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Authors

Caplan, Barbara
Morgan, Julia E
Noroña, Amanda N
et al.

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The nature and nurture of social development: The role of 5-HTTLPR and gene-parenting interactions

Barbara Caplan, C.Phil.[1], **Julia Morgan, Ph.D.**[1], **Amanda N. Noroña, Ph.D.**[2], **Irene Tung, Ph.D.**[3], **Steve S. Lee, PhD.**[1], **Bruce L. Baker, Ph.D.**[1]

[1]Department of Psychology, University of California, Los Angeles

[2]Department of Psychiatry, University of Colorado Denver

[3]Department of Psychiatry, University of Pittsburgh

Abstract

Social skills are traditionally viewed as acquired through social environments including parenting. However, biopsychosocial models highlight the importance of genetic influences and gene-environment interactions (GxEs) in child development. Extant GxE investigations often fail to account for developmental changes in the phenotype or rigorously assess the social environment using observational measures. The present study prospectively assessed 110 children (44.5% female) and their parents to explore biologically plausible independent and interactive associations of the serotonin transporter-linked polymorphic region (5-HTTLPR) and observed positive *and* negative parenting in prediction of: (a) initial levels of social skills at school entry (age 6 years), and (b) developmental changes in social skills across the early school years (ages 6–9 years). Overall, the SS (vs. SL/LL) 5-HTTLPR genotype inversely predicted social skills across all domains, although parenting behavior moderated these associations wherein putative GxE effects differed by developmental timing and social skills domain. Positive parenting positively predicted concurrent (age 6) overall social skills for children with SL/LL genotypes, but not the SS genotype. However, for the SS group only, age 6 positive parenting positively predicted prospective growth in social responsibility, while negative parenting positively predicted growth in social cooperation. Findings suggest that 5-HTTLPR may signal differential sensitivities to parenting styles and patterns of social development, which may help to inform targeted intervention approaches to enhance person-environment fit.

Keywords

Social development; parenting; gene-environment interaction

The development of social competence is a major developmental milestone during childhood that plays a central role in academic, vocational, and emotional adjustment (Denham, 2006). Social *skills,* or the specific behaviors that an individual employs to accomplish social tasks, are keys predictor of social competence (Merrell & Gimpel, 2014). Childhood social skills

Correspondence concerning this article should be addressed to Barbara Caplan, Department of Psychology, University of California, Los Angeles, 3423 Franz Hall, Los Angeles, CA 90095. barbara.caplan@ucla.edu. .

include social communication (e.g., appropriate conversation skills, eye contact), assertiveness with peers and adults, responsibility for oneself and others, and self-regulation in interpersonal situations (Gresham, Elliott, Vance, & Cook, 2011). Children demonstrate tremendous growth in their social skills across development as emerging skills in cognition, perspective taking, and regulation come online (Racz, Putnick, Suwalsky, Hendricks, & Bornstein, 2017). However, some children deviate from this typical developmental trajectory and are at risk for poor outcomes in adolescence and adulthood including internalizing (i.e., depression, anxiety) and externalizing disorders (i.e., ADHD, conduct problems; e.g., Bornstein, Hahn, & Haynes, 2010). This substantial link between social functioning and developmental psychopathology warrants a better understanding of the social and biological contributors to child social skills development.

Although individual differences in social skills were traditionally viewed as acquired primarily though social interactions (Michelson, Sugai, Wood, & Kazdin, 1983), important aspects of social behavior are moderately heritable (e.g., prosocial behavior, antisocial behavior; Knafo, Israel, & Ebstein, 2011), suggesting that genetic and social environment jointly contribute to social development. In particular, the serotonin system contributes to social functioning via its regulatory role in the development and function of neural networks involved basic cognitive processes (e.g., attention, arousal) as well as downstream self-regulation and social cognition (Brummelte, Mc Glanaghy, Bonnin, & Oberlander, 2016). Moreover, medications targeting the serotonin system (e.g., selective serotonin reuptake inhibitors) have been shown to alter social behavior (Adolphs, 2001), further suggesting a causal link between serotonin neurotransmission and social functioning.

One key regulator of serotonin availability, the serotonin transporter-linked polymorphic region (5-HTTLPR), has been linked to a range of phenotypes relevant to adaptive social functioning including neuroticism, aggression, anxiety, and mood (Mueller & Canli, 2013). The short (S) and long (L) alleles of 5-HTTLPR differentially modulate the expression and function of the serotonin transporter (Homberg & Lesch, 2011). Meta-analytic evidence supports that the presence of two short alleles (i.e., the SS genotype) is linked to increased emotional reactivity, attentional bias, and empathic responding (Pergamin-Hight, Bakermans-Kranenburg, van IJzendoorn, & Bar-Haim, 2012; Gyurak et al., 2013), processes essential to adaptive social functioning. 5-HTTLPR variation directly predicted social traits and prosocial behaviors in humans and is proposed to operate on social cognition via altered neurobiological structure and function in brain regions associated with social functioning (Canli & Lesch, 2007; Stoltenberg, Christ, & Carlo, 2013). Studies of non-human animals further suggest a biologically plausible relation between 5-HTTLPR and social behavior. For example, serotonin knockout rodents show deficits in social interaction and play behavior (Kalueff, Olivier, Nonkes, & Homberg, 2010), and the orthologous rhesus macaque rh5-HTTLPR predicts social behaviors (e.g., submissive behaviors gestures and vocalizations; Bailey, Patterson, & Fairbanks, 2015). Yet, little is known about the role of 5-HTTLPR on social development in childhood, a time when social environments are likely to jointly play an influential role.

Parenting behavior is arguably the most prominent social environmental influence on child development. In fact, caregiving is necessary for normal offspring brain development across

species (Tottenham, 2015). Developmental theories, including attachment theory (Bowlby, 1969) and social information processing theory (Crick & Dodge, 1994), highlight parent-child interactions as central to the formation of cognitive schemas that provide children with a framework for future social learning and relationship formation. Indeed, high quality parenting predicts children's social, behavioral, and academic adjustment, particularly during the early- to mid-elementary school years (Eisenberg et al., 2001; NICHD, 2002). Caregiving that is sensitive and responsive is consistently related to cognitive-affective processes (Tottenham, 2015) and downstream social behavior, including more secure attachment, social competence and affect regulation (Cassidy & Shaver, 2016; NICHD, 2002). Parents may further support optimal child social adjustment through cognitive stimulation activities (Gauvin & Perez, 2015), and affect expression and emotion socialization (e.g. Eisenberg et al., 2001). In contrast, intrusive parenting is associated with poor emotional and behavioral adjustment (Barber, 2002). Importantly, child and parenting behavior occur in a dynamic transaction across development (Sameroff, 2009); children that lack social engagement to elicit parent interaction may be at risk for less exposure to positive parenting, which in turn may maintain poor social functioning. Thus, it is essential to take into account relevant child characteristics to adequately estimate parents' influence on social development over time.

Genetic and parenting factors independently influence offspring social development, but biopsychosocial models define the boundaries of these effects as well as resolve important inconsistencies (Calkins, Propper, & Mills-Koonce, 2013). Two primary models have been used to characterize the manner in which genes and environments interact (i.e. gene-environment interactions; GxEs) to predict clinical and developmental phenotypes. The diathesis-stress (or dual risk) model proposes that certain genetic variants not only serve as risk factors for poor development, but also heighten vulnerability to environmental stress (e.g., Monroe & Simons, 1991). However, many studies assuming a diathesis-stress process exclusively consider negative environments. However, evolutionary theory suggests that some biomarkers may differentially confer susceptibility to environments "for better and for worse" (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011). The low expressing SS genotype of 5-HTTLPR may be a marker of differential susceptibility (Ellis et al., 2011), simultaneously increasing sensitivity to positive *and* negative environmental influences (e.g., Taylor et al., 2006) due to increased extracellular and synaptic serotonin (Homberg & Lesch, 2011). Increased serotonin secondary to the SS genotype has been linked to hypervigilance to environmental stimuli, which may lead to adaptation or increased risk depending on the environmental context (Brummelte et al., 2016; Pergamin-Hight, Bakermans-Kranenburg, van IJzendoorn, & Bar- Haim, 2012). Individual differences in susceptibility to environmental influence may explain why previously considered risk variants (e.g., SS genotype) have been conserved across evolution, as these same factors also improve fitness in certain contexts (Ellis et al., 2011).

Although multiple functional polymorphisms (e.g., DRD4, OXTR) moderated predictions of social behavior (e.g., aggression) from parenting, 5-HTTLPR is a particularly compelling given its role in social cognition (Homberg & Lesch, 2011; Tielbeek et al., 2016). To date, GxE models in social functioning have primarily emphasized the interactive role of 5-HTTLPR and adverse childhood environments (e.g., maltreatment) on poor social outcomes,

with findings suggestive of *diathesis stress* (Tielbeek et al., 2016). While this model is commensurate with previous 5-HTTLPR studies across other domains of functioning (e.g., stressful life events, depression; Karg, Burmeister, Shedden, & Sen, 2011), as well as studies of rh5-HTTLPR in non-human primates (Canli & Lesch, 2007), the primary focus of GxE studies on *abnormal* social functioning raises questions as to whether GxEs influence social behavior on a continuum, including positive social behavior. Thus, current evidence supports the role of 5-HTTLPR x caregiving interactions in human and non-human animals, yet simultaneous examination of negative *and* positive environments on social functioning is needed to test competing models of GxE (i.e., diathesis stress, differential susceptibility; Belsky, 2016) and to adequately characterize the role of GxE in social development.

Existing GxE studies have largely relied on cross-sectional designs, limiting directional inferences and assessments of change over time. Longitudinal tests of GxE across development are strongly indicated given critical social and developmental milestones in childhood (e.g., teacher relationships, new peer groups; Ladd, 1990) and given the improved statistical power secondary to repeated measures designs. The present study builds on the extant literature by employing rigorous observational measurement of positive *and* negative parenting quality as well as longitudinal assessment of adaptive social functioning. It also examines subdomains of social skills (cooperation, assertion, responsibility, self-control) to assess the specificity of GxE effects on social development. As these social skills subdomains are differentially related to aspects of positive child adjustment (e.g., Lane, Wehby, & Cooley, 2006) and the caregiving environment (Hindman & Morrison, 2012), they may be differentially sensitive to 5-HTTLPR × parenting interactions. Thus, we tested independent and interactive effects of positive and negative parenting quality and child 5-HTTLPR genotype in prediction of: (1) concurrent social skills at school entry (child age 6), and (2) prospective changes in child social skills from age 6 to 9). We hypothesized that a model of differential susceptibility would be supported, such that positive and negative parenting would predict initial levels and growth in social skills, but only for SS children.

Method

Participants

Participants were 110 children and their families drawn from the Collaborative Family Study, a longitudinal study of family processes and child development based at three universities: University of California Los Angeles, University of California Riverside and Pennsylvania State University. Children with typical development or developmental delays were recruited from local schools and developmental service centers at age 3 or 5 years in Southern California (84%) and Central Pennsylvania (16%). Thus, the cognitive abilities of the present sample represent a wide range of functioning (IQ range: 45–137), with 78.2% of children falling within the normative IQ range (i.e., IQs > 70). Children with severe motor impairments (i.e., not ambulatory) or a diagnosis of autism spectrum disorder were excluded from study participation. Participating families represented varied socioeconomic backgrounds (29.8% with highest educational degree of high school diploma; 33.9% with household income below $50,000). Most child participants were Caucasian (58.8%), with

other race-ethnicities including Hispanic (17.5 %), African American (7.9%), Asian (1.8%), and Other (14.0%).

Procedures

All research procedures were approved by the Institutional Review Boards at the three participating universities. Most families were initially recruited at child age 3; the current study includes data from annual assessments from child age 5 to 9 years to assess social development across the early school years. Informed consent was obtained prior to each assessment. All procedures (detailed below) comply with APA Ethical Standards. Children completed a laboratory-based cognitive assessment at age 5 years. At age 6, children were observed with their mothers during a naturalistic interaction in the home. Mothers also completed standardized ratings of child social skills at child age 6, 7, 8 and 9 years. Families returned to the center at child age 13 years and children were asked to provide a DNA saliva sample. The present study included participants who provided a viable saliva sample at age 13, participated in a parent- child interaction at age 6 years, and had an assessment of social skills by mother report at a minimum of one assessment year (ages 6, 7, 8 or 9 years). Children that completed the DNA collection (n =110) did not differ from those who did not (n = 86) with respect to sex, race/ethnicity, positive and negative parenting (all $p > .10$). Those who completed the DNA collection had higher IQs and mean social skills scores at ages 6, 7, and 8 years (all $p < .05$), but not at 9 years. The vast majority of participants completed social skills ratings at all time points. The percentage of missing data for mother-reported social skills is as follows: 2.7% at age 6, 3.6% at ages 7 and 9, and 9.1% at age 8. Individuals with missing data at child ages 7, 8 or 9 [n = 16 (missing one (12) two (3) or three (1) time points)] did not differ from those without missing data regarding age 6 social skills, IQ, positive and negative parenting, mother education, family income, or genotype.

Measures

Cognitive functioning.—The Stanford-Binet IV (Thorndike et al., 1986) was administered to assess each child's cognitive ability at age 5 years. This widely used assessment instrument yields an IQ score with a normative mean of 100 and a standard deviation of 15. The Stanford-Binet IV demonstrates high internal consistency (Gluttig, 1989) and good evidence of validity (Thorndike et al., 1986). The assessment is particularly well suited to assess a wide range of functioning, as the administration requires that the examiner adapt starting points based on the child's developmental level.

Parenting.—Parenting quality was assessed during naturalistic, in-home observation of families at child age 6 years using the Parent-Child Interaction Rating System (PCIRS; Belsky, Crnic & Gable, 1995). Families were observed in the evening time for a period of 60 minutes. Coders observed for 10 minutes each, followed by a 5-minute scoring period. Ratings were then averaged across four 10-minute observation periods. Six aspects of parent behaviors were rated on a 5-point Likert scale (1 = not at all characteristic, 5 = highly or predominantly characteristic) that considered both the frequency and intensity of the expressed affect or behavior. Prior factor analyses (e.g., Fenning, Baker, Baker, & Crnic, 2007) have found these parenting dimensions to represent two factors: positive parenting and negative parenting. Positive parenting included positive affect expressed toward the child

(Positive Affect), sensitivity to the child's mood, interests and abilities (Sensitivity), the quality of attempts to scaffold learning opportunities (Cognitive Stimulation), and engagement with the child (Detachment, reverse coded). Negative parenting included expression of negative affect towards the child (e.g., anger, frustration, disappointment (Negative Affect) and attempts to impose an agenda or control the interaction with the child (Intrusiveness). Subscales within each dimension were converted to z-scores and combined to create positive and negative parenting composites. Coders were trained using videotapes of home observations and by attending live home observations with an experienced coder until reliability was established. Raters were considered reliable once they met at least 70% exact agreement with the expert coder and 95% agreement within one scale point. After meeting these criteria, coders checked 20% of their tapes with the master coder to ensure ongoing reliability. The average kappa for independent raters was 0.73, reflecting adequate levels of interrater reliability.

Genotype.—Saliva samples were collected from study participants using Oragene DNA Collection Kits (Ottawa, ON, Canada). Buccal cells were genotyped for 5-HTTLPR using standard primers, yielding products of 484 or 528 bp. The upstream single nucleotide variant rs25531 (A > G) was genotyped in addition to the S (14-repeated units) and L (16-repeated units) alleles, as the rs25531 Lg allele has been associated with decreased serotonin transporter transcription, similar to the S allele (Hu et al., 2006). To simplify the presentation of findings, diallelic labeling will be utilized (SS, SL, LL) in which L_G alleles are represented by S notation. SL was the most common genotype in the present sample (59.1%), followed by SS (22.7%) and LL (18.2%). Allele frequencies did not deviate from Hardy-Weinberg equilibrium [χ^2 (1)= 2.92, p= .09]. No group differences by genotype (SS v. SL/LL) were found for child sex, IQ, race/ethnicity, family income, or mother's level of education (all p > .10).

Social Functioning.—Child social skills were assessed at child ages 6, 7, 8 and 9 years via mother report on the Social Skills Rating System (SSRS-P; Gresham & Elliott, 1990). The parent form of the SSRS measures social skills in the following domains: Cooperation (e.g., helps with household chores, gives compliments), Assertion (e.g., joins group activities, starts conversations), Responsibility (e.g., asks for permission, reports accidents appropriately) and Self-Control (e.g., controls temper, receives criticism well). Parents report the frequency of behaviors on a 3-point Likert scale from 0 (Never) to 2 (Very Often). Each scale contains 10 items; two items contribute to two scales, yielding 39 total items. The SSRS Social Skills scale (SSRS-Total) yields standard score with a mean of 100 and standard deviation of 15. The SSRS-P demonstrated high internal consistency for the social skills Total score (α = .87), moderate to high internal consistency across subscales (α = .65 to .80), strong 4-week test-retest reliability (r = .80), and convergent validity with the Social Behavioral Assessment (Gresham & Elliott, 1990; Gresham, Elliott, Vance, & Cook, 2011).

Data Analytic Plan—Latent growth curve modeling (LGCM) was used to examine the independent and interactive effects of genotype and parenting quality in prediction of: (a) initial levels of child social skills at age 6 years and (b) growth in social skills from ages 6 to 9 years. LGCM allows for the examination of individual differences in social skills growth

over time, as well as predictors of growth. Estimates of latent intercept and slope were derived from mother-reported social skills at child ages 6, 7, 8 and 9 years. Predictors of latent intercept and slope were as follows: child 5-HTTLPR genotype, positive parenting, negative parenting, positive parenting x 5-HTTLPR interaction, and negative parenting x 5-HTTLPR interaction. Parenting variables were mean centered prior to creating interaction terms. Genotypes were dummy coded using a recessive model, such that children homozygous for the S or L_G alleles (SS = 1) were compared to SL/LL children (SL/LL = 0). This recessive model was employed based on meta-analytic evidence for the association of the SS genotype with sensitivity to the environment (vs. SL/LL genotypes; Pergamin-Hight et al., 2012) and evidence that SL and LL genotypes perform more similarly than the SS genotype with respect to GxE (e.g. 5-HTTLPR interactions with envrionmental adversity; Uher & McGuffin, 2008). Observed interactions for latent growth were probed per Preacher, Curran, and Bauer (2006), whereby simple slopes were computed at one standard deviation (SD) above and below the means of the parenting variables. When no significant GxEs were present ($p > .10$), interaction terms were dropped and main effects of parenting quality and child genotype were evaluated. Full information maximum likelihood (FIML) estimation was used to account for missing data. Child IQ and maternal education level were included as covariates in all models to control for their potential influence on parenting behavior and child social skills development. Further, although unlikely in this sample, population stratification, or the unequal distribution of alleles across different races, may threaten internal validity. Although chi-squared tests revealed no racial group differences in the distribution of 5-HTTLPR alleles (χ^2 (4) = 2.19, $p = .70$), African American children demonstrated significantly lower social skills than Caucasian children at assessment age 9 only ($F = 2.53$, $p < .05$). Thus, child race-ethnicity was covaried in all models.

Results

Descriptive statistics

Social skills did not differ by genotype at ages 6, 7 and 8 years. At age 9 years, the SL/LL group demonstrated greater levels of mother-reported social skills than the SS group 9 ($t = 2.40$, $p < .05$). No group differences were found for positive parenting ($t = 0.10$) or negative parenting ($t = -0.08$) by child genotype. Table 1 reports the means, standard deviations, and correlations for other key study variables. The mean levels of observed positive parenting (rated on a 5-point Likert scale) map onto “some” to “moderate” levels, while mean negative parenting levels fell between “minimal” to “some” negative parenting, as described in the PCIRS coding manual. Positive parenting was positively correlated with child total social skills across all assessment years, while negative parenting was not associated with child total social skills. Interestingly, observed positive and negative parenting were not significantly correlated in the present study ($r = .04$, $p = .64$). As anticipated, child IQ was positively associated with social skills at all times points, while mother education was positively associated with positive parenting and social skills at child age 6 only.

5-HTTLPR x parenting interactions predicting concurrent social skills

LGCMs for all models demonstrated adequate to good model fit for the sample size and number of variables assessed (Table 2). We first examined whether 5-HTTLPR x parenting

interactions predicted initial (age 6) child social skills (Table 2A). The 5-HTTLPR x positive parenting interaction predicted total social skills, such that there was a positive association between positive parenting and concurrent (age 6) social skills among SL/LL children (B = 4.86, p < .01), but not SS children (B = −2.70, p = .43). The 5-HTTLPR x negative parenting interaction did not predict total social skills.

To further probe the observed 5-HTTLPR x positive parenting interaction predicting total social skills, we examined this same interaction but in prediction of the individual social skills subdomains (cooperation, assertion, responsibility, self-control; Table 2A). We found that the observed interaction predicting total social skills was primarily driven by predictions of cooperation (B = −1.56, p = .04) and responsibility (B = −1.73, p = .02), although a positive parenting x 5-HTTLPR interaction also approached significance for self-control (B = −1.30, p = .07). Across these three subdomains, positive parenting was positively associated with initial social skills, but only for those with the SL/LL genotype (cooperation: $B_{SL/LL}$ = 1.20, p < .01, responsibility: $B_{SL/LL}$ = 0.83, p = .01, self-control: $B_{SL/LL}$ = 1.02, p < .01). No significant 5-HTTLPR x negative parenting interactions were found in prediction of initial social skills across subdomains. However, unexpected main effects of negative parenting were observed such that increased negative parenting was associated with higher assertion and responsibility at age 6 (Table 2A).

5-HTTLPR x parenting interactions predicting growth in social skills

Next, we examined whether 5-HTTLPR x parenting interactions predicted growth in child social skills from ages 6 to 9 years (i.e., slope; Table 2B). We observed an unexpected 5-HTTLPR x negative parenting interaction predicting growth in total social skills (Table 2B), whereby negative parenting was only marginally associated with more positive growth in social skills for SS children (B = 1.54, p = .07), but not SL/LL children (B = −.64, p = .13). Thus, although this interaction predicting growth in total social skills was significant, simple slopes by genotype revealed no significant effects. The 5-HTTLPR x positive parenting interaction did not predict growth in total social skills (Table 2B).

We also examined 5-HTTLPR x negative parenting in predicting subdomains of social skills. The 5-HTTLPR x negative parenting interaction predicted growth in child cooperation (Table 2B), such that negative parenting was *positively* associated with growth in cooperation skills for SS children (Bss = .42, p < .05), but not associated with growth in the SL/LL group ($B_{SL/LL}$ = −.09, p = .39). Simple slope tests were calculated at one SD above and below the mean of negative parenting for SS children. Of note, one SD above the mean equates roughly to “some” levels of negative parenting on the PCIRS (or a rating of 2.2 on a 1–5 Likert scale), while one SD below the mean equates to “minimal” levels of negative parenting (1.1 on a 1–5 Likert scale). At one SD above the mean of negative parenting, the cooperation slope for SS children did not differ from zero (B = .11, p = .70), while at one SD below the mean, the SS group demonstrated a significant decline in these skills (B = −.72, p = .01; Figure 1).

The 5-HTTLPR x *positive* parenting interaction significantly predicted child responsibility (Table 2B). Positive parenting was positively associated with growth in responsibility for SS children (B = .58, p < .01), but not SL/LL children (B = −.03, p = .73). Simple slope tests

were calculated at one SD above and below the mean of positive parenting for SS children (Figure 2). At one SD above the mean (i.e., “moderate” levels of positive parenting), SS children demonstrated a significant increase is responsibility skills ($B = .66$, $p = .01$). At one SD below the mean (i.e., “some” levels of positive parenting), SS children demonstrated a decline in responsibility skills ($B = -.50$, $p < .05$). No 5-HTTLPR x negative parenting interaction was found for child responsibility growth, though a main effect of negative parenting revealed that greater levels of negative parenting associated with declines in responsibility skills.

5-HTTLPR x parenting interactions did not predict growth in child assertion or self-control. For these subdomains, a main effect 5-HTTLPR emerged, such that SS children demonstrated greater relative declines in these domains of social skills across ages 6 to 9 years as compared to SL/LL children (see Figure 3). This same main effect of 5-HTTLPR was also found for the other social skills subdomains and total score. SL/LL children demonstrated significant *growth* in total social skills and responsibility skills, marginal growth in self-control, and no significant growth in cooperation or assertion. In contrast, SS children demonstrated significant declines in total social skills ($B = -1.61$, $p < .05$) and assertion ($B = -.51$, p = <.01), but not cooperation ($B = -.30$, ns), responsibility ($B = .08$, ns) or self-control ($B = -.21$, ns).

Discussion

This study is the first to test interactions between 5-HTTLPR and observed parenting behavior predicting *prospective* youth social skill development. While previous research has extensively examined 5-HTTLPR x environment interactions for child psychopathology and antisocial behavior, it is unclear how this putative GxE influences *prosocial* behavior. Findings revealed differential GxE effects by development timing and social skills domain, providing partial support for study hypotheses. Whereas the SL/LL group demonstrated associations between positive parenting and *concurrent* social skills at age 6, the SS group demonstrated relations between negative and positive parenting and prospective *growth* in specific aspects of social skills (cooperation and responsibility, respectively) from ages 6 to 9 years.

First, at age 6, observed positive parenting behavior was positively associated with social skills in the SL/LL group (but not SS group), contrary to study hypotheses. This finding suggests a dominant GxE effect for concurrent social skills, in which the dominant L allele, rather than the S allele, confers susceptibility to the environment. Several recent studies have found an L-allele driven effect (e.g., Little et al., in press), although findings are mixed (Weeland, Overbeek, Castro, & Matthys, 2015). Divergent findings have been attributed to differing GxE pathways by allele, in which either the S or L allele confers environmental susceptibility depending on the type of environment and developmental outcome assessed (see Weeland et al., 2015). For example, the S allele predicted heightened emotional and physiological reactivity as well as emergent irritability, aggression, and related psychopathology in negative contexts (e.g., Cicchetti, Rogosch, & Thibodeau, 2012). In contrast, L allele carriers demonstrated hyporeactivity to negative affect and punishment (Glenn, 2011) and attentional bias toward positive emotional stimuli (Fox, Ridgewell, &

Ashwin, 2009), which may mediate the link between positive parenting and adaptive social functioning for this group. However, given that the dominant GxE found was specific to age six social skills, we cannot rule out the role of passive gene-environment correlation (see Knafo & Jaffee, 2013). That is, parents of SL/LL children (who themselves are more likely to be L allele carriers) may demonstrate an attentional bias towards positive behavior and respond with greater levels of positive parenting. However, in the current study levels of positive and negative parenting did not differ by child genotype.

Given the dynamic changes in social development across childhood, and potential for bidirectional influences of parent and child behavior (Sameroff, 2009), a key aim of the present study was to characterize 5-HTTLPR x parenting interactions in the context of social skills *growth.* Overall, SS children demonstrated stronger associations between positive and negative parenting and growth in social skills than those with the SL/LL genotypes, consistent with study hypotheses, although this pattern was sensitive to the specific social skill domain assessed. For child cooperation, an unexpected direction of effect emerged such that *negative* parenting positively predicted relative growth in these skills for the SS group (but not the SL/LL group). Yet for the domain of child responsibility, *positive* parenting predicted more positive growth for those with the SS genotype only. Parsing apart these parenting-social skill associations aids our understanding of the specificity of GxE effects.

That negative parenting predicted relative growth in cooperation skills appears to diverge from prior evidence that the SS genotype confers risk for increased reactivity to stressful environments (Gotlib, Joormann, Minor, & Hallmayer, 2008). Yet, this discrepancy may relate to the outcomes assessed and absolute levels of negative parenting present. While several studies found that severe environments (i.e., maltreatment and institutionalization) more negatively impact children with the SS genotype (e.g., Cicchetti et al., 2012), the present study reflected normative levels of negative parenting in a community-based sample. Indeed, the mean levels of negative parenting observed (i.e., “minimal” to “some”) include mild negative affect and intrusive or controlling behavior. Previous literature is mixed regarding the impact of parents’ negative emotional expression on child social functioning, with multiple studies observing a positive relationship between low to moderate levels of parent negative affect and child social development (e.g. Valiente et al., 2004). Consistent with the negativity bias hypothesis (Vaish, Grossmann, & Woodward, 2008), these mildly negative parenting behaviors, as opposed to detached or neglectful parenting, may attract a child’s attention and increase his or her ability to learn from the emotional expression. This might be especially relevant for SS children who may display attentional preferences for negative emotions (Pergamin-Hight et al., 2012).

Further, the observed positive association of negative parenting among SS children also appeared to be specific to cooperative social behavior (e.g., helps with chores, does tasks without being asked). Similarly to child compliant behavior, cooperative behavior may be encouraged by the anticipation of parents’ negative affect (e.g., disappointment) and parents’ use of control strategies, particularly at modest levels (Kalb & Loeber, 2003). Thus, mild levels of negative parenting may be protective against risk for relatively poor development in this domain for SS children. This pattern of results suggests that the absolute levels of negative parenting in the present study may not have allowed for the formal testing of

differential susceptibility. Future studies may choose to include samples at higher risk for negative parenting (e.g. families with a history of child maltreatment, low SES). Further, given the unexpected direction of effect, results regarding 5-HTTLPR x negative parenting interaction should be considered exploratory in nature, and interpreted with caution until supported with replication.

We also found that positive parenting was positively associated with growth in responsibility skills (e.g., attends to adults, asks for permission) for SS children but not SL/LL children. Positive parenting, including parent engagement, sensitivity to child mood and developmental level, and scaffolding of learning opportunities, is associated with positive social functioning and broader positive developmental outcomes (Cassidy & Shaver, 2016). These parenting practices are thought to influence children's understanding of social relationships, particularly during the first 5 to 7 years of life (Belsky, Steinberg, & Draper, 1991). In the present study, positive parenting effects were specific to growth in child responsibility skills in the SS group, which may reflect the type of positive social behavior modeled by parents. Viewed in light of social learning theory (Bandura, 2012), positive parenting may be a manner of modeling social responsibility. For example, by noticing and responding sensitively to the child's mood and functioning, parents may be modeling guidelines for responsible social behavior such as considering others' feelings or perspectives before taking action. That this association was present for the SS group only coincides with meta-analytic and experimental evidence supporting the S allele as a marker of sensitivity to positive environments (e.g. Van IJzendoorn et al., 2012).

Finally, we examined developmental trends in social skills by genotype. Across all domains, SS children demonstrated greater relative decline in social skills than those with SL/LL genotypes. Parenting behavior moderates these effects for cooperation and responsibility, but not for assertion and self-control. Trends suggest that the SS genotype may be a marker of risky social *development,* consistent with previous research linking this variant to maladaptive social behavior in human and non-human animals (e.g. Stoltenberg et al., 2013; Bailey et al., 2015). Initial social functioning did not differ between groups, highlighting the importance of assessing genetic effects in the context of development. Heritability of certain behavioral phenotypes increase with age due to processes such as active or evocative gene-environment correlation and increases in gene expression across development (Bergen, Gardner, & Kendler, 2007), which may explain developmental changes in 5-HTTLPR-social skills associations. Findings suggests that 5-HTTLPR studies should continue to employ rigorous, repeated measures designs to accurately assess and interpret GxE effects in the context of development.

Findings should be interpreted in light of study limitations. First, the present study featured observational parenting measures; multi-method assessment including subjective ratings would likely enhance understanding of the specificity of gene-parenting interactions on social development. Further, the study lacked parent genetic data, which limited the ability to control for the potential effects of passive gene-environment correlation (Knafo & Jaffee, 2013). The sample size was also modest for this type of analysis and thus underpowered to detect effects of small magnitude; this might be particularly relevant when interactions were not observed, as genetic effects on human behavior tend to be small (Manuck & McCaffery,

2014). It will be important for larger, independent studies to test the reproducibility of results. Finally, the present study should be considered within the context of challenges faced by the field of candidate GxE research more broadly, including the potential for type I error and replication failure (Duncan & Keller, 2011). Indeed, GxE research remains a controversial field with some criticizing the use of sample sizes below 1,000 due to presumed very small effect sizes (Dick et al., 2015). These considerations are particularly poignant for findings with unexpected directions of effect, which should be interpreted as exploratory until supported with replication. In contrast, the majority of large-scale GxE studies lack rigorous assessment of the social environment and child phenotype (i.e., observational and repeated measures assessment). Therefore, it is important to balance these large-scale designs with more modestly-sized yet rigorously assessed samples, as repeated and observational measures increase power and may provide convergent evidence or elucidate developmental specificity in GxE.

The present study found that the 5-HTTLPR SS genotype conferred developmental sensitivity to parenting behavior, including effects to certain social domains. This evidence may help to maximize the benefit of psychosocial interventions by employing interventions that are targeted to the child's social needs and genetically-informed style of learning. If replicated, these findings suggest that parenting interventions targeting child cooperation skills may choose to teach healthy communication of parent negative emotions and limit setting, especially for families of SS children. Likewise, treatments promoting responsibility in these children may choose to focus more on increasing positive parenting skills (e.g., sensitivity, engagement). Overall, results suggest that the influence of parenting on social development varies by child genotype and holds meaningful implications for delivering targeted interventions to foster person-environment fit.

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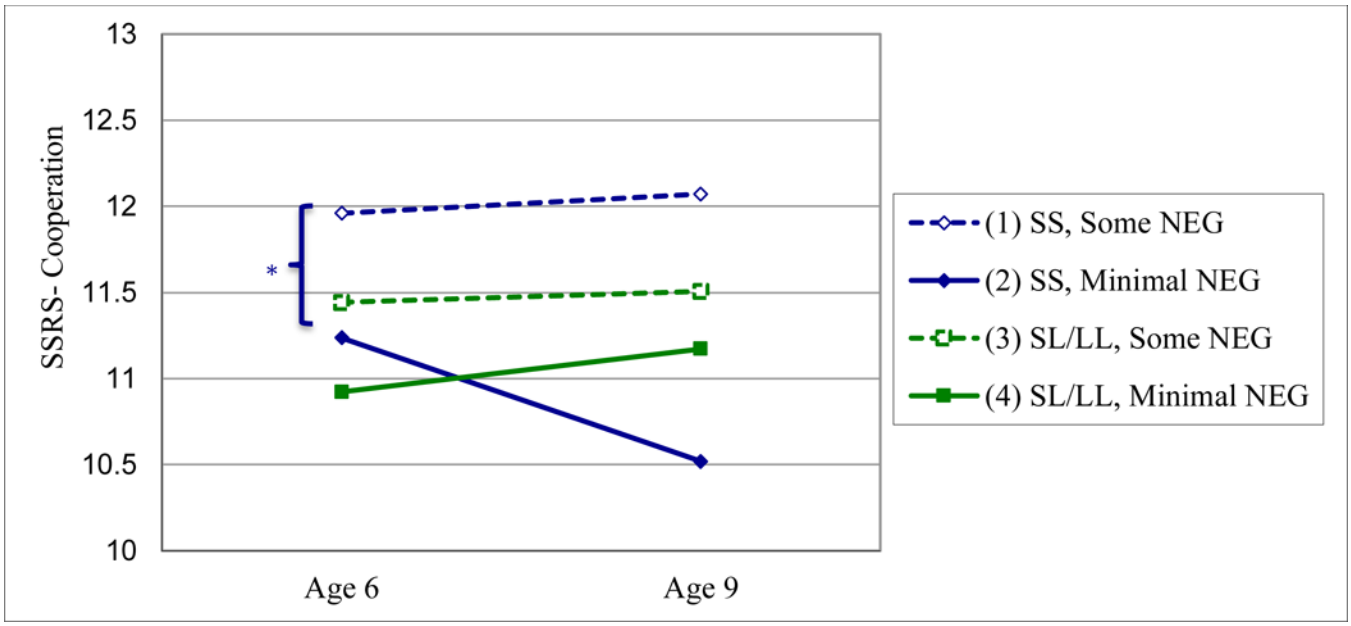

Figure 1.

5-HTTLPR x negative parenting interaction predicting growth in SSRS-Cooperation from child ages 6 to 9 years. SSRS: Social Skills Rating System. NEG: Negative parenting. Minimal: 1 SD below the mean; approximately equivalent to "minimal" negative parenting on the PCIRS (1.1 on a 1–5 Likert scale); Some: 1 SD above the mean; approximately equivalent to "some" negative parenting on the PCIRS (2.2 on a 1–5 Likert scale). Asterisk (*) reflects the significant simple effect of negative parenting in the SS group.

*p<.05

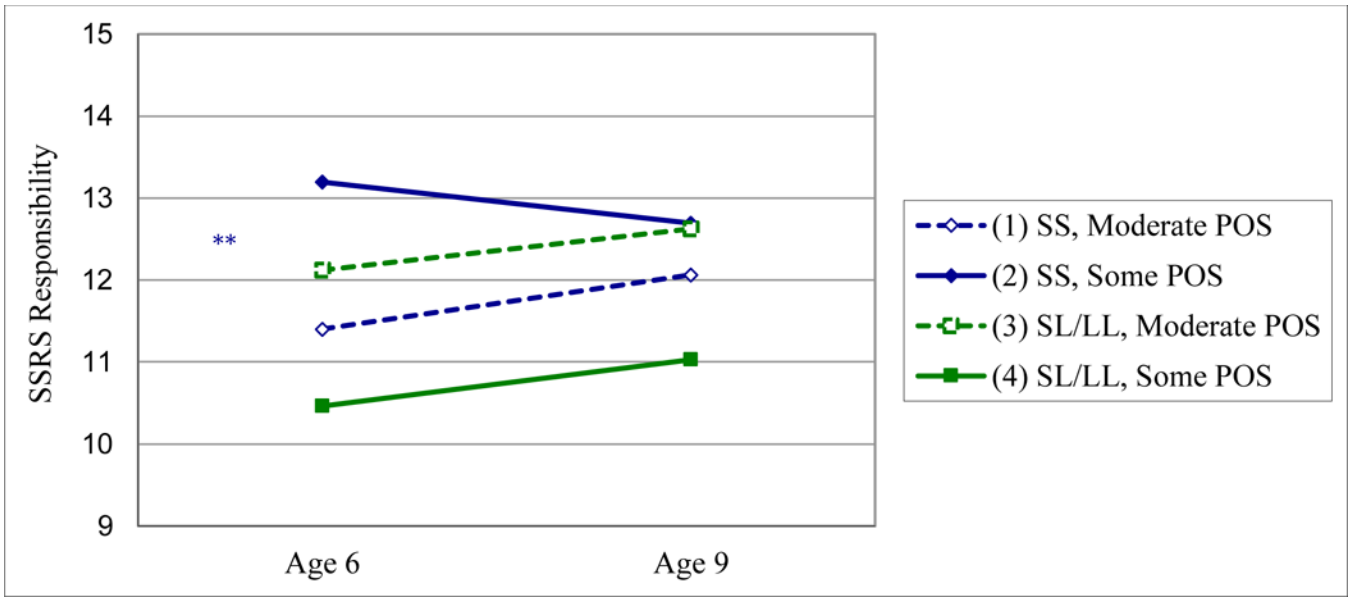

Figure 2.

5-HTTLPR x positive parenting interaction predicting growth in SSRS-Responsibility from child ages 6 to 9 years. SSRS: Social Skills Rating System. POS: Positive parenting. Moderate: 1 SD above the mean; approximately equivalent to “moderate” levels of positive parenting on the PCIRS (3.1 on a 1 −5 Likert scale). Low: 1 SD below the mean; roughly equivalent to “some” levels of positive parenting on the PCIRS (2.2 on a 1–5 Likert scale). Asterisk (*) reflects the significant simple effect of positive parenting in the SS group. **p<.01

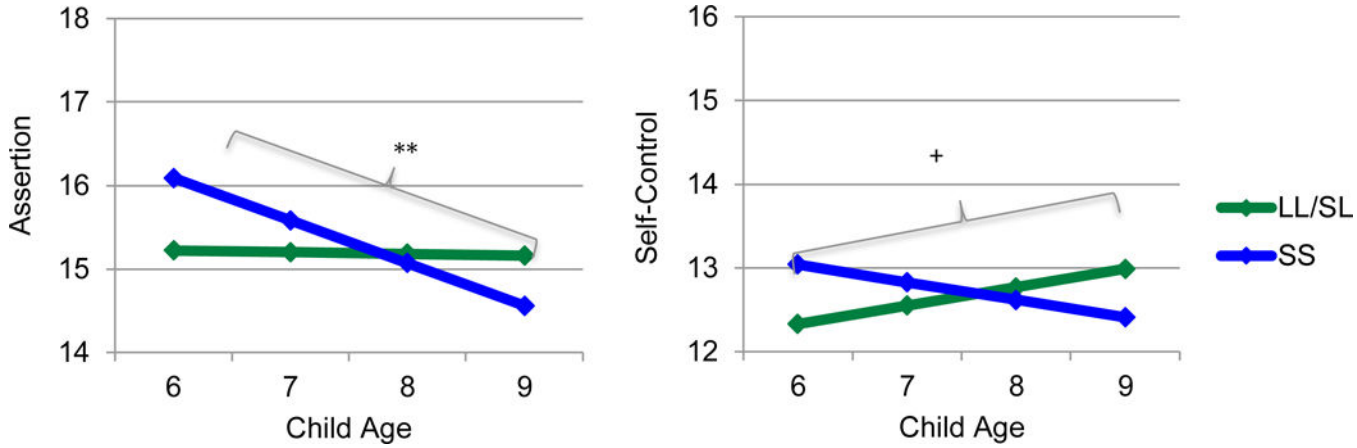

Figure 3.

Main effect models of 5-HTTLPR on Social Skills Rating System (SSRS) scales: Assertion (5-HTTLPR: $B = -.49$, $p < .01$) and Self-Control (5-HTTLPR: $B = -.43$, $p < .05$). **p<.01,+p>.10

Table 1

Descriptive statistics and correlations for key study variables (N = 110)

Variable (age)	Mean (SD)	1	2	3	4	5	6	7	8	9
1. Child Sex[a]	0.55	1								
2. IQ (5)	90.4 (23.4)	−.02	1							
3. Mother education[b]	15.2 (2.3)	.09	.33***	1						
4. Positive parenting (6)[c]	2.45 (0.6)	−.12	.12	.36***	1					
5. Negative parenting (6)[c]	1.66 (0.5)	.09	−.10	.14	.06	1				
6. SSRS-P (6)[d]	95.9 (18.4)	.16	.50***	.23*	.27**	.09	1			
7. SSRS-P (7)[d]	98.7 (17.2)	.01	.44***	.15	.31**	.12	.83***	1		
8. SSRS-P (8)[d]	98.7 (17.7)	−.01	.40***	.10	.26**	.02	.80***	.85***	1	
9. SSRS-P (9)[d]	99.6 (17.9)	.11	.41***	.02	.28**	.01	.73***	.77***	.83***	1

Note. SSRS-P: Social Skill Rating System, Parent report.

[a] 1= boys, 0 = girls

[b] Mother education is assessed as the highest grade completed per mother report.

[c] Parenting values depicted in the table are the average score across parenting subindices, reported on a 1–5 (high) Likert scale.

[d] Social Skills: SSRS-Social Skills Total standard score, mother report; n = 107 (age 6), n = 106 (age 7), n = 100 (age 8), n = 106 (age 9).

* p<.05

** p.>01

*** p<.001

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Table 2

Latent growth curve models (LCGMs) predicting domains of mother-reported social skills from child ages 6 to 9 years (N= 110)

	SSRS-Total			SSRS-Cooperation			SSRS-Assertion			SSRS-Responsibility			SSRS- Self-Control		
	B	**SE (*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**
A. Intercept	**95.01*****	**1.97**	**5.68**	**11.18*****	**0.41**	**3.66**	**15.22*****	**0.36**	**5.23**	**11.29*****	**0.40**	**3.36**	**12.32*****	**0.38**	**4.22**
Child IQ	**0.41*****	**0.07**	**0.02**	**0.06*****	**0.01**	**0.44**	**0.06*****	**0.12**	**0.44**	**0.09*****	**0.01**	**0.60**	**0.07*****	**0.01**	**0.57**
Race 1[a]	−6.09	4.01	−0.36	−0.89	0.83	−0.14	**−1.86***	**0.74**	**−0.31**	−0.74	0.82	−0.11	−0.58	0.78	−0.10
Race 2[a]	0.12	2.29	0.01	−0.19	0.47	−0.05	0.08	0.42	0.02	−0.21	0.47	−0.05	−0.14	0.44	−0.04
Race 3[a]	3.24	3.07	0.19	0.74	0.62	0.14	1.09	0.57	0.22	0.47	0.63	0.08	0.47	0.59	0.09
Mother Education[b]	−0.49	0.70	−0.03	−0.21	0.14	−0.16	0.04	0.13	0.03	−0.05	0.14	−0.03	−0.21	0.14	−0.16
Positive Parenting	**4.86****	**1.65**	**0.29**	**1.20*****	**0.14**	**0.39**	0.41	0.30	0.14	**0.83***	**0.33**	**0.25**	**1.02****	**0.32**	**0.35**
Negative Parenting	2.69	1.69	0.16	0.09	0.11	0.09	**0.62***	**0.31**	**0.21**	**0.87***	**0.34**	**0.26**	0.35	0.33	0.12
5-HTTLPR[c]	3.81	3.36	0.23	0.42	0.68	0.06	0.87	0.62	0.13	1.01	0.68	0.13	0.69	0.65	−0.10
GxE: Positive	**−7.56***	**3.72**	**−0.45**	**−1.56***	**0.76**	**−0.22**	−0.77	0.68	−0.11	**−1.73***	**0.75**	**−0.22**	−1.30†	0.73	−0.19
GxE: Negative	−0.63	3.30	−0.04	0.10	0.67	0.02	−0.05	0.60	−0.01	−0.24	0.67	−0.04	−0.58	0.64	−0.10
R-square (S.E.)	***0.38******	***(.08)***	--	***0.27*****	***(.08)***	--	***0.31******	***(.08)***	--	***0.43******	***(.08)***	--	***0.36******	***(.09)***	--
B. Slope	**1.24***	**0.50**	**0.40**	0.16	0.13	0.20	−0.02	0.10	−0.03	**0.54****	**0.11**	**0.91**	0.21†	0.12	0.44
Child IQ	−0.03	0.02	−0.01	−0.01*	0.00	−0.25	0.00	0.00	−0.12	0.00	0.00	0.05	−0.01	0.00	−0.29
Race 1[a]	−1.77†	1.04	−0.56	0.08	0.26	0.05	−0.27	0.21	−0.23	−0.33	0.22	−0.27	−0.17	0.25	−0.17
Race 2[a]	1.34	0.58	0.43	0.25†	0.15	0.23	**0.24***	**0.12**	**0.32**	0.15	0.13	0.19	0.12	0.14	0.19
Race 3[a]	0.88	0.78	0.28	−0.05	0.19	−0.03	−0.10	0.16	−0.10	0.14	0.17	0.14	0.24	0.19	0.29
Mother Education[b]	**−0.53****	**0.18**	**−0.17**	**−0.09***	**0.04**	**−0.26**	**−0.10****	**0.04**	**−0.38**	−0.07†	0.04	−0.27	0.09†	0.04	−0.41
Positive Parenting	0.20	0.42	0.06	0.01	0.11	0.02	0.07	0.03	0.11	−0.03	0.09	−0.05	0.01	0.10	0.03
Negative Parenting	−0.64	0.42	−0.21	−0.09	0.04	−0.12	−0.11	0.09	−0.19	**−0.19****	**0.09**	**−0.33**	0.00	0.09	−0.01
5-HTTLPR[c]	**−2.85****	**0.87**	**−0.91**	**−0.46***	**0.21**	**−0.24**	**−0.49****	**0.18**	**−0.36**	**−0.46***	**0.19**	**−0.32**	**−0.43***	**0.21**	**−0.38**
GxE: Positive	1.21	0.95	0.39	−0.27	0.24	−0.14	0.22	0.20	0.16	**0.62****	**0.20**	**−0.44**	0.02	0.23	−0.02

	SSRS-Total			SSRS-Cooperation			SSRS-Assertion			SSRS-Responsibility			SSRS- Self-Control		
	B	**SE (*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**	***B***	**SE(*B*)**	**Beta**
GxE: Negative	**2.18** *	**0.93**	**0.70**	**0.51** *	**0.23**	**0.32**	0.35 †	0.19	0.30	0.09	0.20	0.08	0.33	0.23	−0.33
R-square (S.E.)	***0.59*** **	***(.17)***	--	***0.41*** **	***(.13)***	--	***0.50*** *	***(.21)***	--	***0.52*** *	***(.23)***	--	*0.69*	*0.46*	--

Note. Statistically significant results are bolded for emphasis. In models with no significant GxEs (p <.10), these terms were dropped to examine main effects. Main effect coefficients represent the final model. GxE: Gene-environment interaction. *B* = unstandardized coefficient. *SE(B)* = standard error of unstandardized coefficient. β = standardized coefficient. Model fit indices were as follows: SSRS-Total: $\chi^2(25) = 39.0$, p = .04, RMSEA = .07, CFI = .97; SSRS-Cooperation: $\chi^2(25) = 47.2$, p=.01, RMSEA = .09, CFI = .93; SSRS-Assertion: $\chi^2(25) = 31.8$, p =.17, RMSEA = .05, CFI = .98; SSRS-Responsibility: $\chi^2(25) = 34.5$, p=.10, RMSEA = .06, CFI = .98; SSRS-Self-Control: $\chi^2(25) = 26.7$, p=.37, RMSEA = .03, CFI = 0.99. Italics used to distinguish overall R^2 from predictor variable coefficients.

[a] Race is effect coded.

[b] Mother education is assessed as the highest grade completed per mother report.

[c] Genotype for 5-HTTLPR was coded SS =1, SL/LL =0.

† p<.10,

* p<.05,

** p<.01,

*** p<.001.