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## Serotonin Transporter Gene Associations with Psychopathic Traits in Youth Vary as a Function of Socioeconomic Resources

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### Abstract

Although prior research has examined the genetic correlates of antisocial behavior, molecular genetics influences on psychopathic traits remain largely unknown. Consequently, we investigated the influence of polymorphic variation at the serotonin transporter protein gene (*SLC6A4*) and socioeconomic resources (SES) on psychopathic traits in youth across two distinct samples in two separate studies. In Study 1, a main effect of serotonin transporter (5-HTTLPR) genotype was associated with the impulsivity dimension of psychopathy. That is, individuals homozygous for the short allele evidenced more impulsivity than those homozygous for the long allele. In contrast, a gene-environment interaction was associated with the callous-unemotional and narcissistic features of psychopathy. Callous-unemotional and narcissistic traits increased as SES decreased only among youths with the homozygous-long (l/l) genotype, a novel finding replicated and extended in Study 2. These studies provide preliminary results that the l/l genotype confers risk for the emotional deficits and predatory interpersonal traits associated with psychopathy among youths raised in disadvantaged environments.

### Keywords

psychopathic tendencies; 5-HTT; serotonin; socioeconomic status

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Psychopathy represents a constellation of abnormal emotional (shallow affect, fearlessness, callousness), interpersonal (superficial charm, egocentricity, deceitfulness), and behavioral (impulsivity, irresponsibility, aggressiveness) characteristics that lead to frequent engagement in inadequately motivated and violent antisocial behavior (Hare, 2003). Traits that emerge in childhood, labeled psychopathic tendencies, are often precursors to destructive and antisocial behavior in adulthood. The multidimensional nature of psychopathy (e.g., Frick, Bodin, & Barry, 2000) suggests the emotional, interpersonal, and behavioral dimensions represent distinct, albeit related, developmental pathways. However, no research to date has examined the genetic correlates of these facets in youth, data that

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would shed light on the extent to which these dimensions represent biologically distinct pathways.

Research documenting serotonin (5-hydroxyindoleacetic acid; 5-HT) deficiencies among individuals with antisocial, aggressive, and impulsive behavior (Carver & Miller, 2006) suggests that genes that code for proteins important for monoaminergic neurotransmission influence psychopathy. For instance, Soderstrom and colleagues (Soderstrom, Blennow, Manhem, & Forsman, 2001; Soderstrom, Blennow, Sjodin, & Forsman, 2003) have linked cerebrospinal fluid concentrations of a serotonin metabolite (5-HIAA) to psychopathic traits in violent adult offenders, in that low levels of serotonin metabolites and high levels of dopamine metabolites were associated with overall levels of psychopathy. Extending this research, Dolan and Anderson (2003) found serotonergic functioning correlated negatively with the impulsivity dimension and positively with the interpersonal dimension of psychopathy in violent adult offenders, indicating that dimensions of psychopathy may show opposing relationships to serotonergic functioning.

Based on these studies, the serotonin transporter gene (*SLC6A4*) is a promising candidate for genetics research on psychopathy. The polymorphism studied most is a repeat in the promoter region of the *SLC6A4* that affects the expression and function of the gene, denoted 5-HTTLPR, which consists of a common 44-base-pair insertion (long allele) or deletion (short allele). Other alleles are present in some populations (Gelernter et al., 1997). Cells with the long allele produce higher concentrations of 5-HT transporter mRNA than cells with the short allele, which is hypothesized to lead to a more rapid clearance of 5-HT from the synaptic cleft. Individuals with the short allele show reduced 5-HT reuptake, confirmed in human blood platelets (Greenberg et al., 1999). The 5-HTT short allele, relative to the long allele, has been related at the phenotypic level to heightened risk for impulse control problems and psychiatric disorders characterized by affective dysregulation (Beitchman et al., 2006; Haberstick, Smolen, & Hewitt, 2006; Ni et al., 2006) and greater amygdala activation to negative emotional stimuli (Bertolino et al., 2005; Canli et al., 2006; Hariri et al., 2005). These findings are directly relevant to psychopathy as research suggests the affective and interpersonal features originate from deficient reactivity in the emotional circuitry of the brain, particularly the amygdala (Patrick, Bradley, & Lang, 1993).

Models of these genetic effects need to consider the role of environment, because psychopathic traits may only manifest in certain environments (Lykken, 1995). The availability of socioeconomic resources (SES) is a potentially important environmental indicator, given available evidence of its association with aggressive behavior and psychopathic tendencies in youth. Behavioral genetics research, for example, indicates an interaction of SES and genes is associated with adolescent antisocial behavior (Tuvblad, Grann, & Lichtenstein, 2005). Further, longitudinal work suggests that SES is associated with temporal stability in psychopathic tendencies in youth from pre-adolescence to adolescence (Frick, Kimonis, Dandreaux, & Farell, 2003), linking the availability of socioeconomic resources during development directly to psychopathic traits, although a causal relationship has not been established. Animal research has also linked social status to serotonergic function in unstable environments (Raleigh, McGuire, Brammer, Pollack, & Yuwiler, 1991). More specifically, experimental manipulation of serotonin levels in non-human primates shows that serotonin enhancements increase socially dominant behavior in environments lacking a dominant male, whereas serotonin reductions increase social aggression in these same environments. These findings are potentially relevant for understanding how serotonergic function interacts with social status in humans to predict psychopathic traits, including social dominance and aggressive behavior.

## Present Studies

We tested the effects of 5-HTT genotype, SES, and their interaction on the manifestation of psychopathic tendencies in youth in two separate studies. The purpose of Study 1 was to investigate how socioeconomic resources and 5-HTT genotype uniquely and jointly contribute to the manifestation of different psychopathic tendencies in adolescent youth. Since this was a novel research endeavor concerning psychopathy, the primary goal of Study 1 was to provide preliminary data on the risk conferred by the *SLC6A4* and economic disadvantage in a sample of youth with a wide range of psychopathic tendencies. Recent meta-analytic work has called into question the reliability of a specific gene-environment interaction in risk for depression (Risch et al., 2009), although the findings have erroneously been interpreted more broadly. Nonetheless, it is important to demonstrate replicable gene-environment interactions (Risch et al., 2009). Thus, the purpose of Study 2 was to investigate the reliability and generalizability of the results from the first study in a younger sample of youth with different regional characteristics, using a more comprehensive measure of callous-unemotional traits.

## Hypotheses

Based on the demonstrated association between the 5-HTT short allele and behavioral and emotional dysregulation (e.g., Beitchman et al., 2006), we hypothesized a dose effect such that the s/s genotype would evidence the highest levels of the impulsivity dimension followed by the l/s and l/l genotypes. Emotional and behavioral dysregulation, however, do not characterize the affective and interpersonal dimensions of psychopathy, which are marked by callousness, shallow affect, social dominance, and narcissism. Laboratory research shows that these dimensions, often considered the core of the psychopathic syndrome, relate to physiological hypoarousal in response to threatening or noxious stimuli (e.g., Patrick et al., 1993). Thus, in contrast to the behavioral dimension, the absence of the short allele may contribute to the deficient emotionality and physiological hypoarousal present in psychopathy. We therefore hypothesized a dose effect for the 5-HTT genotype such that the l/l genotype would be associated with the highest levels of callous-unemotional and narcissism traits followed by the l/s and s/s genotypes. Importantly, we expected the influence of these genetic effects to vary with the availability of social and economic resources through a gene-environment interaction. We hypothesized that SES would relate negatively to impulsive tendencies in the s/s genotype, given the association between this genotype and aggressive behavior (e.g., Beitchman et al., 2006). In contrast, we expected SES to relate negatively to callous-unemotional and narcissistic traits in l/l genotype instead, based on animal research associating social instability and differential serotonergic function to social dominance and aggression (Raleigh et al., 1991).

## Study 1

### Method

**Participants**—To ensure a diverse sample with a spectrum of psychopathic tendencies, youth from treatment or legal agencies ( $n = 88$ ) and the general community ( $n = 63$ ) were recruited via agency referrals, newspaper advertisements, and posted flyers. Informed consent and assent were obtained from a parental guardian and the youth, respectively. In total, 118 youth (58% girls;  $M age = 14.3$ ,  $SD = 1.5$ ), from a rural Midwestern region of the U.S. volunteered with a parent. Parents identified youth as European-American (75; 63.6%), African-American (19; 16.1%), biracial (14; 11.9%), Hispanic (7; 5.9%), or Asian-American (3; 2.5%). The sample was diverse in terms of income level, categorized into the following ranges: \$1–\$30,000 ( $n = 36$ , 30.5%), \$30,001–\$60,000 ( $n = 29$ , 24.6%), and \$60,001–\$75,000+ ( $n = 53$ , 44.9%).

**Measures**—The 20-item self-report Antisocial Process Screening Device (Frick & Hare, 2001) was used to assess psychopathic tendencies. It contained three subscales: Callous-Unemotional, Narcissism, and Impulsivity, which represent the affective, interpersonal, and behavioral dimensions of psychopathy, respectively. Cronbach's alphas for APSD Total (.74), Callous-Unemotional (.56), Narcissism (.65), and Impulsivity (.53) fell within the range typical for these self-report indices in other studies (Total = .72–.82; Subscales = .36–.72) (Poythress, Dembo, Wareham, & Greenbaum, 2006).

Parents provided information about family income level (as detailed above) and occupation. The latter was coded from one to seven according to the Hollingshead (1975) measure of occupational status. Mother's occupation was reported less frequently than father's occupation. Thus, father's occupation was used to code occupational status, except when it was unavailable. Reliability ratings between two raters were conducted for the Hollingshead employment classification and resulted in an acceptable intraclass correlation of .87. We created a composite SES index by standardizing and summing the annual family income and parental occupation scores.

Genomic DNA was collected using buccal swabs. To generate 484-bp (short allele) or 528-bp (long allele) fragments of the 5-HTTLPR gene (*SLC6A4*), polymerase chain reaction (PCR) amplification was performed using a modified form of the method of Lesch et al., (1996) with forward and reverse primers of, 5'-GGCGTTGCCGCTCTGAATGC-3' and 5'-GAGGGACTGAGCTGGACAACC-3'. Genotype was ascertained by agarose gel electrophoresis of PCR products and visualized by transillumination. The sample was split into three groups based on genotype, s/s ( $n = 24$ , 20%), l/s ( $n = 56$ , 48%) and l/l ( $n = 38$ , 32%), which was in Hardy-Weinberg equilibrium.

**Statistical Analyses**—Separate hierarchical linear regressions were used to test the association of psychopathic tendencies in youth with 5-HTT genotype and SES. To examine the contributions of SES and genotype above the variance accounted for by demographic variables, Block 1 included sex, age, and ethnicity, Block 2 included 5-HTT genotype and SES, and Block 3 included the 5-HTT genotype  $\times$  SES interaction. To control for the number of analyses conducted, we used protected testing and only interpreted the a-priori effects specified by the hypotheses regarding SES and 5-HTT genotype. Supplemental analyses were also conducted to ensure the results were not likely to be accounted for by differences in allelic frequency across ethnicity.

## Results

The regression analysis predicting APSD Impulsivity produced a significant effect of 5-HTT genotype ( $\beta = -.19$ ,  $\Delta R^2 = .036$ ,  $p = .037$ ) and SES ( $\beta = -.21$ ,  $\Delta R^2 = .041$ ,  $p = .026$ ) but not a gene-environment interaction ( $R^2$  for full model = .13). Impulsivity scores increased with replications of the short allele (l/l:  $M = 3.8$ ,  $SD = 1.9$ ; s/l:  $M = 4.0$ ,  $SD = 1.8$ ; s/s:  $M = 4.9$ ,  $SD = 1.6$ ), replicating prior research that linked the short allele of 5-HTT to impulsive and aggressive traits (Beitchman et al., 2006; Haberstick, Smolen, & Hewitt, 2006; Ni et al., 2006).

In contrast, the regression analyses predicting APSD Callous-Unemotional and APSD Narcissism produced the hypothesized gene-environment interactions ( $R^2$  for full model = .11 and .08, respectively). More specifically, Callous-Unemotional evidenced an inverse relationship with SES ( $\beta = -.26$ ,  $\Delta R^2 = .06$ ,  $p < .008$ ), and the 5-HTT genotype  $\times$  SES interaction was associated with both the callous-unemotional,  $\beta = -.19$ ;  $\Delta R^2 = .037$ ;  $p = .036$ , and narcissism,  $\beta = -.24$ ;  $\Delta R^2 = .054$ ;  $p = .012$ , dimensions of psychopathic tendencies. To deconstruct each interaction, we examined the effect of SES separately within each 5-HTT genotype. As hypothesized, SES was negatively associated with Callous-Unemotional ( $\beta =$

$-.51, \Delta R^2 = .25, p < .002$ ) and Narcissism ( $\beta = -.52, \Delta R^2 = .27, p < .002$ ) in the l/l genotype but not in the l/s or s/s genotypes ( $ps > .37$ ). Figure 1 illustrates that youth with the l/l genotype who lived in low-SES environments exhibited relatively higher levels of callous-unemotional and narcissism features than youth from high-SES environments. In contrast, psychopathic traits were unchanged in the l/s and s/s genotypes as a function of SES.

Given that more African-Americans carry the long allele than European-Americans (Gelernter et al., 1997), we re-conducted regression analyses for callous-unemotional and narcissism separately in European-American participants, the only ethnic group for which we had sufficient statistical power to examine the effects of population stratification. The gene-environment interaction associated with APSD Callous-Unemotional remained in this analysis ( $\beta = -.30, \Delta R^2 = .074, p = .018$ ) an effect driven by an association between SES and callous-unemotional traits in the l/l genotype ( $\beta = -.48, \Delta R^2 = .23, p = .017$ ). The gene-environment interaction also replicated for APSD Narcissism ( $\beta = -.27, \Delta R^2 = .064, p = .03$ ), which was also driven by an inverse relationship between SES and narcissism only in the l/l genotype group ( $\beta = -.45, \Delta R^2 = .21, p = .026$ ).

## Study 2

### Method

**Participants**—Youth recruited via newspaper advertisements, school postings, and teen groups from a large, urban, Eastern coastal region participated with a parent. In total, 178 youth (45% girls;  $M$  age = 10.8,  $SD = 1.0$ ). Parents identified youth as European-American (105; 59%), African-American (49; 27.5%), biracial (18; 10.1%), Hispanic (4; 2.2%), and Asian-American (2; 1.1%). Annual family income was measured continuously and spanned from \$4,548 to \$250,000 ( $M = \$94,631, SD = \$52,432$ ), representing a wide-range of income levels.

**Measures**—The 24-item self-report Inventory of Callous-Unemotional Traits (ICU; Frick, 2003) was used to more thoroughly assess the callous-unemotional traits measured by the APSD in Study 1<sup>1</sup>. It contained three subscales: Unemotional, Uncaring, and Callous. Cronbach's alpha for ICU Total (.80) was comparable to that reported in the validation study for this measure (Essau et al., 2006). Data on impulsivity were not collected in Study 2

A composite SES variable was calculated by standardizing and summing parent-reported annual family income (continuous), mother's educational attainment, and father's educational attainment. Educational attainment was a seven-level variable ranging from "Some High School" to "Advanced Degree". The genotyping procedures used have been described previously (Gelernter, Kranzler, & Cubells, 1997) and produced three genotype groups: s/s ( $n = 22, 12\%$ ), l/s ( $n = 84, 47\%$ ) and l/l ( $n = 72, 40\%$ ).

### Results

Regression analyses were identical to those in Study 1, including the use of covariates, and examined the extent to which 5-HTT genotype, SES, and their interaction were associated with ICU total score. As in Study 1, a negative association of SES ( $\beta = -.23, \Delta R^2 = .052, p < .001$ ) as well as the hypothesized 5-HTT genotype  $\times$  SES interaction ( $\beta = -.16, \Delta R^2 = .$

<sup>1</sup>We re-conducted analyses using the four items that overlapped on the ICU and APSD to examine whether results replicated with the same items across psychopathy measures. We found a composite of these items correlated negatively with SES ( $\beta = -.18, \Delta R^2 = .029, p < .013$ ) and related to the 5-HTT genotype  $\times$  SES interaction ( $\beta = -.19; \Delta R^2 = .035; p = .006$ ). Examined within each 5-HTT genotype, SES was negatively associated with the overlapping items in the l/l genotype ( $\beta = -.34; \Delta R^2 = .11; p = .003$ ) but not in the l/s or s/s genotypes ( $ps > .41$ ). Thus, the same pattern of associations emerged in both studies using overlapping items on the two psychopathy measures.

024,  $p = .023$ ) was found for total callous-unemotional traits ( $R^2$  for full model = .09). The latter interaction was decomposed by examining the effect of SES within each genotype. Among youth with the l/l genotype, SES correlated negatively with ICU Total ( $\beta = -.40$ ,  $\Delta R^2 = .15$ ,  $p < .001$ ). As seen in Figure 2, callous-unemotional traits increased as socioeconomic resources decreased among l/l genotype carriers. This finding replicated the association between callous-unemotional traits on the APSD and SES in the l/l genotype obtained in Study 1. As expected, SES was not associated with these traits in either the l/s ( $p = .07$ ) or s/s genotypes ( $p = .38$ ).

Supplemental analysis conducted to confirm that allelic differences in ethnicity could not account for the gene-environment interaction also paralleled Study 1. The 5-HTT genotype  $\times$  SES interaction remained significant when the analysis of callous-unemotional traits was conducted among European-Americans ( $\beta = -.28$ ,  $\Delta R^2 = .06$ ,  $p < .007$ ), the only ethnic group with sufficient sample size to examine the effects separately. This effect was again driven by an association between SES and callous-unemotional traits in the l/l genotype ( $\beta = -.31$ ,  $\Delta R^2 = .12$ ,  $p < .019$ ).

## General Discussion

These studies are the first to uncover and replicate the influence of a specific gene on psychopathic tendencies in youth. Dimensions of psychopathic tendencies assessed in pre-adolescent and adolescent youth with diverse environmental and demographic characteristics were differentially associated with commonly occurring allelic variants of the serotonin transporter gene. In Study 1, 5-HTT genotype was associated with the impulsivity dimension of psychopathic tendencies, and 5-HTT genotype  $\times$  environment interactions emerged for the callous-unemotional and narcissism dimensions. Our finding related to callous-unemotional traits, replicated in Study 2, suggested that these psychopathic tendencies in youth vary as a function of SES.

These results provide the first molecular genetics evidence to support an etiological model of psychopathy in which the heritable components for the impulsive and affective-interpersonal facets of the disorder are differentiated. Youth with the s/s genotype showed the highest average scores on the APSD impulsivity subscale, which is in line with prior research on serotonin dysfunction and impulsive syndromes (Beitchman et al., 2006; Haberstick, Smolen, & Hewitt, 2006; Ni et al., 2006). In contrast, youth with the l/l genotype who were raised in environments characterized by low SES exhibited the highest levels of callous-unemotional and narcissistic traits in Study 1. This differential effect of genotype on distinct psychopathy dimensions suggests that the absence of the 5-HTT short allele, an allelic variant associated with vulnerability towards emotional and behavioral disinhibition, may also confer risk for the development of antisocial deviance at the other end of the spectrum of emotionality, particularly that marked by callousness. Importantly, this novel finding was replicated using a more comprehensive measure of callous-unemotional traits in a sample of younger youth with different regional characteristics.

The present results are also consistent with the differential susceptibility hypothesis proposed in the developmental literature (e.g., Belsky & Pluess, 2009) that posits differences in environmental susceptibility (e.g. gene-environment interactions) will take the form of a crossover interaction (Belsky, 2005; Boyce & Ellis, 2005). That is, particular alleles are theorized to increase individuals' reactivity to both risky and positive rearing environments in relation to a particular phenotype. The present data suggest that the long allele may confer susceptibility in disadvantaged environments for the callous-unemotional and narcissism dimensions of psychopathy in particular, possibly as a function of the reduced emotionality associated by the long allele at a biological level. Although not assessed in the present study,

the same individuals in more affluent environments may express the emotional hyporeactivity associated with the long allele in more prosocial ways, such as fearless athletes or socially-dominant leaders. An avenue for future research is to more thoroughly investigate the traits associated with the long/long genotype in positive rearing environments.

Unexpectedly, SES did not influence the relationship of 5-HTT genotype to impulsivity, which may be a consequence of selecting SES as the candidate environment in our studies. SES might relate to the callous and narcissism dimensions of psychopathy in particular, because these traits allow youth in disadvantaged environments to obtain needed resources, regardless of the financial or emotional consequences to others. A functional link between impulsivity and SES, however, is less obvious. In addition, although SES is likely a proxy for chronic life stress, it differs from past molecular genetics research on conduct problems in youth, which has relied mostly on discrete stressful events that directly affect the individual, such as maltreatment (e.g., Caspi et al., 2002). It is possible that, whereas these more proximal and discrete events serve as environmental risk factors for conduct problems, broader measures of disadvantage such as SES may uncover relations with more specific psychopathic traits, like callousness or narcissism.

Our findings also have implications for functions of the serotonin transporter gene more broadly. A recent proposal implicates the serotonin transporter gene in the functional neural pathway between the amygdala and anterior cingulate cortex (ACC), which is theorized to regulate emotional reactivity and fear extinction (Canli & Lesch, 2007). Importantly, this path is also sensitive to environmental adversity, making it a potential biological instantiation of the observed gene-environment interaction. Research indicates that individuals who do not carry a short allele demonstrate brain activation characterized by relatively less emotional distress, more efficient monitoring of internal emotional state, and lower average resting levels of amygdala and hippocampus compared to those with the l/s or s/s genotypes (Canli et al., 2005). Though these data are often interpreted as evidence for heightened mental health risk among short-allele carriers, another interpretation is that the low reactivity associated with the l/l genotype places individuals at risk for callous traits in certain disadvantaged environments.

The present studies have several strengths, including the discovery and replication of a gene-environment interaction across two samples of youth that differed in age and regional characteristics using two measures of callous-unemotional traits. Although the effect sizes for the main and interactive effects were small, the replication in an independent sample of youth provides evidence the results are not spurious<sup>2</sup>. Nonetheless, notable limitations should be considered. Although supplemental analyses did not suggest a gene-environment correlation could account for the present findings, these effects are difficult to definitively rule-out<sup>3</sup>. Despite a large-scale study showing that only 0.14% of individuals incorrectly identify their race/ethnicity compared to genetic clustering (Tang et al., 2005), the use of self-report rather than DNA analysis to determine ethnicity classification is not ideal. Moreover, our use of SES as an indicator of environmental stress may be subject to criticism based on life stress research conducted in the depression literature (Monroe & Reid, 2008).

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<sup>2</sup>To ensure the results were not an artifact of sampling, we examined the data for outliers and non-linear distributions. No clear univariate or bivariate outliers were present in Study 1, but we excluded three participants with SES scores that were discontinuous from the distribution in Study 2. The SES and the psychopathy indices were not excessively skewed or kurtotic (values ranged from  $-0.91$  to  $1.0$ ), and transforming the data in multiple ways did not change the results for either study.

<sup>3</sup>We examined whether a gene-environment correlation could explain our findings. In Study 2 only, SES differed by genotype ( $p = .05$ ), but this was due to the high prevalence of ethnic minority youth who were both long-allele carriers (l/l and s/l) and low-SES. The relationship between SES and ethnicity completely accounted for the genotype-SES association and the correlation between genotype and SES became non-significant when ethnicity was partialled out ( $p > .42$ ).

However, these critiques may not fully apply to psychopathy, given that they were developed specifically for the study of depression. Our use of SES as an index of environment may well address some of these critiques, in that it is not purely based on self-report in the present study and its measurement is relatively straight-forward. Lastly, the findings need to be considered preliminary. The samples were modest in size and, thus, replication of the findings in larger samples is needed to determine whether SES and the 1/1 genotype confer risk for the deficient emotionality and empathy observed in psychopathy. Despite these limitations, the findings provide preliminary insight into the role of 5-HTT genotype and the availability of social and economic resources for the manifestation of psychopathic tendencies in youth.

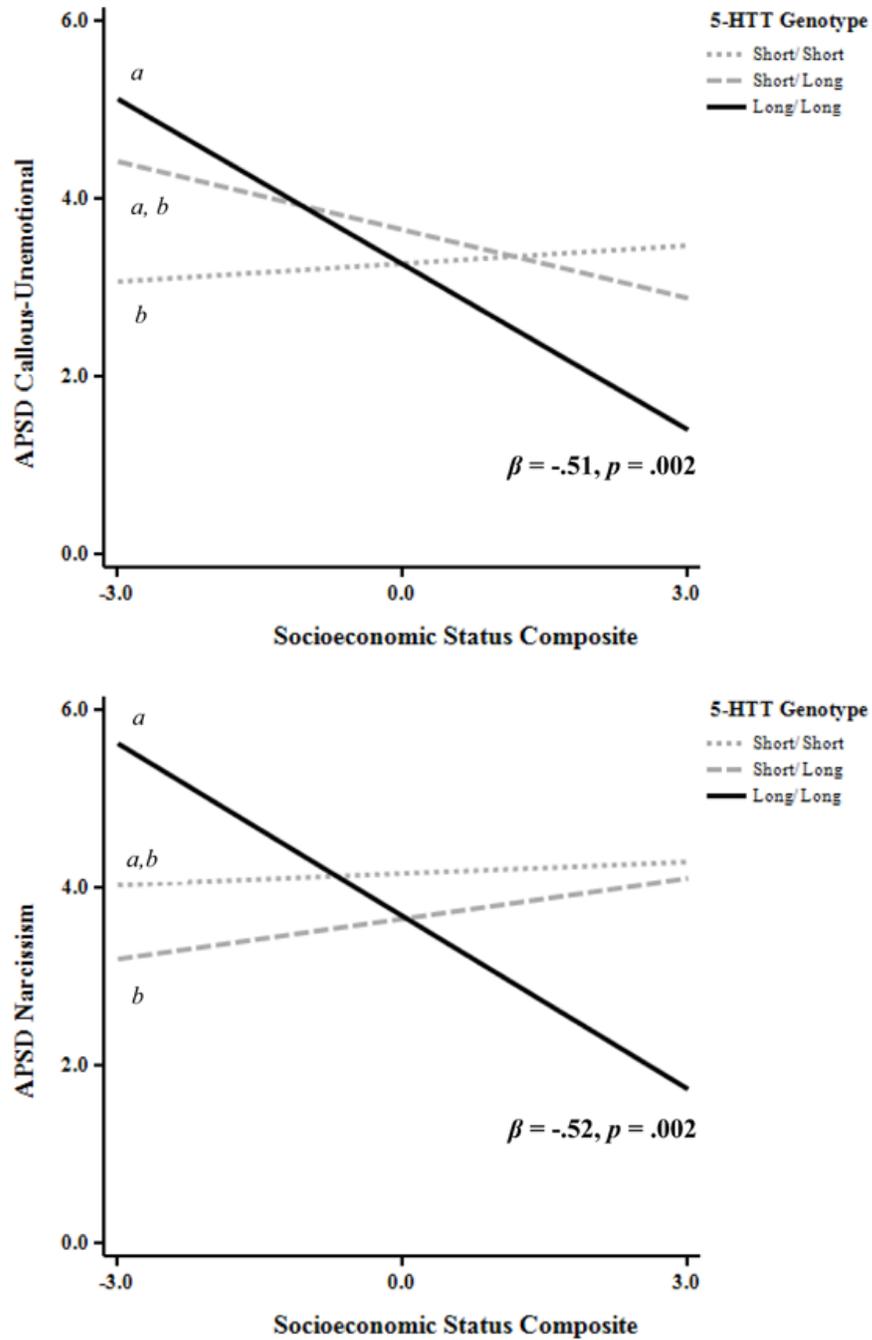
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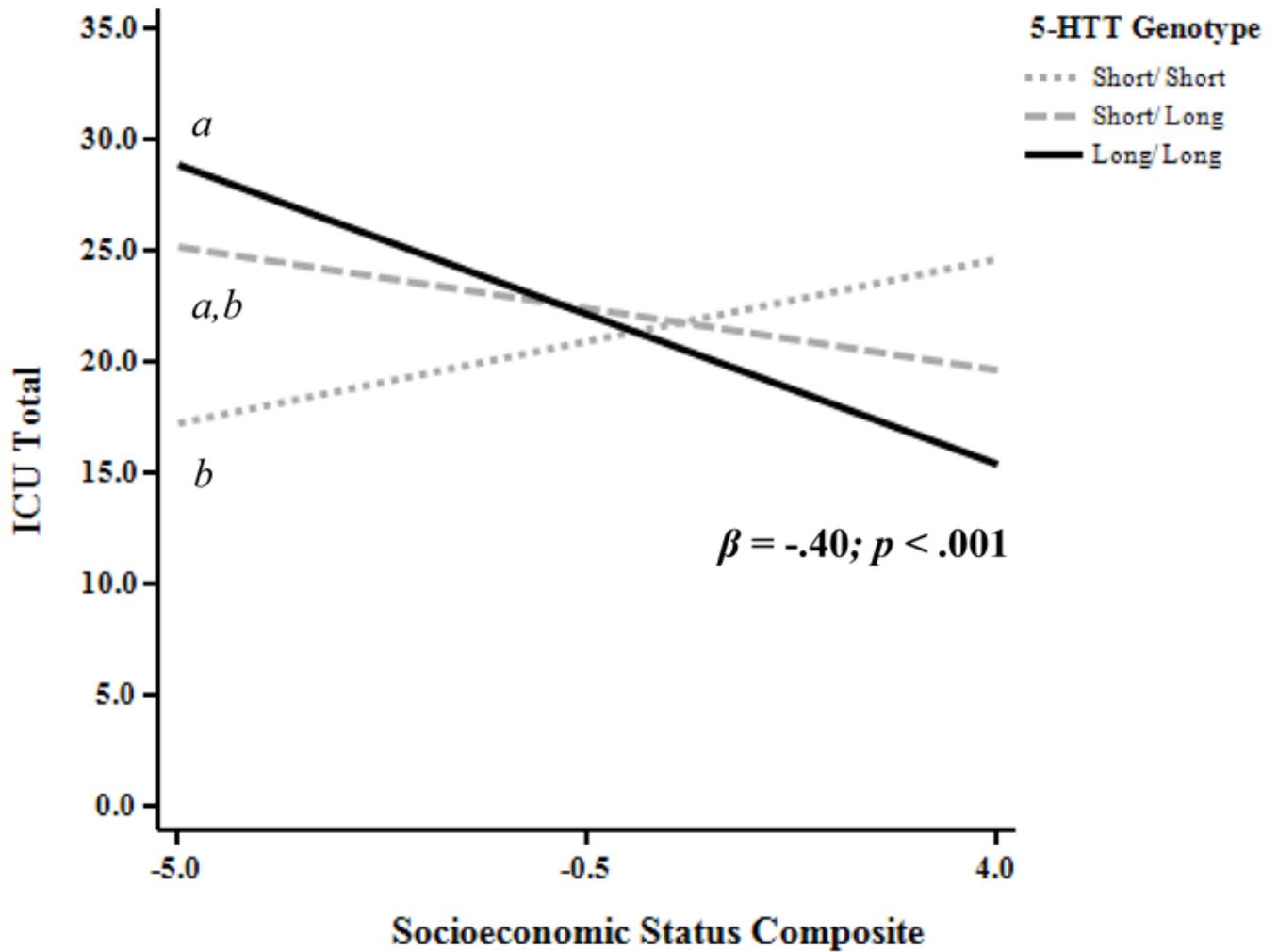
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**Figure 1.** Antisocial Process Screening Device (APSD) Callous-Unemotional Traits and Narcissism by 5-HTT Genotype and Socioeconomic Status. *Note.*  $N = 118$ . Superscripts indicate significantly different slopes,  $ps < .021$ .



**Figure 2.** Inventory of Callous-Unemotional Traits Total Score by 5-HTT Genotype and Socioeconomic Status. *Note.*  $N = 178$ . Superscripts indicate significantly different slopes,  $p = .016$ .