity and are not necessarily vulnerabilities depending upon the environment.

A study by Caspi and colleagues (2002) was one of the first to show how a genotype could moderate the impact of childhood maltreatment on behavioral disturbances in adulthood. They examined MAOA activity in males with histories of maltreatment in relation to antisocial behaviors in adulthood and found that males with low MAOA activity showed a stronger association between childhood maltreatment and antisocial behavior. Moreover, low MAOA activity in maltreated males (for both probable and severe cases) was associated with increased risk of developing conduct disorder in adolescence, greater chance of a conviction for violent crimes in adulthood, higher self-reported dispositions towards violence, and higher informant rated antisocial personality disorder symptoms than non-maltreated males with low MAOA activity.

Kim-Cohen et al. (2006) did a meta-analysis of their own data with 4 additional studies examining MAOA activity in boys with histories of childhood maltreatment. They found evidence of a Gene X Environment interaction suggesting that the relationship between early familial adversity and the development of mental health problems was significantly stronger in the low MAOA activity group compared to the high-activity MAOA group. Widom and Brzustowicz (2006) examined the MAOA genotype and a composite measure of violence and antisocial behavior (VASB) using data from a prospective cohort design of abused and neglected

children and control participants from the mid-western United States. Results showed that in White participants, MAOA activity moderated the association between childhood maltreatment and VASB scores with the effect of child abuse and neglect on juvenile and lifetime antisocial and violent behavior significant in participants with the low MAOA activity genotype. In contrast, high MAOA activity actually appeared to be a protective factor in that for both juvenile and lifetime VASB scores, with abused and neglected children with high MAOA activity had lower VASB scores than controls. Importantly, this was not the case for Black and Hispanic participants. This suggests that there may be other factors, genetic or environmental, that are associated with antisocial and violent behaviors in people of color and that MAOA may not be one of those factors for these individuals. However, in contrast, a previous study that included Black and White males did not find that MAOA genotype significantly moderated the relationship between childhood maltreatment and an index of antisocial behavior (Haberstick et al., 2014). They suggest that perhaps previous replications of the Caspi et al. (2002) study were "false positives" and that observing this type of geneXenvironment interaction can be problematic. However, it is important to note that unlike other replication studies of Caspi et al. (2002), such as Widom and Brzustowicz (2006), Haberstick et al. (2014) used retrospective self-reports of childhood abuse and neglect. Evidence suggests that retrospective reports of childhood maltreatment tend to be underestimations, as a sizable portion of individuals do not report being abused or neglected despite adequate documentation of these experiences (Hardt & Rutter, 2004). Despite some evidence to the contrary, there appears to be a relatively strong evidence base that MAOA activity is related to violent and antisocial behaviors in at least some individuals that were abused and neglected as children.

Given, the suggested overlap between the genetic predispositions for ASPD and BPD, and that both disorders supposedly represent gendered outcomes that lead to a divergence between men and women on similar developmentally dysfunctional tracks, it is possible that there is also a relationship between MAOA activity and BPD traits. However, in one of the only studies of MAOA genotypes associated with BPD, Ni et al. (2007) examined a large sample of mostly female patients with BPD and a healthy control group of men and women recruited from the community. They examined two haplotypes of MAOA, variable number tandem repeat (VNTR) and the rs6323 polymorphisms. VNTR was classified into high and low activity alleles. There were no differences by gender in the genotype frequencies of the two MAOA polymorphisms and they did not find any association between rs6323 and BPD. However, they did observe a trend between MAOA haplotype and BPD with the low activity allele less common in individuals with BPD compared to controls and the high activity allele more common in BPD participants compared to controls. These results are surprising given the studies showing a relationship between low MAOA activity and traits of ASPD (Caspi et al., 2002; Kim-Cohen et al., 2006; Widom and Brzustowicz, 2006). Moreover, their results are in contrast with the model proposed by Beauchaine et al. (2009) suggesting that ASPD and BPD share common inherited biological risks for impulsivity. However, there are several characteristics of this study that could provide a possible explanation for their results. As mentioned earlier, the BPD group was overwhelming female (>80%), in contrast to the control group where about half the participants were female (52%). The BPD group was a clinical sample that also participated in a clinical treatment research project and there were no exclusionary criteria regarding participants taking psychotropic medications or having co-morbid depression, personality disorders, or anxiety disorders. Furthermore, there was no separation between participants that had histories of

childhood maltreatment and those that did not. Therefore, it is unclear if Ni et al. (2007) findings of a "trend" between a high activity allele of MAOA and BPD reflects an actual relationship or are the result of sample characteristics and study design.

Current Limitations of the BPD & ASPD Literature

Systematic reviews of the literature on child maltreatment and BPD have noted that the overwhelming majority of studies are cross-sectional and use retrospective reports of childhood experiences (Ferreira et al., 2018). Heavy reliance on retrospective reports leads to questions of the accuracy of these reports, particularly given the fallibility of memory. Stepp and colleagues (2016) have pointed out that the literature on the relationship between child maltreatment and BPD is not consistent. Some studies have found that child maltreatment is a risk factor for BPD and an almost number of studies have reported no significant association. Moreover, one form of childhood maltreatment, neglect, has been understudied for decades. This is despite the fact that individuals who experience neglect are also at risk of poor outcomes like other forms of abuse (e.g., sexual, physical) such as increased rates of mental illness (e.g., PTSD, MDD), criminal behavior, and substance use (Widom, 1998). Finally, although there is a rather robust literature on serotonergic genotypes associated with BPD traits, there is virtually no research on the relationship between MAOA and BPD. This is in spite of several studies showing a relationship between MAOA and ASPD traits, which according to the leading biosocial model is a common genetic vulnerability shared by ASPD and BPD. Furthermore, one study by Ni and colleagues (2007) found results that are in contrast to the MAOA findings on ASPD suggesting that the low MAOA activity was is associated with increased risk of antisocial and violent traits. However, their findings only reached the trend level and was based on a clinical sample that was overwhelmingly female. It appears there is also a lack of research using prospective studies

examining the relationship between the MAOA genotypes and antisocial traits. Taken together, there remain several unanswered questions about the relationship between BPD, ASPD, and childhood maltreatment, genotypes of impulse-control disorders like MAOA, and the interaction between genetic and environmental factors that lead to the development of these two disorders.

Research Questions and Hypotheses

The Biosocial Model of Borderline Personality Disorder (BPD) originally proposed by Linehan (1993) regarded BPD as a disorder of affective dysregulation and emotional vulnerability that manifests in three ways; heightened emotional sensitivity, inability to regulate intense emotional responses, and a slow return to emotional baseline (Linehan, 1993). More recent evidence has shown support for an inherited emotional vulnerability in individuals who develop BPD and ASPD in studies based on a wide range of methodologies, including self-reports, psychophysiological measures, and neural imaging. Furthermore, there is an increasing body of evidence that suggests at least part of this temperamental predisposition can be linked to deficits in the monoamine neurotransmitter system with serotonergic and dopaminergic genotypes associated with BPD and ASPD showing strong heritability in twin studies. Current thinking suggests that the genotypes associated with impulse-control disorders and negative emotionality, such as low MAOA activity, interact with negative environmental factors such as childhood maltreatment to increase risk for the development of BPD and ASPD. Evidence also suggests that there are a wide range of both child and familial or parental characteristics associated with an increased risk of BPD and ASPD that interact with genetic vulnerabilities to increase risk for these disorders. However, there remain inconsistencies in several important areas related to a biopsychosocial model of BPD and ASPD with discrepant findings surrounding which neurotransmitter-related genotypes are related to BPD, the exact nature of the relationship

between childhood maltreatment and BPD, and other factors that potentially moderate or mediate the relationship between the biological and environmental risk factors associated with BPD and ASPD including gender and race.

The goal of this dissertation is to expand upon the current literature of Gene X Environment interactions in the development of BPD and ASPD. We had several hypotheses examining these relationships. Firstly, we expected that similar to the findings of Widom and colleagues (2009) that childhood maltreatment would predict increased risk of BPD diagnosis in males, but not for females in this sample. Secondly, we hypothesized that there would not be differences by race for BPD and ASPD, with both White and Black participants with a history of childhood maltreatment showing increased risk of these disorders. However, we did expect the interaction between MAOA genotype and childhood maltreatment to differ by race with results similar to Widom and Brzustowicz (2006). Specifically, we expected that MAOA genotype would moderate the relationship between BPD and ASPD, for White, non-Hispanic participants, but not for Black participants. This study is the first to examine the unique effects of MAOA genotype for Black and White females compared to their male counterparts, as previous research has either included these groups, but not specifically examined the unique factors of gender and race or excluded females all together. This study is also unique in that it used court-documented cases of child abuse to provide prospective reports of childhood maltreatment.

Hypotheses

1. Childhood maltreatment will predict increased risk for ASPD, ASPD symptoms, impulsivity, and suicide attempts for males and females, and increased risk of BPD and BPD symptoms in males.

2. There will be not be differences in these relationships by race (e.g., Whites & Blacks) for these predictions.
3. There will be an interaction between child maltreatment and MAOA genotype to predict BPD, BPD symptoms, ASPD, ASPD symptoms, impulsivity, and suicide attempts. However, based on the work of Widom and Brzustowicz (2006), the interaction between MAOA genotype and childhood maltreatment is expected to differ by race (e.g., Whites & Blacks). Specifically, MAOA genotype will moderate the influence of childhood maltreatment on the risk of BPD for White, but not for Blacks.

CHAPTER 2

Method

Design & Participants

The following description of the current study's participants, procedures, and descriptions of variables was adapted from Widom (1989b), Widom et al. (2009), Widom and Brzustowicz (2006) and the documentation process by Dr. Cathy Spatz Widom of the original study's purpose and design. The following information is used with permission from Dr. Widom [see Widom (1989b) for details of the design and subject selection].

The original sample of abused and neglected children consisted of substantiated cases of childhood physical and sexual abuse and neglect processed from 1967 to 1971 in the county juvenile (family) or adult criminal courts of a Mid-western metropolitan area. Cases of abuse and neglect were restricted to children 11 years of age or younger at the time of the incident to represent childhood maltreatment. Excluded from the sample were court cases that represented: (a) adoption of the child as an infant; (b) involuntary neglect only, usually resulting from the temporary institutionalization of the legal guardian; (c) placement only; or (d) failure to pay child support.

A control group of children without documented histories of childhood abuse and/or neglect was matched with the abuse/neglect group on age, sex, race/ethnicity, and approximate family social class during the time that the abuse and neglect records were processed. Matching for approximate family social class was important in this study because it is theoretically plausible that any relationship between child abuse and neglect and subsequent outcomes may be confounded with or explained by social class differences (MacMillan et al., 2001; Widom,

1989a). The matching procedure used here is based on a broad definition of social class that includes neighborhoods in which children were reared and schools they attended. For children of school age, records of more than 100 elementary schools for the same time period were used to find matches with children of the same sex, race, date of birth (+6 months), class in elementary school during the years 1967 to 1971, and home address, preferably within a five-block radius of the abused/neglected child. Children who were under school age at the time of the abuse and/or neglect were matched with children of the same sex, race, date of birth (+/−1 week), and hospital of birth through the use of county birth record information. Overall, there were matches for 74% of the abused and neglected children.

Nonmatches occurred for a number of reasons. For birth records, nonmatches occurred in situations when the abused and neglected child was born outside the county or state or when date of birth information was missing. For school records, nonmatches occurred because of lack of adequate identifying information for the abused and neglected children or because the elementary school had closed over the last 20 years and class registers were unavailable. Re-analyses of earlier findings were conducted using only matched pairs, and the results did not change with the smaller sample size (Widom, 1989b; Widom, DuMont, & Czaja, 2007). Where possible, two matches were found to allow for loss of comparison group members. Thus, individuals who were initially selected for the comparison group who were reported in the official abuse and neglect files were eliminated and replaced with a second matched comparison subject. Any comparison group child with an official record of abuse or neglect was eliminated, regardless of whether the record was before or after the period of the study. This occurred in 11 cases. Importantly, since it is not possible to randomly assign subjects to groups, the assumption of equivalency for the groups is an approximation.

The initial phase of the study compared the abused and/or neglected children to the matched comparison group (total $N = 1,575$) on juvenile and adult criminal arrest records (Widom, 1989b). Of the original sample, 1,307 (85%) were located and 1,196 (76%) were first interviewed in 1989–1995 when the participants were at a mean age of 29.2. Those not interviewed were deceased ($n = 43$), incapable of being interviewed ($n = 8$), not found ($n = 268$), or refused to participate ($n = 60$). A second phase involved locating and interviewing both the abused and/or neglected and comparison groups during 1989–1995, approximately 22 years after the incidents of abuse and neglect ($n = 1,196$). Follow-up interviews were conducted in 2000 to 2002 and in 2003 to 2005, when participants were a mean age of 39.5. Of the 1,196 initial interviews, 1,117 (93%) were located and 896 (75%) participated in the second interview. Importantly, there were no significant differences between the original sample and the follow-up samples with regard to demographic characteristics of sex, race, poverty in census tract, current age, and group status (maltreated vs. matched controls). In general, approximately 56–58% were abuse and neglected; 62–66% were White; and 48–51% were males. Using participant-reported race/ethnicity, the minority group includes Black (35.1%) and Hispanic (4.1%). The average highest grade of school completed for the sample was 11.47 ($SD = 2.19$), and the median occupational level was semiskilled workers. Thus, the overall sample is skewed toward the lower end of the socioeconomic spectrum. Maltreatment was divided into types: physical abuse (7%), sexual abuse (9%), neglect (72%), and multiple maltreatment (12%).

Respondents were interviewed in person both times, usually in their homes, or, if the respondent preferred, another place appropriate for the interview. The interviewers were blind to the purpose of the study, to the participants' group membership, and to the inclusion of an abused and/or neglected group. Similarly, the participants were blind to the purpose of the study

and were told they had been selected to participate as part of a large group of individuals who grew up in that area in the late 1960s and early 1970s. Institutional Review Boards (IRB) approval was obtained for study procedures prior to data collection and individuals who participated signed a consent form acknowledging that they understood the conditions of their participation and that they were participating voluntarily.

Genetic data for this study were collected during 2003–2005 in the context of a medical status examination (including blood collection through venipuncture) and interview (N = 806). Of those interviewed, 638 (82%) consented to provide blood and, of these, 631 gave permission for DNA extraction and analyses. IRB restrictions prevented the collection of blood from an additional 31 study participants who were residing in prisons at the time.

The initial sample included 1196 participants. Participants who did not participate in interview 2 (n = 304), those without BPD data (n = 2), 201 individuals who did not have genotype data, and individuals who did not identify as Black or White, non-Hispanic (n = 45) as the Hispanic group was too small for meaningful analysis. The analytic sample represents 644 participants. The mean of age the analytic sample is 39.48 years old (SD = 3.52, range = 30.15-46.98); approximately half of the sample is female (54.5%), and around a third are Black, non-Hispanic. To assess the representativeness of the sample, characteristics of the original sample of 1196 were compared to those of the analytic sample (n = 644). Table 1 shows that there were no significant differences between the included and excluded groups in terms of rates of childhood maltreatment, age, history of suicide attempts, or ASPD symptoms. However, there were significant differences between the included and excluded groups in terms of sex, race, rates of BPD diagnosis and ASPD diagnosis, BPD symptoms, and impulsivity. The included participants had a higher percentage of females (54.5% vs. 42.6%), individuals with a BPD diagnosis (14.1% vs. 8.5), a higher number of BPD symptoms (M = 2.00 vs. M = 1.60), and impulsivity scores (M = 35.12 vs. M = 33.94), compared to those who were excluded. Excluded participants had higher rates of ASPD diagnosis (17.8% vs. 13.1%).

Measures

Official Reports of Childhood Maltreatment. Childhood physical abuse, sexual abuse, and neglect were defined through case review of official records from 1967 to 1971. Physical abuse included bruises, welts, burns, abrasions, lacerations, wounds, cuts, bone and skull fractures, and other evidence of physical injury. Sexual abuse charges included felony sexual assault, fondling or touching, rape, sodomy, and incest. Neglect cases reflected a judgment that the parents' deficiencies in childcare were beyond those acceptable by community and professional standards at that time. These cases represented extreme failure to provide adequate food, clothing, shelter, and medical attention to children.

Children with documented cases of any one of these forms of maltreatment are included in the overall maltreatment group and in the specific type of maltreatment group (i.e., physical abuse, sexual abuse, and neglect) if they experienced that particular type, but not necessarily only that type. That is, children with physical abuse cases could also have experienced neglect or sexual abuse or some other combination of maltreatment and can be included in both categories. However, only about 10% of the sample experienced more than one type of maltreatment. The control group consisted of children without documented histories of childhood maltreatment that were matched to the maltreatment sample on age, sex, race/ethnicity, childhood neighborhood, and approximate childhood family social class. Importantly, the control group establishes the base rates of pathology we would expect in a sample of adults from comparable circumstances who did not come to court attention as children as victims of abuse or neglect.

Borderline Personality Disorder (BPD). BPD was assessed with a structured interview (Jordan, Schlenger, Fairbank, & Caddell, 1996) based on DSM-III-R criteria (American Psychiatric Association, 1987) and adapted from the BPD module of the Diagnostic Interview

for Personality Disorders, Revised (DIPD-R; Zanarini, Frankenburg, & Chauncey, 1987). Participants received a diagnosis of BPD if they met at least five of the criteria for BPD, consistent with DSM-III-R and Jordan et al. (1996). The instrument assessed current (past year) BPD. BPD diagnoses were made during the follow-up interviews when the mean age for participants was 39.5 years. Validity for the BPD structured interview has been shown in a study of incarcerated women (Jordan et al., 1996). Good correspondence was reported between the survey interview BPD diagnosis and clinicians' assessments of BPD (sensitivity 77% and specificity 81%). Specific BPD characteristics that will also be included in analyses consist of the four of the following DSM-III-R criteria for BPD: impulsiveness in at least two areas that are potentially self-damaging (e.g., spending sex, substance use, shoplifting, reckless driving, or binge eating); recurrent suicidal threats, gestures, behavior or self-harm; difficulty controlling anger or inappropriate intense anger; and emotional lability with marked shifts in mood (e.g., depression, irritability, anxiety) that usually last a few hours and rarely more than a few days.

Antisocial Personality Disorder (ASPD). ASPD was measured using a psychiatric assessment that is based on the NIMH Diagnostic Interview Schedule (DIS), developed by Robins, Helzer, Croughan, and Ratcliff (1981). The DIS was used in a variety of NIMH sponsored research projects that require psychiatric assessments of large numbers of subjects (Von Kurff et al., 1987). It was also used in prisons and jails, including an assessment of the prevalence of mental disorder among inmates in Michigan prisons (Neighbors et al., 1987).

The DIS (pages 17-81) is a fully structured interview schedule (i.e., a fixed sequence of prewritten questions with sub-schedules of "probe" questions, pre-coded response categories, and detailed interviewer instructions). It was designed to allow physicians and nonphysicians to make consistent and accurate psychiatric diagnoses in patients and the general population and

represents a major methodological accomplishment. Thus, the DIS allows the use of lay interviewers in assessing the current and lifetime prevalence of psychiatric illness by DSM-III criteria and Research Diagnostic Criteria (RDC), as well as Feigner criteria. This assessment is how a diagnosis of Antisocial Personality Disorder (ASPD) was given to participants in the present dissertation study. A computer program for scoring the DIS, written in the Statistical Analysis System (SAS) programming languages, has been purchased from the authors.

The language of the DIS interview has been designed to be simple to facilitate interviewing of persons of varying education and intelligence, answerable by persons ranging from less than fifth grade education to post-graduate degrees. Interviews were conducted in person. Although the DIS is a structured interview with questions fully written out exactly as they are to be read, the interview cannot be properly administered by an untrained person no matter how familiar they are with interviews of this sort or with the diagnoses covered by the interview. Thus, prior to data collection, all interviewers received an extensive week-long training and instruction session.

Suicide Attempts. As part of the depression module of the DIS-III-R (Robins et al., 1989), participants were asked whether they had ever attempted suicide. Lifetime suicide attempts were assessed during the first interview when participants were a mean age of 29.2 years and during follow-up interviews when participants were a mean age of 39.5 years. Responses will be coded ‘‘1’’ for yes if a participant makes a positive endorsement of past suicide attempts during the first interview or the follow-up interview and ‘‘0’’ for no if they do not endorse any history of suicide attempts at either timepoint.

Impulsivity. During the interview 2, participants were administered the Barratt Impulsivity Scale (BIS-II: Barratt, 1985). The BIS-II is a 16-item instrument to assess

impulsivity and has been used in social and biological studies of violence (Barratt, 1985; Coccaro et al., 1993). Items included a number of statements about individuals' dispositions (e.g., "I plan things carefully before acting," "I am a careful thinker"); participants responded with the extent to which they agreed with each statement on a 4-point Likert scale. Higher scores indicate greater impulsivity. This measure had acceptable internal consistency ($\alpha = 0.64$).

Monoamine Oxidase-A (MAOA). *MAOA genotypes were assessed based on the same methodology employed by Widom and Brzustowicz (2006). A description of this methodology for assessing MAOA genotypes with this sample appeared in that paper and is used here.* At mean age 41, DNA was obtained from usable blood samples from 617 study members using the PureGene (Gentra Systems Inc) system according to the manufacturer's instructions. The MAOA promoter polymorphism was genotyped by PCR amplification using primers MAOA-F2 5'-(TGCTCCAGAAACAT- GAGCAC)-3' and MAOA-R2 5'-(GGACAGGCTGTAGGAGGT-GTC)-3'. PCR reactions contained 80 ng of template DNA, 1.0 U AmpliTaq Gold polymerase (Applied Biosystems), 1.0 M of each primer, 0.2 mM dNTP, 2.0 mM $MgCl_2$, and 2µl of GeneAmp 10X buffer II (Applied Biosystems), in a 20 µl volume. After 12 min at 96°C, 40 cycles were done at 96°C for 15 s, at 67.7°C for 20s, and at 72°C for 30s, followed by a final extension step at 72°C for 10 min. Products were resolved by Higher Resolution Microplate Array Diagonal Gel Electrophoresis (Day and Humphries 1994), using an 8% polyacrylamide gel run at 150 V for 1h and 15 min.

Representative genotypes were identified and sequenced using a Beckman-Coulter CEQ8000 semi-automated fluorescent sequencing system to confirm the sizes and number of repeats present in the observed alleles. These samples were included as size standards on subsequent gels. DNA was visualized by staining with ethidium bromide and gel images were

captured and analyzed using Kodak 1D image analysis software. Any ambiguous genotypes that could not be resolved by repeat PCR and electrophoresis were determined by direct DNA sequencing. One male individual was identified as heterozygous for the MAOA promoter VNTR polymorphism. Presence of a Y-chromo- some was verified by PCR amplification of the marker DYS392. As it is unknown if this subject has two copies of the MAOA gene (either through an XXY karyotype or a segmental duplication) or is mosaic for MAOA genotype, he was excluded from further analysis.

As shown in Table 2 overall frequencies of the MAOA alleles were .017 for 1 repeats, .018 for 2 repeats, .403 for 3 repeats, .010 for 3.5 repeats, .543 for 4 repeats, and .008 for 5 repeats. Chi-square analysis revealed no evidence for deviation from Hardy-Weinberg equilibrium ($\chi^2(5) = 1.79$, $p = 0.88$). There was no significant difference in allele frequencies for the maltreated group compared to controls ($\chi^2(11) = 11.95$, $p = 0.367$) (See Table 2). There is good agreement about the levels of expression associated with the two most common allelic variants (3 and 4 repeats) of the functional promotor polymorphism in MAOA (Deckert, et al., 1999; Denney, Koch, & Craig, 1999; Nikulina, Widom, & Brzustowicz, 2012; Sabol, Hu, & Hamer, 1998; Widom & Brzustowicz, 2006). As these consisted of 95% of the observed alleles, we limited further analyses to these alleles. Individuals with genotypes other than 3- and 4-repeat allele combinations ($n = 50$) were excluded from analyses because levels of expression associated with these are ambiguous (Deckert, et al., 1999; Denney, Koch, & Craig, 1999; Nikulina, Widom, & Brzustowicz, 2012; Sabol, Hu, & Hamer, 1998; Widom & Brzustowicz, 2006). Two males were identified as heterozygous for the MAOA promotor polymorphism. Presence of a Y-chromosome was verified by PCR amplification of the marker DYS392. As it is unknown if these participants have two copies of the MAOA gene (either through an XXY

karyotype or a segmental duplication) or is mosaic for MAOA genotype, they were excluded from further analysis. Males with one or females with two copies of the 3-repeat allele were designated as low activity and males with one or females with two copies of the 4-repeat allele were designated as high activity (Deckert, et al., 1999; Denney, Koch, & Craig, 1999; Nikulina, Widom, & Brzustowicz, 2012; Sabol, Hu, & Hamer, 1998; Widom & Brzustowicz, 2006). Heterozygous females were also included as a separate group (similar to Nikulina, Widom, & Brzustowicz, 2012). The final sample consisted of 592 participants, 336 (56.8%) maltreated and 256 (43.2%) controls.

Statistical Analyses

Statistical analyses were performed in SPSS version 27. Categorical independent variables included in the analyses were coded as follows: race/ethnicity, Black, non-Hispanic = 1; White, non-Hispanic = 0; sex, female = 1; male = 0, official report of childhood abuse/neglect = 1; control = 0; MAOA Genotype DC 1, males with 3- and females with 3-,3- genotype = 1, all other genotypes = 0; and MAOA Genotype DC 2, heterozygous females with 3-,4- genotype = 1, all other genotypes = 0. Chi-square analyses were used to assess the distribution of genotype frequency, representativeness of the sample, and group (child abuse and neglect versus control) differences.

Logistic regressions were used to determine the effect of child abuse and neglect as well as the interaction between child abuse and neglect and MAOA genotype on the dichotomous variables (BPD diagnosis, ASPD diagnosis, and suicide attempts). Nagelkerke R^2 was used as a pseudo-R^2 measure of effect size and adjusted odds ratios (AOR) were reported for logistic regressions. Ordinary least squares (OLS) regressions were used for the number of BPD

symptoms, ASPD symptoms, and impulsivity scores. Race, age, and sex were controlled for in all analyses.

CHAPTER 3

Results

The results are organized into five sections. The first section presents descriptive statistics for individuals with history of child maltreatment and controls on all variables. The next three sections present the results of the extent to which childhood maltreatment predicts BPD diagnosis and symptoms, ASPD diagnosis and symptoms, and suicide attempts and impulsivity, respectively. The final section presents the results of the gene (MAOA) by childhood maltreatment interactions in predicting all dependent variables.

Demographic Characteristics of the Sample

Table 3 provides the descriptive statistics as well as results for chi-square and t-test analyses for the previously maltreated children and matched controls separately. There were no demographic differences between the two groups in terms of sex, race, or age. Overall, the two groups were around 39 years of age, slightly more than half were female, and approximately a third were Black, non-Hispanic. For all dependent variables except one, maltreated children were at greater risk than controls. The maltreated group showed a higher rate of suicide attempts, BPD symptoms, ASPD diagnoses, and ASPD symptoms, and were more impulsive than controls. In contrast to published findings (Widom, Czaja, & Paris, 2009), there was no significant difference between individuals with histories of childhood maltreatment and controls for BPD diagnosis.

Childhood Maltreatment and Borderline Personality Disorder Diagnosis and Symptoms

Table 4 shows the extent to which child maltreatment overall and for males, females, Blacks, and Whites separately predicts BPD diagnosis and BPD symptoms in adulthood. For

BPD diagnoses, bivariate analyses show that a history of childhood abuse and neglect did not predict increased risk of BPD for the sample overall ($\chi^2(4) = 6.83$, $p = 0.15$) or for females ($\chi^2(3) = 2.17$, $p = 0.54$; AOR = 1.44). However, childhood maltreatment predicted BPD diagnosis for males ($\chi^2(3) = 14.16$, $p = 0.002$), although the effect of child maltreatment for males disappeared (AOR = 1.58, $p = .23$), when controls were introduced for race (AOR = 2.62, $p = .01$) and age covariates (AOR = 7.22, $p = .007$). Child maltreatment did not predict BPD diagnoses for Black participants ($\chi^2(3) = 3.12$, $p = 0.37$; AOR = 1.19, $p = .67$). Bivariate analyses showed that child maltreatment predicted BPD diagnoses for White participants ($\chi^2(3) = 11.12$, $p = 0.01$); however, when controls were introduced, childhood maltreatment became non-significant (AOR = 1.65, $p = .20$).

The results for the relationship between child maltreatment and BPD symptoms in adulthood were different. Individuals with histories of childhood abuse and neglect overall had a significantly higher number of symptoms of BPD ($F(4, 587) = 3.63$, $p = .006$; adj. $R^2 = .02$; $p = .02$) and this was also the case for maltreated males compared to controls ($F(3, 268) = 6.98$, $p \leq .001$; adj. $R^2 = .06$ $p \leq .001$, but not for females ($F(3, 316) = 0.72$, $p = .54$; adj. $R^2 = -.003$; $p = .63$). When analyzed separately by race, the unadjusted model for BPD symptoms was significant for White participants ($F(3, 382) = 3.68$, $p = .01$; adj. $R^2 = .02$, $p = .02$), but became non-significant with the introduction of controls ($t = 1.61$, $p = .11$). Childhood maltreatment was not a significant predictor of BPD symptoms for Black participants ($F(3, 202) = 2.35$, $p = .07$; adj. $R^2 = .02$, $p = .13$).; *SE*; *SE* = standard error.; *SE* = standard error.; *SE* = standard error.

Childhood Maltreatment and Antisocial Personality Disorder Diagnosis and Symptoms

Table 5 shows that individuals with a history of childhood maltreatment were at increased risk of an ASPD diagnosis, with previously abused and maltreated children nearly two

times as likely to be diagnosed with ASPD as an adult compared to controls ($\chi^2(4) = 16.10$, $p = 0.003$; AOR = 1.98, $p = .01$). When examined separately by sex, childhood maltreatment was associated with an increased risk of ASPD for males only ($\chi^2(3) = 8.12$, $p = 0.04$), but not for females ($\chi^2(3) = 1.49$, $p = 0.66$; AOR = 1.39, $p = .42$). When examined separately by race, White abused and neglected children were more than 2.5 times more likely than controls to be diagnosed with ASPD ($\chi^2(3) = 14.76$, $p = 0.002$; AOR = 2.64), whereas this was not the case for abused and neglected Black children ($\chi^2(3) = 4.80$, $p = 0.19$; AOR = 1.47).

Abused and neglected children overall were also at risk of having a higher number ASPD symptoms compared to controls ($F(4,586) = 4.02$, $p = .003$; adj. $R^2 = .02$, $p \leq .001$). When examining the participants separately, by sex and race, the same pattern emerged for ASPD symptoms: for males ($F(3,267) = 3.61$, $p = .01$; adj. $R^2 = .03$, $p = .43$), females ($F(3,316) = 4.65$, $p = .003$; adj. $R^2 = .03$, $p = .24$), and Blacks ($F(3,202) = 5.28$, $p = .002$; adj. $R^2 = .06$, $p = .003$) and Whites ($F(3,383) = 15.26$, $p < .001$; adj. $R^2 = .10$, $p = .02$), those with a history of childhood abuse and neglect had a higher number of symptoms of ASPD as adults compared to controls.

Childhood Maltreatment and Suicide Attempts and Impulsivity

As shown in Table 6, individuals with a history of childhood abuse and neglect were at increased risk of suicide attempts ($\chi^2(4) = 30.78$, $p < 0.001$), with abused and neglected children 2.6 times (AOR = 2.60, $p = .03$) more likely to have attempted suicide as an adult compared to matched controls. There were unique differences when looking at males and females separately. Previously abused and neglected females were nearly three times ($\chi^2(3) = 14.09$, $p = 0.003$; AOR = 2.87, $p \leq .001$) more likely to have attempted suicide as adults compared to controls, whereas for males, there was a significant bivariate relationship ($\chi^2(3) = 10.40$, $p = 0.02$) and a non-significant trend when controls were introduced (AOR = 2.12, $p = .06$). An increase in risk of

suicide attempts was found for both races with Black ($\chi^2(3) = 11.81$, $p = 0.008$; AOR = 2.47, $p = .03$) and White maltreated children ($\chi^2(3) = 18.20$, $p < 0.001$; AOR = 2.63, $p = .001$) being about 2 ½ times more likely to report having made a have suicide attempt compared to matched controls.

Table 6 also shows that a history of childhood abuse and neglect was associated with increased impulsivity in adulthood ($F(4,586) = 3.35$, $p = .01$; adj. $R^2 = .02$, $p = .03$). Only maltreated males had higher impulsivity scores than controls ($F(3,267) = 5.17$, $p = .002$; adj. $R^2 = .04$, $p = .01$). None of the other specific groups showed higher impulsivity scores: for females ($F(3,316) = 1.25$, $p = .29$; adj. $R^2 = .002$, $p = .18$); Blacks ($F(3,202) = 2.00$, $p = .12$; adj. $R^2 = .01$, $p = .10$) or Whites ($F(3,381) = 1.81$, $p = .14$; adj. $R^2 = .006$, $p = .25$).

Interactions Between MAOA Genotype, Childhood Maltreatment, and Race

To test whether there are interactions between MAOA genotype and child maltreatment, a series of regressions were run for each of the dependent variables (see Tables 7-9). Two dummy coded variables for MAOA genotype were included: DC1 represented males with one 3-repeat and females with two 3-repeats (low MAOA activity) compared to all other genotypes and DC2 represented the heterozygous females with the 3,4-genotype compared to all other genotypes (similar to Nikulina, Widom, & Brzustowicz, 2012). The results (see Table 8) show a significant main effect of childhood maltreatment on ASPD symptoms ($F(10,580) = 6.65$, $p < .001$, adj. $R^2 = .09$, $p = .08$; $t = 3.30$, $p = .001$), similar to the prior regressions. There were no other significant main effects for childhood maltreatment and no significant two-way G x E interactions for all outcomes assessed here. However, there was a significant three-way interaction of childhood maltreatment, heterozygous female dummy code (DC2), and race for BPD diagnosis (AOR = 0.19, 95%CI 0.04-0.79, $p = .02$). As shown in Figure 1, Black maltreated

females who were heterozygous for the MAOA genotype were at decreased risk of BPD, that is, that the heterozygous MAOA genotype was protective for Black maltreated females. There was also a marginally significant three-way interaction of maltreatment, maltreatment, heterozygous female dummy code (DC2), and race for BPD symptoms ($F(10,581) = 2.29$, $p = .01$, adj. $R^2 = .02$, $p = .99$; $t = -2.17$, $p = .03$). This also suggested that the heterozygous genotype was protective for Black maltreated females and that they were at decreased risk of BPD symptoms. Finally, there was also a significant main effect of DC1 or Low MAOA for impulsivity, but a follow up analysis was not significant ($t(589) = 1.05$, $p = .30$).

CHAPTER 4

Discussion

The aim of this dissertation was to examine the role of gene-by-environment interactions in the development of Borderline Personality Disorder (BPD) and Antisocial Personality Disorder (ASPD) with particular attention to the influence of childhood maltreatment and the monoamine genotype (e.g., monoamine oxidase A or MAOA). Specifically, this research examined whether MAOA genotype moderates the relationship between childhood maltreatment and BPD and ASPD, respectively. In addition, this work examined BPD's presentation in men, specific symptoms common to both ASPD and BPD, differences by race in gene-by-environment interactions on the risk of developing BPD and ASPD, and the role of neglect in relation to the development of BPD.

Child Maltreatment and Borderline Personality Disorder

Unlike previous studies using this data (Widom, Czaja, & Paris, 2009), the findings of the current analysis revealed that childhood maltreatment was not a significant predictor of BPD diagnosis for the sample overall. Although the rates of BPD diagnosis in the maltreated (16.1%) was higher than in the control participants (8.5%), this difference did not reach significance in the current sample. However, it is noteworthy that the prevalence in the overall sample is substantially higher than rates reported in the general population (1.4%-5.9%; Lenzenweger, Lane, Loranger, & Kessler, 2007; Grant et al., 2008). Previous research with this data (Widom, 1998) has highlighted the importance of contextual factors in understanding the consequences of childhood abuse and neglect. It is possible that the higher rates of BPD are due in part to the low SES status of participants in the present sample, which may have made the two groups similar in

terms of BPD diagnosis. Research has shown that individuals in lower income brackets have higher rates of BPD than individuals in middle-to-upper income brackets (Grant et al., 2008). Several prospective studies also show a positive relationship between low SES (Cohen et al., 2008; Crawford et al., 2009) and/or receiving public assistance (Stepp, Keenan, Hipwell, & Krueger, 2014; Stepp et al., 2014) with number of BPD symptoms in adolescents and adults. Low SES generationally has been shown to be associated with increased risk of personality disorders (Cohen et al., 2008). Specifically, low parental SES is associated with increased rates of BPD symptoms in their offspring, even when controlling for trauma exposure, poor parenting skills, and lower mean verbal IQ (Cohen et al., 2008). As discussed in Widom (1998), children in low SES neighborhoods and communities might have less access to resources and may not have as much social support as their higher SES peers. In essence, there may not be an adequate environmental support network within the family or community to mediate the child's response to trauma.

Despite the lack of a significant relationship between BPD diagnosis and childhood maltreatment, this was not the case for BPD symptoms. Childhood maltreatment was predictive of BPD symptoms. Previous research (Meich et al., 1999; Stepp et al., 2013) has shown more robust associations between childhood variables and BPD symptoms compared to diagnoses. In part, this could be due to the categorical nature of DSM disorders which requires individuals to have a certain number of symptoms in order to receive a diagnosis. A major drawback of this approach is that some individuals will not receive a diagnosis if they do not meet the required symptom threshold, despite potentially experiencing severe impairment and dysfunction associated with the few symptoms they do exhibit. This is especially true for personality disorders, which show a high level of co-occurrence of symptoms from different personality

diagnoses (Huprich & Bornstein, 2007; Skodol, 2012) and further exemplified in Personality Disorder not otherwise specified (PDNOS), the most commonly diagnosed PD (Verheul, Bartak, & Widiger, 2007). To address this issue, some have argued that it would be more beneficial to conceptualize personality disorders using a dimensional model. Notably, the most recent edition of the DSM provided an alternative model for diagnosing disorders of personality in a first step towards modifying the current categorical diagnostic system (APA, 2013).

Regardless of the distinction between diagnoses and symptomology, it is important to underscore that a high number of BPD symptoms suggest significant impairment in an individual's functioning (APA, 2013; Linehan, 1993; Tomko et al., 2014; Trull et al., 2010). One common thread in the literature on BPD is that the symptoms of this disorder cause pervasive instability in emotional, interpersonal, and behavioral functioning (APA, 2013; Linehan, 1993; Tomko et al., 2014; Trull et al., 2010). Many of these individuals engage in potentially dangerous impulsive and self-damaging behavior (Linehan, 1993). People with BPD show higher rates of impaired social and emotional functioning than those without BPD (Tomko et al., 2014), and evidence suggests that individuals with BPD have significantly lower quality of life. As previous studies with this data have shown (Widom, 1998; Widom, 2000), individuals with a history of abuse and neglect can have significant impairment across several domains of functioning. More specifically, research suggests individuals maltreated as children are more likely to be underemployed or unemployed, have a history of divorce or separation, have higher rates of psychopathology, and are more likely to engage in criminal behavior including delinquency, adult criminality, and violence compared to non-maltreated individuals. However, it is important to remember that a sizable percentage of abused and neglected children do not go

on to engage in criminal behavior, violence, or develop personality disorders (Widom, 1998; Widom, 2000).

Childhood Maltreatment and Antisocial Personality Disorder

Previous studies (Luntz & Widom, 1994; Widom, 1998) show that abused and neglected individuals are significantly more likely to meet criteria for ASPD. The current analyses also found this relationship. Previously maltreated children were nearly two times as likely to be diagnosed with ASPD as adults compared to controls. They were also at risk for having a higher number of ASPD symptoms. The link between childhood maltreatment and ASPD underscores the need for early intervention as the consequences of the disorder can have effects not only on an individual level, but societally, as those with ASPD are more likely to engage in crime and violence (Beauchaine et al., 2009). However, it should be noted, though, that the vast majority of the abused and neglected sample did not meet criteria for ASPD, suggesting that factors other than childhood maltreatment are involved in the etiology of ASPD. Other potential contributors to ASPD development include neurological deficits associated with genetics and/or early traumatic injury, biological predispositions for aggression, and operant conditioning in economically disadvantaged or violent neighborhoods (Beauchaine et al., 2009). Low SES has also been associated with conduct disorder and antisocial behavior in young adulthood (Meich et al., 1999). Taken together, ASPD appears to have multiple etiological factors; however, the current findings underscore the negative impact that childhood maltreatment has on the manifestation of antisocial behavior.

Childhood Maltreatment and Suicide Attempts

As expected, childhood maltreatment was associated with increased lifetime rates of suicide attempts. Rates of suicide in individuals with BPD are significantly elevated compared to

the general population, with around 8-10% completing suicide (APA, 2013). Previous studies with this data (Widom, 1998; Widom & Li, 2020) have reported higher rates of suicide attempts in abused and neglected children compared to controls. A recent meta-analysis further underscored this relationship; providing evidence to support a significant positive relationship between childhood maltreatment and suicidal behavior (Liu et al., 2017). In addition to suicide attempts, childhood maltreatment has also been shown to be associated with increased symptoms of depression, dysthymia, PTSD, GAD, substance abuse, and environmental risk factors for suicide like social isolation, homelessness, and physical illness or disabilities (Liu et al., 2017; Widom & Li, 2020). However, it appears that the relationship between childhood maltreatment and suicide attempts could also be mediated by psychiatric disorders like ASPD and substance abuse, in addition to socioeconomic factors like homelessness (Widom & Li, 2020). Research on risk factors and individual vulnerabilities of suicide have shown that a combination of these factors, like childhood maltreatment and psychopathology, are associated with increased risk of completed suicide. Suicide is currently the second leading cause of death among ages 10 to 34 in the US (CDC, 2018), and the presence of impulsive behavior, aggression, and interpersonal conflict like that observed in ASPD and BPD are associated with increased risk of death by suicide (Turecki & Brent, 2016). Furthermore, individuals with ASPD and BPD are more likely to have premature death and to die from suicide compared to those without these disorders (Krasnova et al., 2018; Turecki & Brent, 2016). Taken together, there appears to be a clear link between early childhood adversity, such as childhood maltreatment, and lifetime suicide risk with supportive results from prospective, retrospective, and case-control study designs (Turecki & Brent, 2016; Widom & Li, 2020). It is important for future research to examine interventions

that can mediate the relationship between childhood maltreatment and other risk factors to decrease risk of suicide and suicidal behaviors.

Childhood Maltreatment and Impulsivity

The current findings showed that adults with a history of childhood maltreatment had higher levels of symptoms of impulsivity compared to controls. Impulsive behavior is one of the DSM criterions for both ASPD and BPD (APA, 2013) and, thus, it is not surprising that maltreated children would have higher impulsivity trait scores. Trait impulsivity has been highlighted by several models of BPD and ASPD etiology as a critical precursor to both disorders (Beauchaine et al., 2009; Crowell, Beauchaine, & Linehan, 2009). The bio-social model of ASPD and BPD proposed by Beauchaine and colleagues (2009) specifically suggests there is an interaction between a biologically inherited risk for early impulsivity and a high-risk childhood environment which leads to personality disorder symptoms in early adulthood. There is also evidence that suggests that impulsivity and aggression are important aspects of all Cluster B personality disorders, including ASPD and BPD (Turner, Sebastian, & Tüscher, 2017). In physiological studies of impulsivity in ASPD and BPD, participants with BPD appear to have response inhibition deficits similar to those with ASPD when exposed to stress or high levels of emotionality were induced (Turner et al., 2017). Impulsivity in the presence of BPD has also been associated with increased risk of suicide attempts in adolescents and young adults (Andrewes et al., 2019). Additionally, impulsive-aggression has been shown to be related to familial transmission of suicidal behaviors (Turecki & Brent, 2016). Taken together, it appears that impulsivity may serve as an important developmental indicator of increased lifetime risk of both ASPD and BPD. Future studies should aim to examine impulsivity longitudinally in children with and without histories of maltreatment to better elucidate the role biological

predispositions for impulsive traits play in the development of antisocial and borderline personality disorders.

Sex Differences

When examined separately by sex, childhood maltreatment was only a significant predictor of BPD diagnosis for males, but this effect disappeared once controlling for race and age covariates. This suggests that age and race were more significant predictors of BPD diagnosis than childhood maltreatment. Notably, these results were both similar and divergent from prior studies using this data (Widom, Czaja, & Paris, 2009). Specifically, there was evidence of a significant relationship between childhood maltreatment and borderline personality disorder symptoms for males. However, there was no relationship between childhood maltreatment and BPD symptoms for females. It is worth noting that the earlier study with this data did have a larger sample size (n = 892), which could explain the discrepancy with the present results. Another possible reason for this discrepancy is that there are other characteristics of invalidating or high-risk environments that are stronger predictors for development of BPD diagnosis in females. For example, some studies suggest a moderate relationship between harsh parenting practices and low caregiver warmth and BPD symptoms in adolescence (Stepp et al., 2014). There are also criticisms of the concept of BPD from feminist perspectives. From a social constructionist viewpoint, BPD represents a modern example of historic attempts to pathologize women's behavior, specifically in response to oppression and abuse faced by women in society. In contrast, a social causation perspective poses that the creation of BPD as a disorder is an attempt by society to pathologize women's response to trauma (Shaw & Proctor, 2005). From this perspective, BPD is not a form of psychopathology and instead reflects an attempt to pathologize and subsequently subvert women behaving outside of expected gender norms or to

undermine women's responses to systemic traumatization and lack of adequate societal supports to address their trauma. If this is the case, the DSM criteria of BPD are specifically designed for females and could explain why BPD has historically been diagnosed much more commonly in females than males (Linehan, 1993). However, there is some literature that has failed to find a significant difference in rates of BPD in males and females (Grant et al., 2008; Widom, Czaja, & Paris, 2009; Trull et al. 2014), suggesting that the symptoms of BPD are not as gendered as previously thought. It is also possible that SES has a significant impact on this relationship, with females of low SES background experiencing other burdens of their environment that are more predictive of BPD; this is in contrast to their medium or high SES counterparts where they don't experience as much hardship and therefore you will see the link between childhood maltreatment and BPD.

For ASPD diagnosis, males with a history of childhood abuse and neglect were at increased risk, compared to control males. This is similar to previous literature that shows abused and neglected males show higher rates of ASPD in adulthood compared to controls (Luntz & Widom, 1994; Widom, 1998). More specifically, Luntz and Widom (1994) found that child abuse and neglect predicted ASPD diagnosis and symptoms when controlling for age, sex, race, socioeconomic status, and criminal history. On the other hand, for females, there was no relationship between childhood maltreatment and ASPD diagnosis, despite previous studies with this data (Maxfield & Widom, 1996; Widom, 1998) finding that maltreated females were at increased risk for ASPD diagnosis and criminal behavior. One possibility is that ASPD diagnosis may be underestimated in females due to the requirement of evidence of childhood conduct problems (Dolan & Vollm, 2009). Studies of conduct disorder have shown that girls tend to show disturbed conduct relationally, like bullying or threatening people, lying, cheating, or being

cruel to others (Kim-Cohen et al., 2005). In contrast, boys are more likely to show behaviors classically associated with conduct disorder like hitting others, fighting, destroying property, and stealing. With the latter being more obvious egregious behaviors, it is possible that conduct problems are subjectively observed as more common in boys than in girls as the former's behavior being more classically viewed as "problematic". This biased interpretation of children's behavior leads to the prevalence of conduct disorder being underestimated in girls and subsequently causing the inaccurately low levels of ASPD in adult females.

Conversely, when examining ASPD symptoms, both males and females with a history of maltreatment were at increased risk. These findings provide further support to biosocial models of ASPD development (Beauchaine et al., 2009) that suggest childhood adversity is linked to antisocial behavior. Notably, the present studies lack of sex differences in ASPD symptoms perhaps suggests that males and females are more similar behaviorally as once thought and that there are larger differences within these groups, rather than between, in terms of psychopathology. There has historically been the presumption in psychological research is that males and females express psychological distress differently, with externalizing versus internalizing behaviors respectively. However, research has shown that this is not necessarily the case (Maxfield & Widom, 1996; Widom, 1998) with maltreated females showing externalizing symptoms like ASPD and criminal behavior. With changing gender norms for both men and women, it is likely that these similarities will continue to be observed for behavior classically thought as representation of one sex versus the other.

Maltreated females were specifically at increased risk of suicide attempts showing nearly 3 times higher risk than female controls. For males specifically, there was no longer a significant effect of childhood maltreatment on suicide attempts. These findings contrast with prior studies

using this data (Widom, 1998; Widom & Li, 2020), which observed that both maltreated males and females were at increased risk for suicide attempts compared to controls, even when controlling for age, race, and criminal history. However, the present literature suggests that there are higher rates of suicide attempts in females compared to males (Canetto & Sakinofsky, 1998; Turecki & Brent, 2016). In contrast, males have higher rates of suicide deaths, which is believed to be related to more lethal methods used by males (Turecki & Brent, 2016). Moreover, a meta-analysis by Liu and colleagues (2017) found a stronger effect between childhood maltreatment and suicidal behavior for females, similar to the present results. However, this was only observed for dichotomous outcomes, and although continuous outcomes also showed a significant association with suicidal behaviors, the strength of this relationship was similar for both males and females (Liu et al., 2017).

Unexpectedly, our results showed that only males with a history of childhood maltreatment showed an increased risk for higher levels of impulsive traits; females showed no increased impulsivity scores. Paris (1997) was one of the first to suggest that perhaps BPD and ASPD are gendered representations of the same psychopathology, citing shared symptoms of the disorder including risk of suicide and impulsivity. However, Beauchaine and colleagues (2009) suggest that there are differences by sex, in that males will exhibit features of ASPD, and females will show features of BPD. Unfortunately, we had expected to see that impulsivity would also be a significant predictor of BPD for females as this would support the idea that trait impulsivity is an important precursor for both sexes in developing BPD and ASPD. However, it is possible that due to factors like low SES or experiencing other childhood adversities are more responsible for the development of BPD in females.

Racial Differences

As predicted, we did not find many differences by race. Childhood maltreatment was not a significant predictor of BPD diagnosis in Black participants. And despite the overall model being significant for White participants, maltreatment was not predictive of BPD diagnosis once controls for age and gender were introduced. This was also the case for BPD symptoms. A previous study using this data (Widom, Czaja, & Paris, 2009) found a relationship between childhood maltreatment and BPD diagnosis and symptoms while controlling for race. Furthermore, using data from a large epidemiological study, Tomko and colleagues (2014) found that around 70% of those with BPD identified as White, non-Hispanic compared to only 14% of individuals that identified as Black, non-Hispanic. This pattern of results lends some corroboration to the resiliency hypothesis that suggests that Black children and other children of color develop resiliency due to chronic exposure to stressful social environments and also through cultural factors that can defend against the negative impact of childhood trauma. Lending further support to this idea, one of the few racial differences we did observe was that White maltreated children were slightly over 2.5 times more likely to develop ASPD in adulthood. There was no association between childhood maltreatment and ASPD diagnosis for Black children. In contrast to the current results, Afifi et al. (2011) found that Blacks were nearly 2 times more likely than their White counterparts to meet criteria for a Cluster B personality disorder. This would be supportive of a "double jeopardy" theory that suggests the negative impact of systemic racism that Black children experience is further compounded when they experience childhood maltreatment, putting them at an even higher risk for developing severe psychopathology as adults.

When examined separately by race, both Black and White maltreated children were at increased risk of suicide, a finding that was also found in an earlier publication using this data (Widom et al., 2012; Widom and Li, 2020). This was also the case for ASPD symptoms, with childhood maltreatment associated with increased ASPD symptoms for both Black and White participants. These results lend some support to the "racial invariance hypothesis", which proports that the consequences of negative childhood experiences are comparable for difference races. For impulsivity, there was no significant association between childhood maltreatment for Blacks or Whites. However, there was a non-significant trend for the White participants, suggesting that childhood maltreatment might be predictive of higher levels of impulsivity in this population. This is not consistent with some previous studies; as several studies have found higher mean levels of impulsivity in Black children compared to Whites (Bussing et al., 2008; DuPaul et al., 1998; Pedersen, Molina, Belendiuk, & Donovan, 2012). It is important to note that studies have shown that Black children are more likely to be identified or show impulsive behaviors compared to White children (Pedersen, Molina, Belendiuk, & Donovan, 2012). It could be the case that Black children develop impulsivity when they are in high-risk and classically under-served environments. Additionally, it is possible that racism is the influential factor in other's perceptions of Black children's behavior and observers may pay more attention to impulsive Black children while excusing or overlooking these behaviors in White children. Further examination of the differences in Whites and Blacks in terms of impulsivity is warranted in order to identify and determine the potential impact of environmental factors and systemic racism.

Interactions between MAOA Genotype and Childhood Maltreatment

The model examining whether MAOA genotype moderated the relationship between childhood maltreatment and BPD diagnosis or symptoms was not significant. However, there was a significant three-way interaction (Figure 1) of MAOA x child maltreatment x race predicting BPD diagnosis. These results suggested that the 3,4 genotype was protective for Black females in relation to BPD, leading to lower rates of BPD. There was a similar trend for BPD symptoms, indicating that the 3,4 genotype was protective for Black females, although this interaction did not reach conventional levels of significance.

It is difficult to place these findings in the context of an existing literature because there are currently no studies examining the relationship between MAOA genotype and psychological problems specifically in Black females. The studies that include females (Edwards et al., 2010; Kuepper, Grant, Wielpuetz, & Hennig, 2013; Meyer-Lindenberg et al., 2006; Prom-Wormley et al., 2009; Sjoberg et al., 2007; Verhoeven et al., 2012) either do not specifically examine the impact of race or exclude Black participants all together. In the past, multicultural research has been limited to cross-cultural psychology and treated as a separate issue, despite evidence that shows that race, ethnicity, and culture have a significant impact on psychological concepts (Betancourt & Lopez, 1993; Roberts, Bareket-Shavit, Dollins, Goldie, & Mortenson, 2020). This oversight is likely due in part to ethnocentrism in American psychology (Lee, 1994; Roberts et al., 2020). Black females are arguably one of the most understudied groups in psychological science and have historically experienced the most oppression due to the intersectional combination of racism and sexism. The present study is a beginning step in understanding gene by environment interactions in Black individuals.

Contrary to expectations, there were no significant gene by environment interactions for ASPD diagnosis, ASPD symptoms, suicide attempts, or impulsivity. Although there is a sizable body of research showing that MAOA genotype moderates the relationship between childhood adversity and antisocial behavior (Caspi et al., 2002; Prom-Wormely et al., 2009; Kim-Cohen et al., 2006; Nilsson et al., 2006; Widom & Brzustowicz, 2006), other studies have not observed this association (Haberstick et al., 2014; Huzinga et al., 2006; Prichard, Mackinnon, Jorm, & Easteal, 2008; Young et al., 2006). Haberstick and colleagues (2014) used a power analysis of their sample size to ensure they had adequate power to detect the MAOA genotype moderating the relationship between childhood maltreatment and later antisocial behavior; yet they failed to observe this relationship. Some have even suggested that a positive publication bias for gene by environment studies has caused many to assume there is a robust relationship at play, when this is not actually (Duncan & Keller, 2011). Specifically, Duncan & Keller assert that studies with negative findings are subject to the file-drawer effect. Moreover, there is evidence that replications fail to support previous gene by environment studies with positive findings (Duncan & Keller, 2011). The present findings should be considered .in light of these criticisms and perhaps more effort should be given to correct publication bias and replication failures within the gene by environment literature.

Limitations and Future Directions

Despite the many advantages of the present study, several limitations need to be noted. In the present sample, the assessment of childhood maltreatment is based on court-substantiated cases of abuse and neglect, which could have resulted in more extreme cases of child abuse and neglect. It is also possible that this sample underrepresents childhood abuse and neglect in the control group as the experience of childhood maltreatment is commonly not reported to

authorities. Furthermore, childhood maltreatment cases that have court-intervention are representative of families on the lower-end of the socioeconomic spectrum and limit the ability of these results to be applied to middle-to-upper class families. Another limitation is that diagnoses were based on DSM-III-TR criteria. There are several criticisms of the DSM diagnostic system including but not limited to, the common co-occurrence of the personality disorders and the great amount of heterogeneity within personality disorder diagnoses (Skodol, 2012). For example, past studies have observed 136 different combinations of BPD symptoms within the diagnosis among psychiatric patients, which is only 51% of the possible 256 symptom combinations for BPD (Johansen, Karerud, Pedersen, Gude, & Falkum, 2004). It is possible that the lack of significant findings for BPD diagnosis for the maltreatment group and the absence of an interaction between MAOA and childhood maltreatment in predicting both BPD and ASPD diagnosis is in part due the significant heterogeneity of participants within these groups. Future studies would benefit from ensuring greater homogeneity in their diagnosis positive participants. Another potential explanation for the failure to find a significant interaction between MAOA and childhood maltreatment is that there are likely many factors that contribute to the development of BPD and ASPD that were not examined here. For example, previous research has identified other familial factors and child characteristics are possible contributors to the development of both disorders (Beauchaine et al., 2009; Stepp et al., 2016). More specifically, parental psychopathology and/or substance abuse, a family history of psychiatric hospitalization, harsh and inconsistent parenting, and low levels of parental warmth and satisfaction with their child have all been linked to increased rates of BPD (Beauchaine et al., 2009; Stepp et al., 2016). Furthermore, specific characteristics like emotional dysregulation, low IQ, impulsivity, and the presence of psychopathology in childhood and adolescence are associated with increased

presence of BPD (Stepp et al., 2016). Childhood maltreatment could be a "marker for family dysfunction" (Widom, 1998). Future studies should expand upon the current results by looking at specific familial and child characteristics and their interaction with MAOA genotype in relation the development of BPD and ASPD.

Nonetheless, the present study's results suggested unique relationships between MAOA genotype and childhood maltreatment when predicting BPD symptoms and diagnosis for Black females in particular. Black individuals are historically underrepresented in psychological science. The findings of this study showed specific results for Black females and the potential protective effect of a particular MAOA genotype for BPD diagnoses in the presence of childhood maltreatment and a similar trend for BPD symptoms. Past research has neglected the inclusion of Black females, and therefore specific hypotheses about how the MAOA genotype would function were not specified in advance in the present study. However, the present research suggests that there may be a protective element in the MAOA genotype that is specific to heterozygous Black females, which cannot be ignored given the historic oppression faced by these individuals. Future studies should focus specifically on gene by environment interactions in Black populations in order to better understand these relationships and to hopefully elucidate any interactions between MAOA genotype and negative environmental factors specific to Black individuals.

Conclusion

The results of the present study show that there is a significant relationship between childhood maltreatment and certain aspects of BPD and ASPD. Specifically, we found that childhood maltreatment was predictive of ASPD diagnosis, ASPD symptoms, lifetime history of suicide attempts, and impulsivity. Unexpectedly, we did not find a relationship between BPD

diagnosis and childhood maltreatment, although there was a relationship between childhood abuse and neglect and number of BPD symptoms. We found one significant 3-way interaction suggesting that the heterozygous MAOA genotype (3-,4-) was protective for Black females with a history of childhood maltreatment. However, due to the limited nature of MAOA genotype studies in Black females, it is difficult to put these results into context and future research is needed to better understand the impact of MAOA genotype in this population. Overall, our results underscore the significant relationship between childhood maltreatment and personality psychopathology in adulthood. Our findings also suggest that there may be other environmental and contextual factors, such as systemic racism and low SES, that are more influential in the development of these disorders in disadvantaged groups.

Table 1.

Characteristics of Included and Excluded Participants

	Included Participants (*N* = 644)	Excluded Participants (*N* = 552)			
	N (%)	N (%)	Chi square	*df*	*p value*
Maltreated	363 (56.4)	313 (56.7)	0.01	1	.91
Female	351 (54.5)	235 (42.6)	16.93	1	.000
Black, Non-Hispanic	227 (35.2)	162 (29.3)	89.65	6	.000
Borderline Personality Disorder Diagnosis	91 (14.1)	21 (8.5)	5.06	1	.02
Antisocial Personality Disorder Diagnosis	84 (13.1)	98 (17.8)	5.06	1	.02
Any Suicide Attempt	111 (17.3)	36 (14.6)	0.94	1	.33
	M (*SD*)	*M* (*SD*)	T score		
Age (in years)	39.48 (3.52)	39.48 (3.51)	0.01	890	.99
BPD Symptoms (#)	1.99 (2.05)	1.70 (1.72)	-2.15*	524.82	.03
ASPD Symptoms (#)	3.82 (3.61)	3.88 (3.96)	0.30	1193	.77
Impulsivity Score (#)	35.14 (5.91)	34.10 (5.43)	-2.40*	887	.02

Note: Numbers vary slightly due to missing information for one person for ASPD and suicide attempts; BPD = Borderline Personality Disorder; ASPD = Antisocial Personality Disorder; *M* = mean; *SD* = standard deviation; *df* = degrees of freedom

Table 2.

Overall Distribution of MAOA genotypes

Genotype	Total *N* = 644	Control *N* = 280	Maltreated *N* = 364	Chi square[a]	*p*
1	12 (1.9%)	4 (1.4%)	8 (2.2%)	11.95	0.367
2	6 (0.9%)	1 (0.4%)	5 (1.4%)		
2,3	6 (0.9%)	2 (0.7%)	4 (1.1%)		
2,4	7 (1.1%)	5 (1.8%)	2 (0.5%)		
3	165 (25.6%)	77 (27.5%)	88 (24.2%)		
3,3.5	5 (0.8%)	3 (1.1%)	2 (0.5%)		
3,4	173 (26.9%)	70 (25.0%)	103 (28.3%)		
3.5	1 (0.2%)	1 (0.4%)	0 (0.0%)		
3.5,4	4 (0.6%)	2 (0.7%)	2 (0.5%)		
4	257 (39.9%)	110 (39.3%)	147 (40.4%)		
4,5	5 (0.8%)	2 (0.7%)	3 (0.8%)		
5	3 (0.5%)	3 (1.1%)	0 (0.0%)		

[a]Degrees of freedom = 11

Table 3.

Demographic Characteristics of the Sample

	Childhood maltreatment (*N* = 336)	Controls (*N* = 256)			
	N (%)	N (%)	Chi square	*df*	*p value*
Female	188 (56.0)	132 (51.6)	1.13	1	0.29
Black	109 (32.4)	97 (37.9)	1.90	1	0.17
BPD Diagnosis	54 (16.1)	32 (12.5)	1.49	1	0.22
ASPD Diagnosis	52 (15.5)	23 (9.0)	5.60*	1	0.02
Any Suicide Attempts	80 (23.9)	27 (10.5)	17.40***	1	0.000
	M (*SD*)	*M* (*SD*)	*t* score		
Age at Interview 2	39.44 (3.52)	39.51 (3.56)	0.25	590	0.81
# of BPD Symptoms	2.16 (2.10)	1.79 (2.02)	-2.19*	590	0.03
# of ASPD Symptoms	4.27 (3.78)	3.11 (2.96)	-4.06***	588.59	0.000
Impulsivity Score	35.62 (5.79)	34.58 (6.10)	-2.11*	589	0.04

Note: Numbers vary slightly due to missing information for one person for ASPD and suicide attempts; BPD = Borderline Personality Disorder; ASPD = Antisocial Personality Disorder; *M* = mean; *SD* = standard deviation; *df* = degrees of freedom

Table 4.

Childhood Maltreatment and Borderline Personality Disorder Diagnosis and Symptoms for the Overall Sample and Separately for Males and Females and Blacks and Whites

	Borderline Personality Disorder Diagnosis				Number of BPD Criteria			
Group	*N*	%	AOR	95%CI	Beta	SE	95% CI	*p*
Overall (*n* = 592)								
Child maltreatment	54	16.1	1.44	0.83-2.15	0.09	0.17	0.05-0.72	.03
Control	32	12.5						
Female (*n* = 320)								
Child maltreatment	31	16.5	1.55	0.65-2.16	0.06	0.24	-0.21-0.74	.06
Control	19	14.4						
Male (*n* = 272)								
Child maltreatment	23	15.5	1.58	0.75-3.33	0.13	0.24	0.04-0.99	.03
Control	13	10.5						
Black (*n* = 206)								
Child maltreatment	18	16.5	1.19	0.55-2.57	0.12	0.28	-0.09-1.02	.10
Control	14	14.4						
White (*n* = 386)								
Child maltreatment	36	15.9	1.50	0.81-2.77	0.08	0.22	-0.08-0.77	.11
Control	18	11.3						

Note. BPD = Borderline Personality Disorder; AOR = adjusted odds ratio, controlling for age, sex, and race or sex or race where appropriate; CI = confidence interval; *M* = mean; SE = standard error; SD = standard deviation.

Table 5.

Childhood Maltreatment and Antisocial Personality Disorder Diagnosis and Symptoms for the Overall Sample and Separately for Males and Females and Blacks and Whites

	Antisocial Personality Disorder Diagnosis				Number of ASPD Criteria			
Group	*N*	%	AOR	95%CI	Beta	SE	95% CI	*p*
Overall (*n* = 591)								
Child maltreatment	52	15.5	1.98**	1.17-3.36	0.11	0.03	0.02-0.13	.01
Control	23	9.0						
Female (*n* = 320)								
Child maltreatment	19	10.1	1.39	0.62-3.11	0.19	0.34	0.49-1.84	.001
Control	10	7.6						
Male (*n* = 271)								
Child maltreatment	33	22.4	2.51**	1.25-5.03	0.19	0.45	0.51-2.29	.002
Control	13	10.5						
Black (*n* = 206)								
Child maltreatment	17	15.6	1.47	0.65-3.32	0.15	0.50	0.14-2.10	.03
Control	12	12.4						
White (*n* = 385)								
Child maltreatment	35	15.5	2.64**	1.28-5.41	0.20	0.34	0.72-2.04	.000
Control	11	6.9						

Note. ASPD = Antisocial Personality Disorder; AOR = adjusted odds ratio; CI = confidence interval; *M* = mean; *SD* = standard deviation; *SE* = standard error.
**$p \leq .01$

Table 6.

Childhood Maltreatment and Suicide Attempts and Impulsivity for the Overall Sample and Separately by Sex and Race

	Ever Suicide Attempts				Impulsivity			
Group	*N*	%	AOR	95%CI	Beta	SE	95% CI	*p*
Overall (*n* = 591)								
Child maltreatment	80	23.9	2.60***	1.61-4.18	0.08	0.49	0.02-1.95	.05
Control	27	10.5						
Female (*n* = 319)								
Child maltreatment	56	29.9	2.87***	1.58-5.22	0.03	0.69	-1.00-1.70	.61
Control	17	12.9						
Male (*n* = 272)								
Child maltreatment	24	16.2	2.12	0.97-4.68	0.14	0.70	0.31-3.06	.02
Control	10	8.1						
Black (*n* = 206)								
Child maltreatment	23	21.1	2.47*	1.07-5.70	0.07	0.82	-0.78-2.47	.31
Control	9	9.3						
White (*n* = 385)								
Child maltreatment	57	25.2	2.63***	1.48-4.70	0.09	0.62	-0.18-2.24	.09
Control	18	11.3						

Note. AOR = adjusted odds ratio; CI = confidence interval; *M* = mean; *SD* = standard deviation; *SE* = standard error.
*$p \leq .05$; ***$p \leq .001$.

Table 7.

Three-Way Interactions of Childhood Maltreatment, MAOA Genotype, and Borderline Personality Disorder Diagnosis and Number of Symptoms

	BPD diagnosis			BPD Symptoms			
	AOR	95% CI	*p*	Beta	SE	95% CI	*p*
Childhood Maltreatment	1.44	0.71-2.93	0.31	0.07	0.26	-0.22-0.80	0.26
Female	1.13	0.63-2.02	0.69	0.01	0.21	-0.39-0.44	0.91
Black	1.47	0.82-2.63	0.19	0.08	0.22	-0.07-0.78	0.10
Age	0.93	0.87-0.99	0.03	-0.10	0.02	-0.10- -0.01	0.02
DC1	0.67	0.26-1.76	0.42	-0.02	0.31	-0.67-0.53	0.83
DC2	1.06	0.41-2.76	0.91	0.02	0.35	-0.61-0.76	0.83
DC1*Childhood Maltreatment	0.85	0.22-3.36	0.82	-0.03	0.44	-1.03-0.71	0.72
DC2*Childhood Maltreatment	1.36	0.42-4.39	0.61	0.10	0.45	-0.34-1.43	0.23
DC1*Childhood Maltreatment*Race	1.50	0.36-6.28	0.58	0.08	0.54	-0.28-1.82	0.15
DC2*Childhood Maltreatment*Race	0.19	0.04-0.79	0.02	-0.10	0.47	-1.74-0.10	0.08

Note. DC1 = dummy code of genotype (3,3 or 3 = 1, others = 0); DC2 = dummy code of genotype (heterozygous females = 1, others = 0); BPD = Borderline Personality Disorder; AOR= adjusted odds ratio; CI = confidence intervals; M = mean; SD = standard deviation; Beta = adjusted Beta; SE = standard error. Adjusted analyses control for age, sex, and race.

Table 8.

Three-Way Interactions of Childhood Maltreatment, MAOA Genotype, and Antisocial Personality Disorder Diagnosis and Number of Symptoms

	ASPD diagnosis			ASPD Symptoms			
	AOR	95% CI	*p*	Beta	SE	95% CI	*p*
Childhood Maltreatment	1.67	0.76-3.66	0.20	0.20	0.42	-0.22-0.80	0.001
Female	0.43	0.22-0.86	0.02	-0.29	0.35	-0.40-0.44	0.000
Black	1.51	0.79-2.89	0.21	0.04	0.35	-0.07-0.78	0.40
Age	1.00	0.93-1.07	0.97	0.01	0.04	-0.10- -0.01	0.78
DC1	0.78	0.28-2.23	0.65	-0.07	0.50	-0.67-0.53	0.27
DC2	1.53	0.47-5.05	0.48	0.06	0.57	-0.61-0.76	0.44
DC1*Childhood Maltreatment	2.21	0.59-8.29	0.24	-0.04	0.72	-1.03-0.71	0.58
DC2*Childhood Maltreatment	0.97	0.23-4.04	0.96	-0.04	0.74	-0.34-1.43	0.59
DC1*Childhood Maltreatment*Race	0.88	0.24-3.24	0.85	0.05	0.87	-0.28-1.82	0.30
DC2*Childhood Maltreatment*Race	0.57	0.13-2.44	0.45	-0.01	0.76	-1.74-0.10	0.88

Note. DC1 = dummy code of genotype (3,3 or 3 = 1, others = 0); DC2 = dummy code of genotype (heterozygous females = 1, others = 0); ASPD = Antisocial Personality Disorder; AOR= adjusted odds ratio; CI = confidence intervals; M = mean; SD = standard deviation; Beta = adjusted Beta; SE = standard error. Adjusted analyses control for age, sex, and race.

Table 9.

Three-Way Interactions of Childhood Maltreatment, MAOA Genotype, and Suicide Attempts and Impulsivity

	Suicide Attempts			Impulsivity			
	AOR	95% CI	*p*	Beta	SE	95% CI	*p*
Childhood Maltreatment	1.79	0.88-3.65	0.11	0.04	0.75	-0.93-2.00	0.47
Female	1.93	1.11-3.36	0.02	0.06	0.61	-0.49-1.90	0.25
Black	0.80	0.43-1.50	0.49	-0.06	0.62	-1.90-0.53	0.27
Age	0.97	0.92-1.03	0.34	-0.11	0.07	-0.32- -0.05	0.009
DC1	0.50	0.17-1.48	0.21	-0.14	0.87	-3.61- -0.18	0.03
DC2	0.73	0.28-1.94	0.53	-0.13	1.00	-3.66-0.27	0.09
DC1*Childhood Maltreatment	2.00	0.52-7.65	0.31	0.10	1.28	-0.84-4.17	0.19
DC2*Childhood Maltreatment	2.01	0.63-6.36	0.24	-0.01	1.30	-2.62-2.47	0.95
DC1*Childhood Maltreatment*Race	1.07	0.28-4.08	0.92	-0.01	1.54	-3.28-2.77	0.87
DC2*Childhood Maltreatment*Race	0.72	0.25-2.13	0.56	0.00	1.34	-2.63-2.64	1.00

Note. DC1 = dummy code of genotype (3,3 or 3 = 1, others = 0); DC2 = dummy code of genotype (heterozygous females = 1, others = 0); AOR= adjusted odds ratio; CI = confidence intervals; M = mean; SD = standard deviation; Beta = adjusted Beta; SE = standard error. Adjusted analyses control for age, sex, and race.

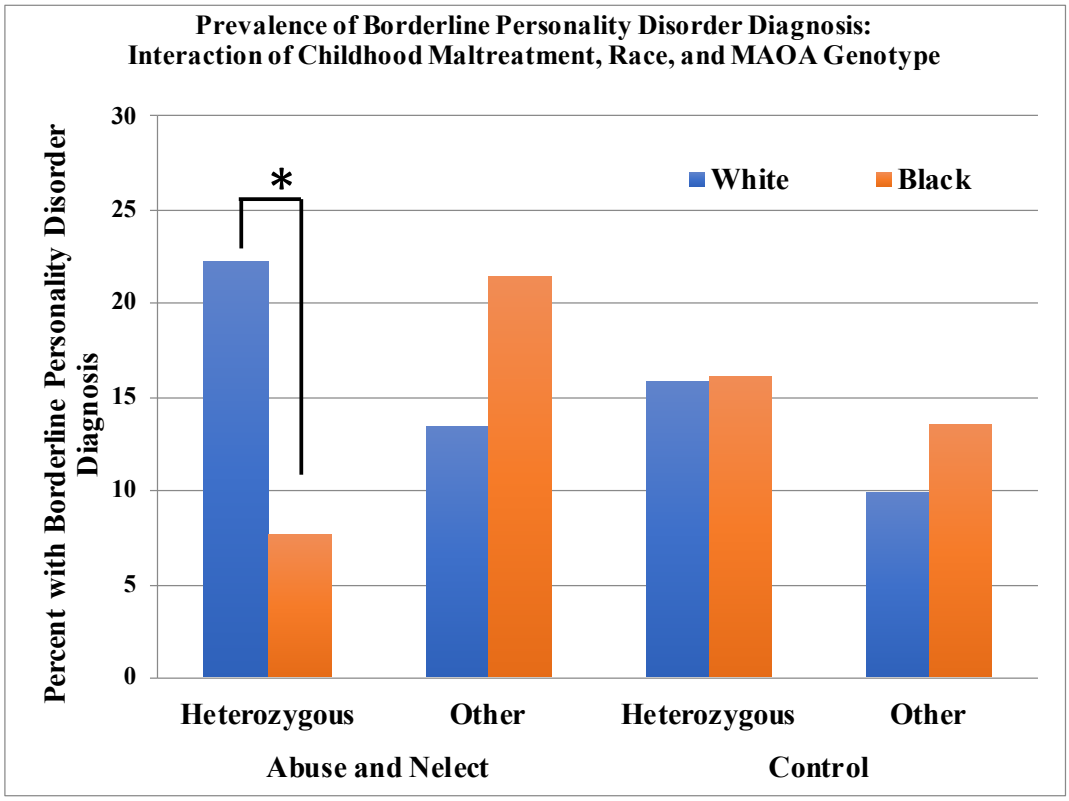

Figure 1. Significant three-way interaction of child maltreatment by race by MAOA for Borderline Personality Disorder (BPD) Diagnosis (AOR = 0.19, 95%CI 0.04-0.79, p = .02) showing that maltreated Black females with the heterozygous MAOA genotype (3,4) show decreased rates of BPD disorder diagnoses compared to all other maltreated individuals.

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