Journal of Abnormal Psychology
2013, Vol. 122, No. 1, 167–179

0021-843X/13/$12.00 DOI: 10.1037/a0029866

Analysis of Monoaminergic Genes, Childhood Abuse, and Dimensions of Psychopathy

Naomi Sadeh
University of Illinois at Urbana–Champaign and University of California at San Francisco

Shabnam Javdani
University of Illinois at Urbana–Champaign and New York University

Edelyn Verona
University of Illinois at Urbana–Champaign

Psychopathy is a multidimensional construct characterized by an interpersonally manipulative and emotionally detached personality profile that differentiates it from other antisocial syndromes. Previous research with youth has linked the long allele of the serotonin transporter gene in the presence of environmental stress with the interpersonal and affective traits of psychopathy, but these relationships have yet to be examined in relation to adult psychopathy. Consequently, we examined how serotonin transporter (5-HTTLPR) polymorphisms, monoamine oxidase-A (MAO-A) variants, and childhood abuse measured with the Childhood Trauma Questionnaire relate to dimensions of psychopathy in a forensic sample of 237 men with elevated levels of environmental adversity. We found that the emotional deficits characterizing the affective factor of psychopathy, as measured by the Psychopathy Checklist: Screening Version, were highest among carriers of the 5-HTT long allele. Furthermore, the impulsive and irresponsible lifestyle features of psychopathy were higher among low-activity than high-activity MAO-A carriers. These genetic effects were unexpectedly not moderated by a history of childhood abuse. Results provide evidence on the molecular genetics correlates of psychopathic traits in adulthood, relationships that should be investigated further in future research.

Keywords: psychopathy, 5-HTT, MAO-A, genes, abuse

Decades of research suggest that important motivational and temperamental distinctions exist across individuals who engage in criminal behavior, a consideration that existing research on the molecular genetics correlates of antisocial behavior has not yet addressed. Psychopathic individuals, like other types of offenders, display impulsive, irresponsible, and aggressive behavior (Hare, 1991; Hart, Cox, & Hare, 1995). However, their seemingly random and often purposeless acts of violence and crime are undergirded by a constellation of interpersonally manipulative and emotionally detached traits that differentiate them from other criminals (e.g., Hare, 1991).

Indeed, although originally conceptualized as a unitary construct (Cleckley, 1941), empirical work supports the conceptualization of psychopathy as a combination of trait factors that vary dimensionally across individuals. Factor analysis of psychopathy instruments suggest that at least two primary trait dimensions undergird the disorder (Harpur, Hare, & Hakstian, 1989), specifically the interpersonal–affective factor that measures the tendency to be socially dominant, deceitful, unemotional, and remorseless, and the more typical impulsive–antisocial factor that indexes an impulsive, irresponsible, aggressive, and antisocial social lifestyle (e.g., Psychopathy Checklist—Revised Factors 1 and 2, respectively; Hare, 1991). There is also strong support for three- and four-factor models of psychopathy with the interpersonal, affective, impulsive lifestyle, and antisocial features of the disorder separating into distinct factors (Cooke & Michie, 2001; Cooke, Michie, & Hart, 2004; Hill, Neumann, & Rogers, 2004; Vitacco, Neumann, & Jackson, 2005).

Research suggests that the psychopathy dimensions index separable etiological pathways to criminal and violent behavior in that they are characterized by distinct biological, psychological, and environmental risk factors (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Fowles & Dindo, 2006; Hall, Benning, & Patrick, 2004; Harpur et al., 1989; Patrick & Bernat, 2009; Ross, Benning, Patrick, Thompson, & Thurston, 2009; Verona, Patrick, & Joiner, 2001). For instance, several studies have found that the unique variance of the interpersonal–affective factor is related to low levels of anxiety and fear, decreased physiological reactivity to threatening cues, and resilience against mood disorders (e.g., Benning et al., 2003; Harpur et al., 1989; Patrick, Bradley, & Lang,

This article was published Online First September 17, 2012.

Naomi Sadeh, Department of Psychology, University of Illinois at Urbana–Champaign and Department of Psychiatry, University of California at San Francisco; Shabnam Javdani, Department of Psychology, University of Illinois at Urbana–Champaign and Department of Applied Psychology, New York University; Edelyn Verona, Department of Psychology, University of Illinois at Urbana–Champaign.

National Institute of Mental Health Grant F31MH086178 supported Naomi Sadeh. University of Illinois Arnold O. Beckman Research Award supported this research. Thanks to Joshua Jackson, Jenessa Sprague, Sima Finy, and Champaign County Jails for help with data collection and processing.

Correspondence concerning this article should be addressed to Naomi Sadeh, 401 Parnassus Avenue, San Francisco, CA 94143. E-mail: naomisadeh@gmail.com

1993; Ross et al., 2009; Vaidyanathan, Hall, Patrick, & Bernat, 2011). In contrast, the impulsive–antisocial dimension is related to high levels of anxiety and distress, a range of comorbid psychopathology (e.g., major depressive disorder, posttraumatic stress disorder, substance dependence), and elevated reactivity to emotional stimuli (Benning et al., 2003; Harpur et al., 1989; Reardon, Lang, & Patrick, 2002; Smith & Newman, 1990; Verona et al., 2001; Verona, Sprague, & Sadeh, 2012). Thus, the interpersonal–affective factor appears to be characterized by low emotional reactivity and blunted affective bonding, whereas the impulsive–antisocial dimension shares the emotional and behavioral dysregulation that characterizes other forms of externalizing psychopathology (Krueger et al., 2002).

The differential correlates of the more recent three- and four-factor models are only beginning to emerge in adult samples. Preliminary evidence suggests that the interpersonal factor differs from the affective factor in that the former is characterized by social dominance, grandiose narcissism, positive emotionality, and achievement, whereas the latter is characterized by low social affiliation, emotional detachment, and reduced reactivity to affective stimuli (Hall et al., 2004; Schoenleber, Sadeh, & Verona, 2011; Verona, Patrick, Curtin, Bradley, & Lang, 2004). Based on these findings, low emotional reactivity and blunted affective bonding in psychopathy appears to be more strongly related to the affective rather than the interpersonal factor. Research comparing the impulsive lifestyle factor and the antisocial factor has found that the latter factor is more strongly related to instrumental aggression and violence than the former factor (Walsh, Swogger, & Kosson, 2009). Similarly, inclusion of antisocial behavior in the four-factor model improves prediction of aggression and violent recidivism compared with the three-factor model (e.g., Vitacco et al., 2005; Walters & Heilbrun, 2010).

The aim of the present study was to extend previous epidemiological molecular genetics research on antisocial behavior by examining whether psychopathic personality traits in particular evidence distinct genetic relationships among individuals who engage in antisocial behavior. Thus, we limited the scope of this investigation to examining the genetic correlates of the two-factor and three-factor models of psychopathy that measure psychopathic personality traits, although we recognize that research supports the relevance of the antisocial behavior factor for the construct of psychopathy (Hill et al., 2004; Vitacco et al., 2005).

Genetic Risk for Psychopathy

Empirical work has only recently begun to examine molecular genetics associations with psychopathy, although preliminary research has documented serotonin deficiencies among individuals with related forms of psychopathology, including antisocial, aggressive, and impulsive behavior (e.g., Carver & Miller, 2006). Research suggests serotonin (5-hydroxyindoleacetic acid [5-HT]) transmission is an important contributor to individual differences in vulnerability to environmental stress and negative emotionality, which has made it a popular mechanism to study in relation to the development of psychopathology (e.g., Canli & Lesch, 2007; Caspi, Hariri, Holmes, Uher, & Moffitt, 2010). Soderstrom and colleagues (Soderstrom, Blennow, Manhem, & Forsman, 2001; Soderstrom, Blennow, Sjodin, & Forsman, 2003) were the first to examine the relationship between cerebrospinal fluid concentrations of a serotonin metabolite and psychopathic traits in violent adult offenders. Results of these studies found that psychopathic traits were positively associated with the serotonin metabolite 5-HIAA and negatively associated with the dopamine metabolite HVA, suggesting impaired serotonin regulation in psychopathy. It is important to note that evidence has also emerged to suggest that serotonergic functioning relates differentially to the interpersonal, affective, and impulsive lifestyle dimensions of psychopathy. For instance, Dolan and Anderson (2003) documented an inverse association between serotonergic functioning and the impulsive dimension, but a positive association between serotonin functioning and the arrogant and deceitful interpersonal dimension in a sample of violent adult offenders (the association of the deficient affective experience dimension with serotonin functioning was nonsignificant). These opposing relationships suggest that the impulsive lifestyle factor may index vulnerability to stress, whereas the interpersonal factor may measure more adaptive stress reactivity. However, only these three studies have examined serotonin functioning in relation to psychopathic traits, making the results preliminary and in need of replication before strong conclusions can be drawn.

Over the past decade, molecular genetics research has emerged to suggest that allelic variants of the serotonin transporter gene may increase risk for the development of psychopathology, particularly in the context of environmental adversity (e.g., Anguelova, Benkelfat, & Turecki, 2003; Karg, Burmeister, Shedden, & Sen, 2011; Lesch et al., 1996) Although these findings were originally met with much enthusiasm and spurred a plethora of research, difficulties replicating genetic relationships with psychological outcomes (Duncan & Keller, 2011) and discrepant findings at the meta-analytic level (e.g., Karg et al., 2011; Risch et al., 2009) have resulted in controversy and skepticism regarding the usefulness of examining genetic risk for psychological disorders especially in the way candidate gene studies are typically conducted in the psychology literature (Munafo & Flint, 2011). For instance, a widely cited meta-analysis of 14 studies conducted by Risch and colleagues (2009) reported no effect of 5-HTT genotype on risk for depression in the context of stressful life events, whereas analysis of 54 studies published by Karg and colleagues (2011) did find evidence of a relationship between the 5-HTT short allele and risk for depression in the context of stress when studies operationalized stress as childhood maltreatment, a medical condition, or stressful life events. Thus, more research is needed to help clarify relationships of genotypes with psychological outcomes in the psychopathology literature.

One widely studied genetic variant is a repeat in the promoter region of the *SLC6A4* (5-HTTLPR) that regulates the expression and function of the serotonin transporter protein (5-HTT), which is involved in reuptake of serotonin from the synaptic cleft (Lesch et al., 1996). The most common variation in the promoter region consists of a 44-base-pair insertion (long allele) or deletion (short allele), although other variations are possible (e.g., Gelernter, Kranzler, & Cubells, 1997). Cells with the long allele produce higher concentrations of 5-HT transporter mRNA than do cells with the short allele, which is hypothesized to lead to a more rapid clearance of 5-HT from the synaptic cleft (Greenberg et al., 1999). As reviewed by Hariri and Holmes (2006), "5-HTT plays an essential role in determining the duration and intensity of the 5-HT communication with its receptors on postsynaptic targets such as

those located in limbic regions mediating emotion, and with presynaptic receptors that exert inhibitory control over the 5-HT neuron itself" (p. 183).

The short allele in combination with environmental adversity has been associated with a range of psychopathology outcomes characterized by high levels of negative emotionality and stress, including anxiety, depression, suicide, aggression, and impulsivity (e.g., Beitchman et al., 2006; Carver, Johnson, Joorman, Kim, & Nam, 2011; Caspi et al., 2003; Haberstick, Smolen, & Hewitt, 2006; Kendler, Kuhn, Vittum, Prescott, & Rile, 2005; Lesch et al., 1996; Verona, Joiner, Johnson, & Bender, 2006). However, a number of studies have failed to detect an association between the short allele and psychopathology, including externalizing traits (e.g., Douglas et al., 2011; Risch et al., 2009; Wang et al., 2012). The short relative to the long allele has also been associated with increased amygdala activation to threatening or unpleasant stimuli according to a meta-analysis of 14 published and three unpublished neuroimaging studies (Munafo, Brown, & Hariri, 2008), suggesting that the former variant is associated with enhanced reactivity to motivationally salient stimuli. These findings may indicate that short-allele carriers experience enhanced reactivity to early life adversity (Caspi et al., 2010), which is consistent with preliminary evidence of increased hypothalamic–pituitary–adrenal reactivity in the short- compared with long-allele carriers (Gotlib, Joorman, Minor, & Hallmayer, 2008).

Expanding on this literature, research has also begun to explore relationships between the serotonin transporter gene, environmental adversity, and psychopathic traits (Glenn, 2011; Sadeh et al., 2010). Sadeh and colleagues (2010) recently investigated whether dimensions of psychopathic tendencies in youth evidence distinct relationships with the 5-HTT genotype based on the posited relationship between the short allele and heightened vulnerability to environmental stress and negative emotionality. Specifically, they investigated whether the 5-HTT short and long alleles relate differentially to the interpersonal, affective, and impulsive behavior factors in youth as a function of socioeconomic status (SES). First, Sadeh et al. found that short-allele carriers showed elevated levels of impulsivity relative to long-allele carriers, regardless of SES. Second, results from two independent samples revealed a specific link between the 5-HTT *long* allele and both the interpersonal and affective factors in youth. A gene–environment interaction emerged, whereby both a narcissistic interpersonal style and callous or unemotional traits increased as SES decreased only among youth who carried the 5-HTT long allele. Although the association between the 5-HTT long allele and psychopathic tendencies in youth replicated in two independent samples, this study was the first to find this association and it requires replication, especially given the problematic nonreplication that abounds in the candidate gene–psychopathology literature.

The association between the long allele and psychopathic features has been emphasized as a key relationship to pursue in future research on the genetics of psychopathy (Glenn, 2011). Furthermore, a recent study (Herman et al., 2011) partially replicated an association between the long allele and psychopathic-like traits in a sample of adults diagnosed with alcohol dependence using the California Psychological Inventory Socialization Scale, a personality measure that correlates −.31 with the total score on the Psychopathy Checklist (Harpur et al., 1989). Specifically, male long-allele carriers reported lower socialization than male short-allele carriers, although the opposite pattern emerged for women (Herman et al., 2011), which again underscores the need for clarity and replication of findings. The association of the 5-HTT long allele with interpersonal–affective traits in youth and socialization scores in adults is consistent with research showing that homozygous long-allele carriers demonstrate lower average resting levels of amygdala activation (Canli et al., 2005) and reduced hypothalamic–pituitary–adrenal axis activity (Gotlib et al., 2008) than those with the long/short or short/short variants. The preliminary nature of these findings underscores the importance of conducting additional studies to further explicate potential relationships between the 5-HTT long allele and deficits in emotional susceptibility that may confer risk for affective and interpersonal psychopathic traits, particularly in persons with histories of environmental adversity.

A second genotype of potential interest in relation to psychopathy stems from research implicating the interaction of the low-activity monoamine oxidase-A (MAO-A) variant and childhood adversity in the manifestation of antisocial and aggressive behavior (Caspi et al., 2002; Foley et al., 2004; Kim-Cohen et al., 2006; Nilsson et al., 2006; Taylor & Kim-Cohen, 2007; Widom & Brzustowicz, 2006). Again, this finding has not been consistently replicated (e.g., Prichard, Mackinnon, Jorm, & Easteal, 2008; Young et al., 2006) and thus requires further study. MAO metabolizes several key neurotransmitters including serotonin, norepinephrine, and dopamine (Shih & Thompson, 1999). A functional 30-bp repeat polymorphism in the promoter region of the MAO-A gene, linked to the X chromosome, encodes expression of the MAO-A enzyme, resulting in at least six allele variants (2, 3, 3.5, 4, 5, or 6 repeat copies) that differ in terms of transcriptional efficiency (Sabol, Hu, & Hamer, 1998; Shih, Chen, & Ridd, 1999; Shih & Thompson, 1999). In comparison to the high-activity variant (≥3.5 repeat copies), the low-activity variant has been associated with increased anxiety, childhood conduct disorder, violent behavior, and Cluster B personality traits (e.g., Caspi et al., 2002; Foley et al., 2004; Jacob et al., 2005; Kim-Cohen et al., 2006). Neuroimaging research suggests that the low-activity variant is associated with exaggerated activity in a circuit of brain regions involved in emotion regulation that include the amygdala, rostral cingulate, and medial prefrontal cortex (for a review, see Buckholtz & Meyer-Lindenberg, 2008), which may make low-activity carriers more susceptible to the stressful effects of environmental stressors such as early adversity.

One study to date has linked the low-activity MAO-A variant to psychopathy in particular. Specifically, carriers of the low-activity variant showed elevated levels of psychopathic traits (total psychopathy and affective factor) in a sample of youth diagnosed with attention deficit–hyperactivity disorder (ADHD; Fowler et al., 2009). Although promising, these findings are in need of further replication, given the limited generalizability of the results to youth with comorbid ADHD.

In summary, existing research suggests that the long and short alleles of the serotonin transporter gene may evidence differential associations with the psychopathy factors. However, this hypothesis requires much more study before it can be confirmed, plus it has yet to be investigated in relation to adult

psychopathy. In addition, the unique variance associated with the interpersonal, affective, and impulsive lifestyle dimensions in terms of genotype has not been examined in youth or adults, leaving open the possibility that redundancy among the scales is influencing the relationship of these factors with the candidate genotypes. Furthermore, the MAO-A low-activity variant has at times been associated with aggressive and antisocial outcomes in the context of early environmental adversity, making it a reasonable candidate gene for psychopathy, particularly in relation to the impulsive lifestyle and antisocial factors. To date, the relationship of MAO-A genotype with the manifestation of psychopathic traits in adulthood has not been investigated.

Childhood Abuse

Research increasingly shows that the effects of genes on psychopathology are moderated by environmental experiences early in life (e.g., Caspi et al., 2010; Moffitt, 2005). For instance, a history of childhood maltreatment has been implicated in antisocial and aggressive outcomes across a number of studies (e.g., Jaffee, Caspi, Moffitt, & Taylor, 2004; Lahey, Moffitt, & Caspi, 2003; Margolin & Gordis, 2000), including as an environmental stressor that interacts with genetic risk to predict antisocial behavior (e.g., Caspi et al., 2002; Frazzetto et al., 2007; Kim-Cohen et al., 2006; Widom & Brzustowicz, 2006). In addition to antisocial behavior more generally, research has linked maltreatment in childhood to psychopathy in particular (Lang, Klinteberg, & Alm, 2002; Marshall & Cooke, 1999; Weiler & Widom, 1996), specifically in relation to the impulsive lifestyle factor (Poythress, Skeem, & Lilienfeld, 2006). Thus, research suggests that early experience of childhood abuse is a stressor that increases risk for the development of psychopathic traits.

Caspi and Moffitt (2006) argued that an ideal candidate environmental risk factor must have a truly environmentally mediated effect that cannot be accounted for by gene–environment correlations. Nelson et al. (2002) reported that history of childhood sexual abuse independently predicted adolescent conduct problems even when controlling for family background (conflict, divorce, neglect) and parental alcohol-related problems and fighting behaviors. Jaffee and colleagues (2004) examined the effects of physical abuse and parental antisocial behavior on children's conduct problems in a longitudinal twin study of 5- to 7-year-old children. Physical abuse prospectively predicted children's antisocial behavior problems even after statistically adjusting for early (before age 5) behavior problems, parental antisocial behavior, and genetic influences on antisocial behavior calculated from the twin study methodology. Although this research suggests that childhood abuse is associated with antisocial outcomes beyond the effects of genes or general family background, it is very difficult to partition genetic from environmental effects with the measurement of family-level variables, such as childhood abuse (Turkheimer, D'Onofrio, Maes, & Eaves, 2005). Although it will take more study before it is clear whether the effects of abuse are fully environmentally mediated, childhood abuse history, even when assessed retrospectively, seems to be important in understanding antisocial behavior. Thus, history of childhood abuse was examined as a potential moderator of genetic effects in the present study, given preliminary research showing that it interacts with MAO-A genotype to confer risk for antisocial behavior (e.g., Caspi et al., 2002) and 5-HTT genotype to confer risk for psychopathology more broadly (e.g., Karg et al., 2011).

Present Study

Given the paucity of molecular genetics research on adult psychopathy, the first aim of the study was to examine whether the 5-HTT and MAO-A genotypes are associated with the primary trait dimensions from the two-factor model of psychopathy in men. Based on research supporting a three-factor structure, our second aim was to examine whether differential genetic correlates emerge when the interpersonal and affective factors are examined separately. We selected a clinical-forensic sample with elevated levels of antisocial and psychopathic traits that is heavily characterized by a history of environmental stress (e.g., economic disadvantage, trauma experiences) and examined childhood abuse as a potential moderator of the genetic effects.

Two-Factor Model

We first hypothesized a relationship between the 5-HTT long allele and scores on the interpersonal–affective factor. Specifically, our hypothesis was specific to the contrast between long/long carriers and short/short carriers, based on research conducted with youth (Sadeh et al., 2010), as well as theoretical links between the long allele and reduced stress reactivity (Canli et al., 2005; Gotlib et al., 2008). In contrast, we expected scores on the impulsive–antisocial factor to be higher in short/short carriers than long/long carriers and in carriers of the MAO-A low-activity than high-activity variant based on the extant literature on genetic correlates of impulsivity, antisocial behavior, and conduct problems (e.g., Beitchman et al., 2006; Caspi et al., 2002; Sadeh et al., 2010). We did not expect differences between the 5-HTT heterozygous carriers and the two 5-HTT homozygous groups in relation to psychopathic traits, given the results of previous studies (Herman et al., 2011; Sadeh et al., 2010).

Three-Factor Model

We expected the association of the 5-HTT long allele to be stronger with the affective than interpersonal factor, given that the former indexes the emotional deficits of psychopathy (Cooke & Michie, 2001; Verona et al., 2004). Scores on the lifestyle factor were expected to be higher for 5-HTT short/short carriers than long/long carriers and in the MAO-A low-activity than high-activity variant.

We expected the genetic effects specified above to be enhanced by experiences of psychosocial adversity during development, which we measured in the present study as a combination of childhood physical and sexual abuse reported retrospectively by participants.

Method

Participants

To test the present hypotheses, we purposefully selected a sample of individuals with high base rates of antisociality and

psychopathic traits that also had high levels of psychosocial adversity, including economic disadvantage, childhood abuse, and life stress. Our sample consisted of 237 adult men age 18–61 years (M = 30.9 years, SD = 9.2). The final sample reported at least one instance of criminal behavior, including theft (73%), robbery (28%), drug offenses (82%), assault (72%), murder/attempted murder (11%), and fraud (28%). Participants were characterized by low SES (51% reported an annual income level below $15,000 and 100% reported an annual income below $45,000). Income was unavailable for 10 participants. More than half of participants self-identified as African American (62.4%), followed by European American (29.1%), mixed ethnicity/other (5.5%), and Hispanic (3.0%).

Participants were recruited from probation/parole agencies, county jail, and mandated treatment centers, as well as via newspaper advertisements targeting individuals with legal convictions. Individuals with a lifetime diagnosis of a psychotic (non–substance-induced) or bipolar disorder, determined using the Structured Clinical Interview for *DSM–IV–TR* (First, Spitzer, Gibbon, & Williams, 2002), were ineligible to participate because the acute effects of these disorders can artificially inflate scores on measures of psychopathy (e.g., engagement in antisocial behavior during a manic episode). Individuals with a pervasive developmental disorder (i.e., autism) or mental retardation were also ineligible to participate out of concern about their ability to provide informed consent and understand questions in the clinical interview.

Measures

The Psychopathy Checklist: Screening Version (PCL: SV; Hart et al., 1995) is a 12-item clinician-rated measure designed to index psychopathic traits. In the present study, data collected over a 2- to 3-hr assessment that included a life history interview, a structured diagnostic interview, and a review of public criminal records were used to rate participants on the dimensions of psychopathy. The screening version of the Psychopathy Checklist—Revised (Hare, 1991) was used to shorten the length of the testing protocol based on research that indicates that the PCL: SV maintains the same psychometric properties as the full-version Psychopathy Checklist—Revised (Cooke, Michie, Hart, & Hare, 1999; Guy & Douglas, 2006). The two-factor model of psychopathy (Harpur et al., 1989) consists of two intercorrelated dimensions: Factor 1 (Interpersonal–Affective) is a summed composite of superficial charm, grandiosity, deceitfulness, lack of remorse, shallow affect, and failure to accept responsibility; Factor 2 (Impulsive–Antisocial) is a summed composite of impulsivity, poor behavioral controls, lack of goals, irresponsibility, adolescent antisocial behavior, and adult antisocial behavior. The three-factor model of psychopathy (Cooke & Michie, 2001) is composed of the factors Arrogant and Deceitful Interpersonal Style (superficial charm, grandiosity, deceitfulness), Deficient Affective Experience (lack of remorse, shallow affect, failure to accept responsibility), and Impulsive and Irresponsible Behavioral Style (impulsivity, lacks goals, irresponsibility).

Interrater reliability was randomly evaluated in 19% of interviews, resulting in the following intraclass correlations: Interpersonal–Affective factor = .96, Impulsive–Antisocial factor = .95, Interpersonal factor = .95, Affective factor = .93, Lifestyle factor = .85. Specifically, trained independent raters who observed or listened to audio recordings of the interviews provided secondary ratings of the PCL: SV items. We examined the data for outliers and nonlinear distributions to ensure that the results were not an artifact of sampling; these measures were not excessively skewed or kurtotic (these values ranged from −.65 to .45).

The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003) is a 28-item self-report measure that was used to index history of childhood abuse. Each participant rated the extent to which he experienced various forms of maltreatment as a child ranging from 1 (*never true*) to 5 (*always true*). In the present study, the Physical Abuse (Cronbach's alpha = .88) and Sexual Abuse (Cronbach's alpha = .94) subscales were summed to create a composite Childhood Abuse score, which was normalized using a Blom transformation to reduce skewness (final skew < .07; Prom-Wormley et al., 2009). We also transformed this variable using log10 and square root transforms to ensure results were not due to variable distributions. On this measure, scores on the Physical Abuse and Sexual Abuse subscales spanned the full range of possible scores (5–25), with participant scores averaging 8.9 (SD = 4.3) and 6.3 (SD = 3.4), respectively. Only 25% of participants marked "never true" in response to 10 questions about childhood physical or sexual abuse, which suggests that, unlike most population-based studies, the majority of the sample experienced some form of childhood abuse. The 28-item CTQ demonstrates convergent validity with independent ratings of maltreatment provided by primary therapists about adolescent psychiatric patients following discharge from the hospital (e.g., intercorrelations were .59 for physical abuse and .75 for sexual abuse; Bernstein et al., 2003). The CTQ is also characterized by high agreement with retrospective adult interview (Lobbestael, Arntz, Harkema-Schouten, & Bernstein, 2009) and computer-assisted assessments of child abuse (e.g., agreement was 92% for sexual abuse and 80% for physical abuse; DiLillo et al., 2006) and has been used in previous gene–environment studies to assess childhood abuse (e.g., Aguilera et al., 2009). The frequency of childhood abuse did not vary as a function of either the 5-HTT genotype, $F(2, 233) = 0.11$, $p > .90$, or MAO-A genotype, $F(1, 225) = 0.92$, $p > .34$.

Genomic DNA was collected using buccal swabs, and genotyping was conducted by the Salimetrics Corporation (http://www.salimetrics.com). The serotonin transporter gene (SLC6A4) polymorphism 44-base-pair repeat in the promoter region was investigated according to Wendland (2008), with forward and reverse primers, 5′-TCCTCCGCTTTGGCGCCTCTTCC-3′ and 5′-TGGGGGTTGCAGGGGAGATCCTG-3′. The MAO-A polymorphism 30-base-pair repeat in the 3′ untranslated region was investigated according to Sabol et al. (1998), using forward and reverse primers, 5′-ACAGCCTGACCGTGGAGAAG-3′ and 5′-GAACGGACGCTCCATTCGGA-3′. Genotyping was reconducted in approximately 5% of samples to confirm genotype results; these quality control analyses produced identical results in 100% of the samples that were retested.

Table 1 provides data on genotype distribution and the bivariate relationship between the 5-HTT and MAO-A genotypes. Chi-square analysis indicates that the genotypes do not covary ($p >$ .56). Genotype distributions are also provided separately below for the two largest ethnic groups in this sample, European Americans and African Americans. For European American participants, the 5-HTT genotype distribution was short/short = 13 (18.8%), short/

Table 1
Cross-Tabulation of 5-HTT Genotype and Monoamine Oxidase-A (MAO-A) Genotype (n = *226)*

	MAO-A genotype		
5-HTT genotype	Low-activity	High-activity	Total
Short/short	12	13	25
Long/short	53	46	99
Long/long	47	55	102
Total	112	114	226

long = 35 (50.7%), and long/long = 20 (29.0%), which is consistent with normative samples of primarily European American participants (Caspi et al., 2003; Lesch et al., 1996). For African American participants, the 5-HTT genotype distribution was short/short = 10 (6.8%), short/long = 58 (39.2%), and long/long = 80 (54.1%), consistent with research showing the long allele is more prevalent among individuals of African American descent (Gelernter et al., 1997). For European American participants, MAO-A genotype distribution was 26 (37.7%) individuals with the low-activity variant (defined as 2- or 3-repeat alleles) and 42 (60.9%) individuals with the high-activity variant (defined as 3.5-repeat alleles and longer), which is consistent with previous research (Caspi et al., 2002; Fowler et al., 2009; Kim-Cohen et al., 2006; Sabol et al., 1998). For African American participants, MAO-A genotype distribution was 77 (52.0%) individuals with the low-activity variant and 62 (41.9%) individuals with the high-activity variant, which is consistent with research showing higher rates of the low-activity variant in African American than European American samples (Reti et al., 2011). The 5-HTT genotype was in Hardy–Weinberg equilibrium ($\chi^2 = 0.20$, $p = .64$) for the whole sample and within the European American (5-HTT: $\chi^2 = 0.11$, $p > .74$) and African American (5-HTT: $\chi^2 = 0.01$, $p > .91$) subsamples. It was not possible to assess Hardy–Weinberg equilibrium for MAO-A genotype given the lack of heterozygotes. One and 10 samples did not yield results for the 5-HTT and MAO-A analyses, respectively.

Data Analysis

Descriptive statistics and intercorrelations for the two-factor and three-factor models of psychopathy are presented in Table 2. Study aims were investigated using hierarchical regression analyses with 5-HTT genotype, MAO-A genotype, and CTQ Childhood Abuse, as well as the interaction between each genotype and abuse, entered as predictors of the psychopathy factors. Ethnicity, age, age^2, and income were entered as covariates in analyses. We decomposed 5-HTT genotype into orthogonal contrasts of long versus short (linear contrast; short/short vs. long/long) and heterozygous versus homozygous (quadratic contrast; short/long vs. short/short and long/long) to fully represent the variance associated with 5-HTT genotype. The long versus short contrast was used to test a priori hypotheses concerning the two homozygous allele groups, specifically that (a) interpersonal and affective traits are higher among long/long than short/short carriers, and (b) impulsive and antisocial traits are higher among short/short than long/long carriers. The heterozygous versus homozygous contrast captures the remaining variance associated with 5-HTT genotype and tests whether heterozygous carriers differ from homozygous carriers, which is an alternative hypothesis to our a priori predictions. Ethnicity (African American, European American, Hispanic, and mixed ethnicity) was dummy coded before entering it as a covariate. Analyses were also conducted with the nonpredicted psychopathy factors (based on either the two-factor or three-factor model of psychopathy) entered in the first block as covariate(s) to test for suppressor effects among the psychopathy dimensions. Supplementary analyses were also conducted to assess whether the genetic effects differed across ethnicity. To help protect against Type I error, we used a Bonferroni correction to maintain a family-wise error rate of .05 by dividing an alpha of .05 by the number of regression analyses conducted to test our primary hypotheses (10). Thus, results were interpreted as significant when the p value was less than or equal to .005.

Results

Two-Factor Model of Psychopathy

PCL: SV Factor 1. Results of the regression analyses are presented in the left columns of Table 3. As predicted, the 5-HTT long versus short contrast was significant for the interpersonal–affective traits on PCL: SV Factor 1, $t(204) = 2.89$, $R^2 = .04$, $p = .004$, whereby scores on PCL: SV Factor 1 were higher in long/long carriers than short/short carriers (short/short: $M = 4.1$, $SD = 2.9$; short/long: $M = 5.2$, $SD = 2.5$; long/long: $M = 5.8$, $SD = 2.7$). Neither the heterozygous versus homozygous contrast for 5-HTT genotype nor MAO-A genotype was associated with

Table 2
Descriptive Statistics and Intercorrelations for the Psychopathy Checklist: Screening Version (PCL: SV; N = *237)*

	Two-factor model		Three-factor model		
Statistic	Factor 1	Factor 2	Interpersonal	Affective	Lifestyle
M (*SD*)	5.1 (2.7)	7.3 (2.7)	2.5 (1.7)	2.9 (1.4)	3.5 (1.4)
Minimum/maximum	0/12	0/12	0/6	0/6	0/6
Intercorrelations					
PCL: SV Factor 1		.46*	.88*	.82*	.31*
PCL: SV Factor 2			.28*	.53*	.78*
PCL: SV Interpersonal				.46*	.20*
PCL: SV Affective					.33*

* $p < .05$.

Table 3
Regression Analysis of the Psychopathy Checklist: Screening Version (PCL: SV) Two-Factor Model

Variable	Factor 1			Factor 2		
	t	R^2	*p*	*t*	R^2	*p*
Block 1						
Age	0.84	.00	.39	−0.76	.00	.45
Age^2	−0.63	.00	.52	0.42	.00	.68
Ethnicity (European American)	0.57	.00	.56	−0.23	.00	.82
Ethnicity (African American)	0.40	.00	.68	−0.43	.00	.67
Ethnicity (mixed ethnicity)	0.63	.00	.52	−0.95	.00	.34
Income	−0.46	.00	.64	−3.00	.04	**.003**
Block 2						
CTQ Childhood Abuse	2.63	.03	.009	3.31	.05	**.001**
5-HTT linear (l/l vs. s/s)	2.89	.04	**.004**	2.84	.03	**.005**
5-HTT quadratic (s/l vs. l/l & s/s)	−0.49	.00	.62	−1.15	.01	.25
MAO-A genotype	1.17	.01	.23	2.87	.03	**.005**
Block 3						
5-HTT Linear × CTQ Childhood Abuse	1.42	.01	.15	1.02	.00	.31
5-HTT Quadratic × CTQ Childhood Abuse	−0.07	.00	.93	−1.04	.00	.30
MAO-A × CTQ Childhood Abuse	1.93	.02	.055	0.64	.00	.53

Note. MAO-A = monoamine oxidase-A; CTQ = Childhood Trauma Questionnaire. 5-HTT linear: short/short = –1, short/long = 0, long/long = 1. 5-HTT quadratic: short/short = 1, short/long = 0, long/long = 1. MAO-A genotype: low activity = 1, high activity = 0. Factor 1: Block 1 R^2 = .04, Block 2 ΔR^2 = .06, Block 3 ΔR^2 = .02. Factor 2: Block 1 R^2 = .05, Block 2 ΔR^2 = .10, Block 3 ΔR^2 = .01. Bolded *p*-value terms are interpreted as significant at $p \leq .005$. Block 1 degrees of freedom = 208, Block 2 degrees of freedom = 204, Block 3 degrees of freedom = 201.

scores on PCL: SV Factor 1. CTQ Childhood Abuse was not significantly associated with this psychopathy factor, and neither genotype interacted with childhood abuse to account for scores on PCL: SV Factor 1.

We next examined whether removing the shared variance between the PCL: SV factors changed the observed relationships with genotype and childhood abuse history. Accounting for shared variance with PCL: SV Factor 2 weakened the relationship of PCL: SV Factor 1 with the 5-HTT long versus short contrast, $t(203) = 1.74$, $p = .083$, to nonsignificance. MAO-A genotype, childhood abuse, and the Childhood Abuse × Genotype interactions remained nonsignificant when PCL: SV Factor 2 was entered into the model.

PCL: SV Factor 2. Results of the regression analyses are presented in the right columns of Table 3. MAO-A genotype was associated with the expression of impulsive–antisocial traits on PCL: SV Factor 2, $t(204) = 2.87$, $R^2 = .03$, $p = .005$, such that carriers of the low-activity MAO-A variant evidenced higher PCL: SV Factor 2 scores than those with the high-activity variant (low-activity variant: $M = 7.9$, $SD = 2.5$; high-activity variant: $M = 7.2$, $SD = 2.6$). A significant relationship between the 5-HTT long versus short contrast and PCL: SV Factor 2 unexpectedly emerged, $t(204) = 2.84$, $R^2 = .03$, $p = .005$, such that scores on PCL: SV Factor 2 were higher for homozygous long than homozygous short allele carriers (short/short: $M = 6.5$, $SD = 2.6$; short/long: $M = 7.5$, $SD = 2.4$; long/long: $M = 7.8$, $SD = 2.6$). CTQ Childhood Abuse also evidenced a positive relationship with scores on this psychopathy dimension, $t(204) = 3.31$, $R^2 = .05$, $p = .001$, although a history of childhood abuse did not interact with the genotypes.

Accounting for shared variance with PCL: SV Factor 1 weakened the relationship of PCL: SV Factor 2 with the 5-HTT long versus short contrast to nonsignificance, $t(203) = 1.65$, $p = .10$, suggesting that the relationship of Factor 2 with the long allele is largely accounted for by overlapping variance with Factor 1. Relationships between MAO-A genotype, $t(203) = 2.61$, $p = .01$, and CTQ Childhood Abuse, $t(203) = 2.30$, $p = .022$, with PCL: SV Factor 2 were no longer significant when Factor 1 was entered into the model. The Childhood Abuse × Genotype interactions remained nonsignificant.

Three-Factor Model of Psychopathy

PCL: SV Arrogant and Deceitful Interpersonal Style (Interpersonal factor). Results of the regression analyses are presented in the left columns of Table 4. The regression analysis conducted on the interpersonal features of psychopathy did not produce significant effects for the 5-HTT genotype contrasts, MAO-A genotype, or CTQ Childhood Abuse. No new significant effects emerged for the PCL: SV Interpersonal factor when the nonpredicted psychopathy factors were entered into the regression analysis as covariates.

PCL: SV Deficient Affective Experience (Affective factor). Results of the regression analyses are presented in middle columns of Table 4. As expected, the 5-HTT long versus short contrast was associated with the PCL: SV Affective factor, $t(203) = 2.96$, $R^2 = .04$, $p = .003$, with long/long carriers evidencing higher scores on this psychopathy dimension than short/short carriers (short/short: $M = 2.3$, $SD = 1.3$; short/long: $M = 2.8$, $SD = 1.4$; long/long: $M = 3.1$, $SD = 1.4$). The 5-HTT heterozygous versus homozygous contrast, MAO-A genotype, CTQ Childhood Abuse, and Childhood Abuse × Genotype interactions were unrelated to this psychopathy dimension.

When controlling for the other two factors, the PCL: SV Affective factor was no longer significantly related to the 5-HTT long versus short contrast, $t(201) = 2.05$, $p = .041$, and no new

Table 4
Regression Analysis of the Psychopathy Checklist: Screening Version (PCL: SV) Three-Factor Model

Variable	Interpersonal			Affective			Lifestyle		
	t	R^2	*p*	*t*	R^2	*p*	*t*	R^2	*p*
Block 1									
Age	1.32	.01	.18	−0.13	.00	.90	0.39	.00	.69
Age^2	−1.11	.01	.27	0.27	.00	.78	−0.49	.00	.62
Ethnicity (European American)	1.36	.01	.17	−0.55	.00	.58	0.55	.00	.58
Ethnicity (African American)	1.37	.01	.17	−0.89	.00	.37	−0.18	.00	.85
Ethnicity (mixed ethnicity)	1.28	.01	.20	−0.35	.00	.72	−0.12	.00	.90
Income	0.92	.00	.35	−2.01	.02	.046	−2.33	.02	.021
Block 2									
CTQ Childhood Abuse	2.31	.02	.022	2.14	.02	.033	2.29	.02	.023
5-HTT linear (l/l vs. s/s)	2.06	.02	.040	2.96	.04	**.003**	1.24	.01	.21
5-HTT quadratic (s/l vs. l/l & s/s)	−0.26	.00	.79	−0.66	.00	.50	−1.08	.01	.28
MAO-A genotype	1.19	.01	.23	0.73	.00	.46	2.85	.04	**.005**
Block 3									
5-HTT Linear × CTQ Childhood Abuse	1.99	.02	.048	0.27	.00	.78	1.75	.01	.082
5-HTT Quadratic × CTQ Childhood Abuse	0.20	.00	.84	−0.34	.00	.73	−0.78	.00	.43
MAO-A × CTQ Childhood Abuse	1.91	.02	.058	1.32	.01	.19	0.14	.00	.88

Note. MAO-A = monoamine oxidase-A; CTQ = Childhood Trauma Questionnaire. 5-HTT linear: short/short = –1, short/long = 0, long/long = 1. 5-HTT quadratic: short/short = 1, short/long = 0, long/long = 1. MAO-A genotype: low activity = 1, high activity = 0. Interpersonal: Block 1 R^2 = .05, Block 2 ΔR^2 = .04, Block 3 ΔR^2 = .04. Affective: Block 1 R^2 = .04, Block 2 ΔR^2 = .06, Block 3 ΔR^2 = .01. Lifestyle: Block 1 R^2 = .04, Block 2 ΔR^2 = .06, Block 3 ΔR^2 = .01. Bolded *p*-value terms are interpreted as significant at $p \leq .005$. Block 1 degrees of freedom = 207, Block 2 degrees of freedom = 203, Block 3 degrees of freedom = 200.

significant relationships emerged between predictors and the Affective factor.

PCL: SV Impulsive and Irresponsible Behavioral Style (Lifestyle factor). Results of the regression analyses are presented in the right columns of Table 4. Neither of the 5-HTT genotype contrasts was related to scores on the PCL: SV Lifestyle factor. However, a significant relationship did emerge between the PCL: SV Lifestyle factor and MAO-A genotype, $t(203) = 2.84$, $R^2 = .04$, $p = .005$. Specifically, the low-activity variant ($M = 3.7$, $SD = 1.4$) was associated with higher levels of impulsive and irresponsible behaviors than the high-activity variant ($M = 3.3$, $SD = 1.4$). History of childhood abuse was unrelated to this factor and did not moderate relationships of the genotypes with the PCL: SV Lifestyle factor.

MAO-A genotype continued to be associated with the PCL: SV Lifestyle factor with the other two factors entered as covariates in the analysis, $t(201) = 2.71$, $R^2 = .03$, $p = .007$, with low-activity carriers reporting more impulsive and irresponsible traits than high-activity carriers. No new relationships emerged as significant for the PCL: SV Lifestyle factor with the two nonpredicted factors entered as covariates.

Analysis of Ethnic Differences

Based on literature that indicates that more African Americans carry the 5-HTT long allele than European Americans (Gelernter et al., 1997), we conducted analyses to test whether the reported genetic effects differed as a function of ethnicity. Specifically, we created interaction terms with ethnicity for 5-HTT genotype contrasts, MAO-A genotype, and the Genotype × CTQ Childhood Abuse interactions and reconducted analyses to investigate the primary study aims using the African American and European American participants, the only ethnic groups with sufficient representation to examine genetic effects. Results of these analyses indicate that ethnicity did not significantly moderate relationships of the genotypes with the psychopathy factors in the present sample.

Discussion

The present study provides preliminary data to advance molecular genetics research on psychopathic traits that emerge in clinical-forensic samples. Consistent with hypotheses, we found that homozygous carriers of the 5-HTT long allele compared with homozygous carriers of the 5-HTT short allele exhibited higher scores on the affective–interpersonal traits that constitute the hallmark features of psychopathy (PCL: SV Factor 1 and Deficient Affective Experience; Cleckley, 1941). As expected, we also found that the MAO-A low-activity variant compared with the high-activity variant was associated with higher levels of the impulsive–antisocial traits (PCL: SV Factor 2 and Impulsive and Irresponsible Behavioral Style) that psychopathy shares with other disorders on the externalizing spectrum of psychopathology (Krueger et al., 2002). Given our relatively small sample size and evidence that genetic relationships with psychopathology variables often fail to replicate (e.g., Duncan & Keller, 2011; Risch et al., 2009), these findings should be interpreted cautiously and considered preliminary evidence for relationships between psychopathic personality traits and 5-HTTLPR and MAO-A polymorphisms.

These results can serve as a basis for future molecular genetics research on psychopathy and contribute to the growing nomological networks of the psychopathy factors in forensic samples, including the less researched three-factor model. Upon replication in larger epidemiological samples, the present results support an etiological model of psychopathy in which the heritable components for the interpersonal, affective, and lifestyle traits are differentiated. Analysis of redundancy among the psychopathy dimensions revealed some specificity in the relationships of each trait

dimension with the genotypes. In particular, MAO-A genotype was most consistently associated with the impulsive and irresponsible traits that characterize the Lifestyle factor. Although we replicated previous work in adolescents showing a link between the 5-HTT long allele and interpersonal–affective features of psychopathy in this adult clinical-forensic sample, relationships between 5-HTT genotype and the interpersonal–affective psychopathy traits were weakened to nonsignificance when the nonpredicted psychopathy factors were entered into analyses as covariates. This pattern of results may indicate that it is the shared variance among the psychopathy factors that relates to 5-HTT genotype. However, the relatively small sample size for a molecular genetics study requires tenuous interpretation of these null results, and redundancy in psychopathy relationships with 5-HTT and MAO-A genotypes needs to be investigated further in larger samples.

Although preliminary, the present results are consistent with the broader literature linking both the 5-HTT long allele and interpersonal–affective deficits in psychopathy with decreased limbic reactivity to emotional stimuli (e.g., Hariri et al., 2002; Gordon, Baird, & End, 2004). They also correspond with evidence that carriers of the MAO-A low-activity variant and individuals high in impulsive–antisocial traits both show enhanced subcortical reactivity to aversive stimuli (e.g., Gordon et al., 2004; Meyer-Lindenberg et al., 2006). Future research should examine the extent to which biological, affective, and cognitive processes mediate relationships between 5-HTT and MAO-A genotypes and psychopathic traits. This line of research would further our understanding of the functional significance of genotype–psychopathology relationships and their etiological implications.

One unexpected finding was the lack of moderation of the genetic effects by childhood abuse history, which the literature links to both increased prevalence for general antisocial behavior and psychopathic traits in particular (Caspi et al., 2002; Frazzetto et al., 2007; Kim-Cohen et al., 2006; Lang et al., 2002; Marshall & Cooke, 1999; Poythress et al., 2006; Weiler & Widom, 1996; Widom & Brzustowicz, 2006). Recruitment of a clinical-forensic sample in the present study resulted in elevated base rates of psychosocial adversity, including childhood abuse history. Thus, there may have been a restricted range concentrated at the high end of the continuum of environmental stress that limited our ability to measure and detect moderation of genetic effects by environmental adversity. Previous research has found associations between the 5-HTT long allele and the interpersonal–affective features of psychopathy to be the strongest in low SES contexts (Sadeh et al., 2010). However, at least one study to date has found that the 5-HTT long allele is directly associated with psychopathic-like traits (i.e., low socialization) in men with alcohol dependence rather than a gene–environment interaction (Herman et al., 2011). Inconsistent findings in the relationship of 5-HTTLPR polymorphisms and environmental adversity with psychopathic traits across studies and samples underscore the need for future research to examine how sample characteristics and type of environmental adversity affect these relationships.

The present study benefited from recruitment of a clinical-forensic sample, use of a well-validated assessment of psychopathic traits, and examination of multiple genotypes. In terms of the latter, the genotypes analyzed were chosen on the basis of evidence of their functional relationships with emotional processes theorized to be associated with the psychopathy dimensions. The present results were generally consistent with a priori hypotheses and broadly replicate previous studies examining relationships between the 5-HTT and MAO-A genotypes with antisocial behavior and psychopathic traits. Furthermore, this study is the first to examine the genetic correlates of psychopathy in a high-risk sample of adults with a history of illegal behavior, economic disadvantage, and life stress.

As with any investigation, the present study has limitations that should be considered when interpreting the results. First, it is important to note that the current sample is not representative of the general population, particularly because it included purposive selection of men with antisocial histories. These purposive sampling methods resulted in an overrepresentation of men with exposure to early adversity, criminal justice involvement, low SES, and racial heterogeneity. Thus, results of the current study are best positioned to inform similar high-adversity male samples, and inferences about the genetic correlates of psychopathic traits in the general population should not be made. Until replication of the present findings is established in population-based samples, the generalizability of the present research should be limited to similar high-risk male populations.

Second, although the 5-HTT and MAO-A genotypes were not related to childhood abuse in the present study, it was not possible to rule out the effects of gene–environment correlations, given the large number of gene–environment relationships that were not assessed. Indeed, behavioral genetics research has highlighted the difficulty in partitioning genetic from environmental influences when measuring family-level variables, and researchers cannot assume that family experiences, such as childhood abuse, are fully environmentally determined (Turkheimer et al., 2005). Only two genotypes and one candidate environment were examined in the present study, leaving open the possibility that other genes and environments are important for the manifestation of psychopathic traits and account for the genetic relationships observed in the present study.

Third, our measure of childhood abuse is limited by measurement bias in the form of self-report biases, such as social desirability, and retrospective recall biases. Research examining the limitations associated with using retrospective measures of adverse events in childhood suggests that this methodological approach has substantial measurement bias, but it can provide relevant information as long as the events assessed are adequately operationalized, do not rely on detailed accounts, and are serious enough to be recalled (Hardt & Rutter, 2004). Measurement of childhood physical and sexual abuse using the CTQ meets these requirements, suggesting it provides a valid measure of childhood abuse, despite the bias inherent in the use of a retrospective measure of childhood experiences. In addition, the CTQ is a widely used measure that has shown good temporal reliability and criterion-related validity, including therapist assessment of childhood adversity experiences and clinician-rated interviews of childhood abuse (Bernstein et al., 1994, 2003). Nonetheless, failure to detect relationships in the present sample may be an artifact of the use of a self-report retrospective childhood abuse measure or the restricted range in adverse childhood experiences inherent in the forensic sample studied. Thus, the present results should not be interpreted as evidence that childhood abuse does not interact with genes in

relation to psychopathy or that it is not an important environmental contributor to the development of psychopathic traits.

Finally, the present sample was modest in size and racially heterogeneous. Recent research suggests that there is publication bias in gene–environment studies, such that only 27% of studies replicate the findings of novel gene–environment relationships with psychological outcomes (Duncan & Keller, 2011), which may result from the use of small samples that increases the likelihood that results are spuriously driven by sample characteristics rather than a true effect. Thus, it is important to be cautious when interpreting our fairly novel genetic relationships until data on the replicability of the findings in larger samples are available. Furthermore, our relatively small sample for a molecular genetics study limits interpretation of null results, which could be driven by a lack of power to detect relationships with small effect sizes or genetic differences among ethnic groups, which we did not have the power to adequately examine. Although we took precautions to reduce the likelihood that the present findings are spurious (e.g., correcting for multiple comparisons, assessing bivariate outliers, conducting multiple transformations of the data), the small and heterogeneous sample increases concern that the results will not replicate or are an artifact of ethnic differences. Thus, results should be interpreted as preliminary pending replication in larger, more homogeneous samples of individuals.

With these limitations in mind, the present results provide new preliminary evidence on the molecular genetics correlates of psychopathic traits in a forensic sample of men. Results suggest that psychopathy may be etiologically multidetermined, and it would be fruitful to further explore these relationships in future research.

References

Aguilera, M., Arias, B., Wichers, M., Barrantes-Vidal, N., Moya, J., Villa, H., . . . Fañanás, L. (2009). Early adversity and 5-HTT-BDNF genes: New evidences of gene–environment interactions on depressive symptoms in a general population. *Psychological Medicine, 39,* 1425–1432. doi:10.1017/S0033291709005248

Anguelova, M., Benkelfat, C., & Turecki, G. (2003). A systematic review of association studies investigating genes coding for serotonin receptors and the serotonin transporter: II. Suicidal behavior. *Molecular Psychiatry, 8,* 646–653. doi:10.1038/sj.mp.4001336

Beitchman, J. H., Baldassarra, L., Mik, H., De Luca, V., King, N., Bender, D., . . . Kennedy, J. L. (2006). Serotonin transporter polymorphisms and persistent, pervasive childhood aggression. *The American Journal of Psychiatry, 163,* 1103–1105. doi:10.1176/appi.ajp.163.6.1103

Benning, S. D., Patrick, C. J., Hicks, B. M., Blonigen, D. M., & Krueger, R. F. (2003). Factor structure of the Psychopathic Personality Inventory: Validity and implications for clinical assessment. *Psychological Assessment, 15,* 340–350. doi:10.1037/1040-3590.15.3.340

Bernstein, D. P., Fink, L., Handelsman, L., Foote, K., Lovejoy, M., Wenzel, K., . . . Ruggiero, J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *The American Journal of Psychiatry, 151,* 1132–1136.

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., . . . Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse & Neglect, 27,* 169–190. doi:10.1016/S0145-2134(02)00541-0

Buckholtz, J. W., & Meyer-Lindenberg, A. (2008). MAOA and the neurogenetic architecture of human aggression. *Trends in Neurosciences, 31,* 120–129. doi:10.1016/j.tins.2007.12.006

Canli, T., & Lesch, K. (2007). Long story short: The serotonin transporter in emotion regulation and social cognition. *Nature Neuroscience, 10,* 1103–1109.

Canli, T., Omura, K., Haas, B. W., Fallgatter, A., Constable, R. T., & Lesch, K. P. (2005). Beyond affect: A role for genetic variation of the serotonin transporter in neural activation during a cognitive attention task. *Proceedings of the National Academy of Sciences, USA, 102,* 12224–12229. doi:10.1073/pnas.0503880102

Carver, C. S., Johnson, S. L., Joorman, J., Kim, Y., & Nam, J. Y. (2011). Serotonin transporter polymorphism interacts with childhood adversity to predict aspects of impulsivity. *Psychological Science, 22,* 589–595. doi:10.1177/0956797611404085

Carver, C. S., & Miller, C. J. (2006). Relations of serotonin function to personality: Current views and a key methodological issue. *Psychiatry Research, 144,* 1–15. doi:10.1016/j.psychres.2006.03.013

Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *The American Journal of Psychiatry, 167,* 509–527. doi:10.1176/appi.ajp.2010.09101452

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., . . . Poulton, R. (2002, August 2). Role of genotype in the cycle of violence in maltreated children. *Science, 297,* 851–854. doi:10.1126/science.1072290

Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: Joining forces with neuroscience. *Nature Reviews Neuroscience, 7,* 583–590. doi:10.1038/nrn1925

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., . . . Poulton, R. (2003, July 18). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301,* 386–389. doi:10.1126/science.1083968

Cleckley, H. (1941). *The mask of sanity*. St. Louis, MO: Mosby.

Cooke, D. J., & Michie, C. (2001). Refining the construct of psychopathy: Towards a hierarchical model. *Psychological Assessment, 13,* 171–188. doi:10.1037/1040-3590.13.2.171

Cooke, D. J., Michie, C., & Hart, S. D. (2004). Cross-national differences in the assessment of psychopathy: Do they reflect variations in raters' perceptions of symptoms? *Psychological Assessment, 16,* 335–339. doi:10.1037/1040-3590.16.3.335

Cooke, D. J., Michie, C., Hart, S. D., & Hare, R. D. (1999). Evaluating the Screening Version of the Hare Psychopathy Checklist—Revised (PCL: SV): An item response theory analysis. *Psychological Assessment, 11,* 3–13. doi:10.1037/1040-3590.11.1.3

DiLillo, D., Fortier, M. A., Hayes, S. A., Trask, E., Perry, A. R., Messman-Moore, T., . . . Nash, C. (2006). Retrospective assessment of childhood sexual and physical abuse: A comparison of scaled and behaviorally specific approaches. *Assessment, 13,* 297–312. doi:10.1177/1073191106288391

Dolan, M. C., & Anderson, I. M. (2003). The relationship between serotonergic function and the Psychopathy Checklist: Screening Version. *Journal of Psychopharmacology, 17,* 216–222. doi:10.1177/0269881103017002011

Douglas, K., Chan, G., Gelernter, J., Arias, A. J., Anton, R. F., Poling, J., . . . Kranzler, H. R. (2011). 5-HTTLPR as a potential moderator of the effects of adverse childhood experiences on risk of antisocial personality disorder. *Psychiatric Genetics, 21,* 240–248. doi:10.1097/YPG.0b013e3283457c15

Duncan, L. E., & Keller, M. C. (2011). A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. *The American Journal of Psychiatry, 168,* 1041–1049. doi:10.1176/appi.ajp.2011.11020191

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured Clinical Interview for DSM–IV–TR Axis I Disorders, Re-*

search Version, Nonpatient Edition. New York, NY: Biometrics Research, New York State Psychiatric Institute.

Foley, D. L., Eaves, L. J., Wormley, B., Silberg, J. L., Maes, H. H., Kuhn, J., & Riley, B. (2004). Childhood adversity, monoamine oxidase A genotype, and risk for conduct disorder. *Archives of General Psychiatry, 61,* 738–744. doi:10.1001/archpsyc.61.7.738

Fowler, T., Langley, K., Rice, F., van den Bree, M. B. M., Ross, K., Wilkinson, L. S., . . . Thapar, A. (2009). Psychopathy trait scores in adolescents with childhood ADHD: The contribution of genotypes affecting MAOA, 5HTT and COMT activity. *Psychiatric Genetics, 19,* 312–319. doi:10.1097/YPG.0b013e3283328df4

Fowles, D. C., & Dindo, L. (2006). A dual deficit model of psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 14–34). New York, NY: Guilford Press.

Frazzetto, G., Di Lorenzo, G., Carola, V., Proietti, L., Sokolowska, E., Siracusano, A., . . . Troisi, A. (2007). Early trauma and increased risk for physical aggression during adulthood: The moderating role of MAOA genotype. *PLoS One, 2,* e486. doi:10.1371/journal.pone.0000486

Gelernter, J., Kranzler, H., & Cubells, J. F. (1997). Serotonin transporter protein (SLC6A4) allele and haplotype frequencies and linkage disequilibria in African and European American and Japanese populations and in alcohol dependent subjects. *Human Genetics, 101,* 243–246. doi:10.1007/s004390050624

Glenn, A. L. (2011). The other allele: Exploring the long allele of the serotonin transporter gene as a potential risk factor for psychopathy: A review of the parallels in findings. *Neuroscience & Biobehavioral Reviews, 35,* 612–620. doi:10.1016/j.neubiorev.2010.07.005

Gordon, H. L., Baird, A. A., & End, A. (2004). Functional differences among those high and low on a trait measure of psychopathy. *Biological Psychiatry, 56,* 516–521. doi:10.1016/j.biopsych.2004.06.030

Gotlib, I. H., Joorman, J., Minor, K. L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry, 63,* 847–851. doi:10.1016/j.biopsych.2007.10.008

Greenberg, B. D., Tolliver, T. J., Huang, S. J., Li, Q., Bengel, D., & Murphy, D. L. (1999). Genetic variation in the serotonin transporter promoter region affects serotonin uptake in human blood platelets. *American Journal of Medical Genetics, 88,* 83–87. doi:10.1002/(SICI)1096-8628(19990205)88:1<83::AID-AJMG15>3.0.CO;2-0

Guy, L. S., & Douglas, K. S. (2006). Examining the utility of the PCL: SV as a screening measure using competing factor models of psychopathy. *Psychological Assessment, 18,* 225–230. doi:10.1037/1040-3590.18.2.225

Haberstick, B. C., Smolen, A., & Hewitt, J. K. (2006). Family-based association test of the 5-HTTLPR and aggressive behavior in a general population sample of children. *Biological Psychiatry, 59,* 836–843. doi:10.1016/j.biopsych.2005.10.008

Hall, J. R., Benning, S. D., & Patrick, C. J. (2004). Criterion-related validity of the three-factor model of psychopathy: Personality, behavior, and adaptive functioning. *Assessment, 11,* 4–16. doi:10.1177/1073191103261466

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *Journal of Child Psychology and Psychiatry, 45,* 260–273. doi:10.1111/j.1469-7610.2004.00218.x

Hare, R. D. (1991). *Manual for the Hare Psychopathy Checklist—Revised*. Toronto, ON, Canada: Multi-Health Systems.

Hariri, A. R., & Holmes, A. (2006). Genetics of emotional regulation: The role of the serotonin transporter in neural function. *Trends in Cognitive Sciences, 10,* 182–191. doi:10.1016/j.tics.2006.02.011

Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachanal, B., Feral, F., Goldman, D., . . . Weinberger, D. R. (2002, July 19). Serotonin transporter genetic variation and the response of the human amygdala. *Science, 297,* 400–403. doi:10.1126/science.1071829

Harpur, T., Hare, R. D., & Hakstian, A. (1989). Two-factor conceptualization of psychopathy: Construct validity and assessment implications. *Psychological Assessment, 1,* 6–17. doi:10.1037/1040-3590.1.1.6

Hart, S. D., Cox, D. N., & Hare, R. D. (1995). *The Hare Psychopathy Checklist: Screening Version*. Toronto, ON, Canada: Multi-Health Systems.

Herman, A. I., Conner, T. S., Anton, R. F., Gelernter, J., Kranzler, H. R., & Covault, J. (2011). Variation in the gene encoding the serotonin transporter is associated with a measure of sociopathy in alcoholics. *Addiction Biology, 16,* 124–132. doi:10.1111/j.1369-1600.2009.00197.x

Hill, C. D., Neumann, C. S., & Rogers, R. (2004). Confirmatory factor analysis of the Psychopathy Checklist: Screening Version in offenders with Axis I disorders. *Psychological Assessment, 16,* 90–95. doi:10.1037/1040-3590.16.1.90

Jacob, C. P., Muller, J., Schmidt, M., Hohenberger, K., Gutknecht, L., Reif, A., . . . Lesch, K. P. (2005). Cluster B personality disorders are associated with allelic variation of monoamine oxidase A activity. *Neuropsychopharmacology, 30,* 1711–1718. doi:10.1038/sj.npp.1300737

Jaffee, S. R., Caspi, A., Moffitt, T. E., & Taylor, A. (2004). Physical maltreatment victim to antisocial child: Evidence of an environmentally mediated process. *Journal of Abnormal Psychology, 113,* 44–55. doi:10.1037/0021-843X.113.1.44

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry, 68,* 444–454. doi:10.1001/archgenpsychiatry.2010.189

Kendler, K. S., Kuhn, J. W., Vittum, J., Prescott, C. A., & Rile, B. (2005). The interaction of stressful life events and a serotonin transporter polymorphism in the prediction of episodes of major depression. *Archives of General Psychiatry, 62,* 529–535. doi:10.1001/archpsyc.62.5.529

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W., & Moffitt, T. E. (2006). MAOA, maltreatment, and gene–environment interaction predicting children's mental health: New evidence and a meta-analysis. *Molecular Psychiatry, 11,* 903–913. doi:10.1038/sj.mp.4001851

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111,* 411–424. doi:10.1037/0021-843X.111.3.411

Lahey, B., Moffitt, T. E., & Caspi, A. (Eds.). (2003). *Causes of conduct disorder and juvenile delinquency*. New York, NY: Guilford Press.

Lang, S., Klinteberg, B., & Alm, P. O. (2002). Adult psychopathy and violent behavior in males with early neglect and abuse. *Acta Psychiatrica Scandinavica, 106,* 93–100. doi:10.1034/j.1600-0447.106.s412.20.x

Lesch, K. P., Bengel, D., Heils, A., Sabol, S. Z., Greenberg, B. D., Petri, S., . . . Murphy, D. L. (1996, November 29). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science, 274*. 1527–1531. doi:10.1126/science.274.5292.1527

Lobbestael, J., Arntz, A., Harkema-Schouten, P., & Bernstein, D. (2009). Development and psychometric evaluation of a new assessment method for childhood maltreatment experiences: The interview for traumatic events in childhood. *Child Abuse & Neglect, 33,* 505–517. doi:10.1016/j.chiabu.2009.03.002

Margolin, G., & Gordis, E. B. (2000). The effects of family and community violence on children. *Annual Review of Psychology, 51,* 445–479. doi:10.1146/annurev.psych.51.1.445

Marshall, L. A., & Cooke, D. J. (1999). The childhood experiences of psychopaths: A retrospective study of familial and societal factors. *Journal of Personality Disorders, 13,* 211–225. doi:10.1521/pedi.1999.13.3.211

Meyer-Lindenberg, A., Buckholtz, J. W., Kolachana, B., Hariri, A. R., Pezawas, L., Blasi, G., . . . Weinberger, D. R. (2006). Neural mecha-

nisms of genetic risk for impulsivity and violence in humans. *Proceedings of the National Academy of Sciences, USA, 103,* 6269–6274. doi:10.1073/pnas.0511311103

Moffitt, T. E. (2005). The new look of behavioral genetics in developmental psychopathology: Gene–environment interplay in antisocial behaviors. *Psychological Bulletin, 131,* 533–554. doi:10.1037/0033-2909.131.4.533

Munafo, M. R., Brown, S. M., & Hariri, A. R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry, 63,* 852–857. doi:10.1016/j.biopsych.2007.08.016

Munafo, M. R., & Flint, J. (2011). Dissecting the genetic architecture of human personality. *Trends in Cognitive Sciences, 15,* 395–400.

Nelson, E. C., Heath, A. C., Madden, P. A. F., Cooper, M. L., Dinwiddie, S. H., Bucholz, K. K., . . . Martin, N. G. (2002). Association between self-reported childhood sexual abuse and adverse psychosocial outcomes: Results from a twin study. *Archives of General Psychiatry, 59,* 139–145. doi:10.1001/archpsyc.59.2.139

Nilsson, K. W., Sjoberg, R. L., Damberg, M., Leppert, J., Ohrvik, J., Alm, P. O., . . . Oreland, L. (2006). Role of monoamine oxidase A genotype and psychosocial factors in male adolescent criminal activity. *Biological Psychiatry, 59,* 121–127. doi:10.1016/j.biopsych.2005.06.024

Patrick, C. J., & Bernat, E. M. (2009). Neurobiology of psychopathy: A two-process theory. In G. G. Berntson & J. T. Cacioppo (Eds.), *Handbook of neuroscience for the behavioral sciences* (pp. 1110–1131). New York, NY: Wiley.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology, 102,* 82–92. doi:10.1037/0021-843X.102.1.82

Poythress, N. G., Skeem, J. L., & Lilienfeld, S. O. (2006). Associations among early abuse, dissociation, and psychopathy in an offender sample. *Journal of Abnormal Psychology, 115,* 288–297. doi:10.1037/0021-843X.115.2.288

Prichard, Z., Mackinnon, A., Jorm, A. F., & Easteal, S. (2008). No evidence for interaction between MAOA and childhood adversity for antisocial behavior. *American Journal Medical Genetics Part B: Neuropsychiatric Genetics, 147,* 228–232. doi:10.1002/ajmg.b.30581

Prom-Wormley, E. C., Eaves, L. J., Foley, D. L., Gardner, C. O., Archer, K. J., Wormley, B. K., . . . Silberg, J. L. (2009). Monoamine oxidase A and childhood adversity as risk factors for conduct disorder in females. *Psychological Medicine, 39,* 579–590. doi:10.1017/S0033291708004170

Reardon, M. L., Lang, A. R., & Patrick, C. J. (2002). Antisociality and alcohol problems: An evaluation of subtypes, drinking motives, and family history in incarcerated men. *Alcoholism: Clinical and Experimental Research, 26,* 1188–1197. doi:10.1111/j.1530-0277.2002.tb02655.x

Reti, I. M., Xu, J. Z., Yanofski, J., McKibben, J., Uhart, M., Cheng, Y., . . . Nestadt, G. (2011). Monoamine oxidase A regulates antisocial personality in Whites with no history of physical abuse. *Comprehensive Psychiatry, 52,* 188–194. doi:10.1016/j.comppsych.2010.05.005

Risch, N., Herrell, R., Lehner, T., Liang, K. Y., Eaves, L., Hoh, J., . . . Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *Journal of the American Medical Association, 301,* 2462–2471. doi:10.1001/jama.2009.878

Ross, S. R., Benning, S. D., Patrick, C. J., Thompson, A., & Thurston, A. (2009). Factors of the Psychopathic Personality Inventory: Criterion-related validity and relationship to the BIS/BAS and five-factor models of personality. *Assessment, 16,* 71–87. doi:10.1177/1073191108322207

Sabol, S. Z., Hu, S., & Hamer, D. (1998). A functional polymorphism in the monoamine oxidase A gene promoter. *Human Genetics, 103,* 273–279. doi:10.1007/s004390050816

Sadeh, N., Javdani, S., Jackson, J. J., Reynolds, E. K., Potenza, M. P., Gelernter, J., . . . Verona, E. (2010). Serotonin transporter gene associations with psychopathic traits in youth vary as a function of socioeconomic resources. *Journal of Abnormal Psychology, 119,* 604–609. doi:10.1037/a0019709

Schoenleber, M., Sadeh, N., & Verona, E. (2011). Parallel syndromes: Two dimensions of narcissism and the facets of psychopathic personality in criminally involved individuals. *Personality Disorders: Theory, Research, Treatment, 2,* 113–127.

Shih, J. C., Chen, K., & Ridd, M. J. (1999). Monoamine oxidase: From genes to behavior. *Annual Review of Neuroscience, 22,* 197–217. doi:10.1146/annurev.neuro.22.1.197

Shih, J. C., & Thompson, R. F. (1999). Monoamine oxidase in neuropsychiatry and behavior. *American Journal of Human Genetics, 65,* 593–598. doi:10.1086/302562

Smith, S. S., & Newman, J. P. (1990). Alcohol and drug abuse in psychopathic and nonpsychopathic criminal offenders. *Journal of Abnormal Psychology, 99,* 430–439. doi:10.1037/0021-843X.99.4.430

Soderstrom, H., Blennow, K., Manhem, A., & Forsman, A. (2001). CSF studies in violent offenders: I. 5-HIAA as a negative and HVA as a positive predictor of psychopathy. *Journal of Neural Transmission, 108,* 869–878. doi:10.1007/s007020170036

Soderstrom, H., Blennow, K., Sjodin, A. K., & Forsman, A. (2003). New evidence for an association between the CSF HVA: 5-HIAA ratio and psychopathic traits. *Journal of Neurology, Neurosurgery & Psychiatry, 74,* 918–921. doi:10.1136/jnnp.74.7.918

Taylor, A., & Kim-Cohen, J. (2007). Meta-analysis of gene–environment interactions in developmental psychopathology. *Development and Psychopathology, 19,* 1029–1037. doi:10.1017/S095457940700051X

Turkheimer, E., D'Onofrio, B. M., Maes, H. H., & Eaves, L. J. (2005). Analysis and interpretation of twin studies including measures of the shared environment. *Child Development, 76,* 1217–1233. doi:10.1111/j.1467-8624.2005.00845.x-i1

Vaidyanathan, U., Hall, J. R., Patrick, C. J., & Bernat, E. M. (2011). Clarifying the role of defensive reactivity deficits in psychopathy and antisocial personality using startle reflex methodology. *Journal of Abnormal Psychology, 120,* 253–258. doi:10.1037/a0021224

Verona, E., Joiner, T. E., Johnson, F., & Bender, T. (2006). Gender specific gene–environment interactions on laboratory-assessed aggression. *Biological Psychology, 71,* 33–41. doi:10.1016/j.biopsycho.2005.02.001

Verona, E., Patrick, C. J., Curtin, J. J., Bradley, M. M., & Lang, P. J. (2004). Psychopathy and physiological response to emotionally evocative sounds. *Journal of Abnormal Psychology, 113,* 99–108. doi:10.1037/0021-843X.113.1.99

Verona, E., Patrick, C. J., & Joiner, T. E. (2001). Psychopathy, antisocial personality, and suicide risk. *Journal of Abnormal Psychology, 110,* 462–470. doi:10.1037/0021-843X.110.3.462

Verona, E., Sprague, J., & Sadeh, N. (2012). Inhibitory control and emotional processing in psychopathy and antisocial personality. *Journal of Abnormal Psychology, 121,* 498–510. doi:10.1037/a0025308

Vitacco, M. J., Neumann, C. S., & Jackson, R. L. (2005). Testing a four-factor model of psychopathy and its association with ethnicity, gender, intelligence, and violence. *Journal of Consulting and Clinical Psychology, 73,* 466–476. doi:10.1037/0022-006X.73.3.466

Walsh, Z., Swogger, M. T., & Kosson, D. S. (2009). Psychopathy and instrumental violence: Facet level relationships. *Journal of Personality Disorders, 23,* 416–424. doi:10.1521/pedi.2009.23.4.416

Walters, G. D., & Heilbrun, K. (2010). Violence risk assessment and Facet 4 of the Psychopathy Checklist: Predicting institutional and community aggression in two forensic samples. *Assessment, 17,* 259–268. doi:10.1177/1073191109356685

Wang, T., Lee, S., Chen, S., Chang, Y., Chen, S., Chu, C., . . . Lu, R. B. (2012). Interaction between Serotonin Transporter and Serotonin Receptor 1 B genes polymorphisms may be associated with antisocial alcoholism [Abstract]. *Behavioral and Brain Functions, 8,* 18. doi:10.1186/1744-9081-8-18

Weiler, B. L., & Widom, C. S. (1996). Psychopathy and violent behavior in abused and neglected young adults. *Criminal Behaviour and Mental Health, 6,* 253–271. doi:10.1002/cbm.99

Wendland, J. R., Moyal, P. R., Krusel, M. R., Ren-Patterson, R. F., Jensen, C. L., Timpano, K. R., & Murphy, D. L. (2008). A novel, putative gain-of-function haplotype at SLC6A4 associates with obsessive–compulsive disorder. *Human Molecular Genetics, 17,* 717–723. doi: 10.1093/hmg/ddm343

Widom, C. S., & Brzustowicz, L. M. (2006). MAOA and the "cycle of violence": Childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. *Biological Psychiatry, 60,* 684–689. doi:10.1016/j.biopsych.2006.03.039

Young, S. E., Smolen, A., Hewitt, J. K., Haberstick, B. C., Stalings, M. C., Corley, R. P., & Crowley, T. J. (2006). Interaction between MAO-A genotype and maltreatment in the risk for conduct disorder: Failure to confirm in adolescent patients. *The American Journal of Psychiatry, 163,* 1019–1025. doi:10.1176/appi.ajp.163.6.1019

Received May 6, 2011
Revision received June 25, 2012
Accepted July 17, 2012 ■