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How and Why Does the 5-HTTLPR Gene Moderate Associations Between Maternal Unresponsiveness and Children's Disruptive Problems?

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Abstract

This study tested the 5-HTTLPR gene as a moderator in the relation between maternal unresponsiveness and child externalizing symptoms in a disadvantaged, predominantly Black sample of 201 two-year-old children and their mothers. Using a multi-method, prospective design, SEM analyses indicated that maternal unresponsiveness significantly predicted increases in externalizing symptoms two years later only for children possessing the LL genotype. Moderation was expressed in a “for better” or “for worse” form hypothesized in differential susceptibility theory. In examining why the risk posed by maternal unresponsiveness differed across the 5-HTTLPR polymorphism, mediated moderation analyses showed that children's angry reactivity to maternal negativity partly accounted for the greater susceptibility of homozygous L carriers to variations in maternal unresponsiveness.

Although early stress in the family has been consistently documented as a significant risk factor for the development of psychopathology, the magnitude of family adversity as a predictor of individual differences in psychological problems is commonly modest (Repetti, Robles, & Reynolds, 2011; Taylor, Way, & Seeman, 2011). To account for variability in the outcomes of children, conceptualizations have designated genes as central sources of individual differences in the risk posed by family adversity (e.g., Belsky & Pluess, 2009; van Goozen, Fairchild, Snoek, & Harold, 2007). As a pivotal gene, the serotonin transporter (5-HTTLPR) polymorphism has been shown to moderate associations between early family adversity and mental health outcomes (e.g., Karg, Burmeister, Shedden, & Sen, 2011). However, gaps remain in identifying the form of moderation and the underlying mechanisms that account for the moderating role of 5-HTTLPR. Because research on the moderating effects of 5-HTTLPR gene has largely been limited to White adolescents or adults, little is known about the interplay between parenting and the 5-HTTLPR genotype in predominantly Black samples of younger children. Moreover, with a primary aim of cataloguing the moderating effects of genes, research has yet to systematically identify the mechanisms that account for why 5-HTTLPR modifies the path between family adversity and mental health

(Belsky & Pluess, 2009). To address these gaps, this paper tests the 5-HTTLPR genotype as a moderator of links between parenting difficulties and disruptive behavior problems in a mostly Black sample of preschool children, with the objective of characterizing the form of moderation and its underlying mediating mechanisms.

As a framework for organizing our aims, Figure 1 provides a conceptual model of the hypothesized interplay between early family adversity and the 5-HTTLPR gene in predicting child externalizing symptoms. As depicted by Path 1 of Figure 1, maternal unresponsiveness is regarded as a particularly deleterious form of early adversity for children. Within the broad class of parenting difficulties that have been identified as risk factors for disruptive behavior problems during early childhood (Campbell, 2006), maternal unresponsiveness has been conceptualized as a highly potent dimension of adversity (Rothbaum & Weisz, 1994). Although conceptual definitions vary in the literature, maternal unresponsiveness is commonly characterized by mothers' difficulties in identifying and attending to the emotional needs, psychological states, and competencies of their children. Children who experience maternal unresponsiveness exhibit heightened psychological problems, particularly in the form of externalizing symptoms. For example, maternal unresponsiveness has been prospectively linked with children's disruptive externalizing symptoms (Stams, Juffer, & van IJzendoorn, 2002; Wakschlag & Hans, 1999). However, in spite of its potency as a risk factor, many children exposed to maternal unresponsiveness do not exhibit high levels of externalizing problems (Rothbaum & Weisz, 1994). Thus, these findings beg the question of whether genetic factors moderate associations between unresponsive parenting and child disruptive problems.

Toward identifying genetic sources of variability in children's vulnerability to maternal unresponsiveness, Path 2 of Figure 1 further proposes that the pathway between maternal unresponsiveness and child externalizing problems is moderated by allelic variation in the 5-HTTLPR genotype. In playing a significant role in regulating serotonin activity in the brain, the 5-HTTLPR gene encodes the serotonin transporter (5-HTT) and its primary function of removing serotonin from nerve synapses to store for subsequent redistribution (Bevilacqua & Goldman, 2011). Relative to the long (L) allele, the short (S) allele of the 5-HTTLPR gene is associated with diminished efficiency of serotonin transporters in regulating levels of serotonin in the brain (e.g., Heinz, Mann, Weinberger, & Goldman, 2001). Although not always consistent, some findings in the literature have shown that early adversity is a significant predictor of psychopathology for individuals carrying the S allele (Cicchetti, Rogosch, & Thibodeau, 2012; Karg et al., 2007; van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012).

Genetic susceptibility, itself, can vary in its form depending on the specific characteristics of the genes and environments under investigation. According to diathesis-stress models, "susceptibility" is expressed as a diathesis or disposition toward experiencing psychological problems that increase disproportionately in the context of high levels of environmental adversity. Conversely, differential susceptibility frameworks propose that genetic "susceptibility" does not simply reflect heightened sensitivity to adverse socialization contexts, but also greater plasticity to supportive rearing environments (Belsky & Pluess, 2009). Because genetic "susceptibility" is characterized by greater plasticity in a "for better

or for worse” fashion, differential susceptibility theory is novel in its hypothesis that children with the “susceptibility” attribute actually fare better than children without the attribute in contexts of supportive parenting. Although earlier findings on 5-HTTLPR as a moderating factor were commonly explained in the context of diathesis-stress models, careful re-analysis suggests that many of these findings are more consistent with differential susceptibility theory. That is, some of the studies correspond with the thesis that the S allele is a plasticity attribute that eventuates in significantly poorer and better outcomes in adverse and supportive contexts, respectively (Belsky & Pluess, 2009; van IJzendoorn et al., 2012). However, at this early stage of expanding the scope of $G \times E$ to a multiplicity of outcomes, little is known about the nature of the moderating effects of the 5-HTTLPR gene in the prediction of externalizing symptoms. Therefore, our analysis of 5-HTTLPR as a moderator will directly test the relative viability of the diathesis-stress and differential susceptibility frameworks (Roisman et al., 2012).

From a process-oriented perspective, cataloging the moderating effects of 5-HTTLPR is only one step in the process of understanding how and why associations between maternal unresponsiveness and child externalizing symptoms vary as a function of genetic factors. Thus, a key remaining task is to identify mechanisms that are more proximal products of the interplay between unresponsiveness and the 5-HTTLPR genotype and, in turn, act as precursors to trait-like patterns of disruptive behavior problems. According to the psychobiological model formulated by van Goozen and colleagues (2007), disinhibited emotional reactivity is regarded as a candidate mechanism that mediates the moderating role of the 5-HTTLPR in associations between early childhood adversity and externalizing problems. Therefore, the putatively stronger pathway between maternal unresponsiveness and disruptive behavior problems among children carrying the S allele may be due to their greater emotional sensitivity and reactivity to subsequent negative events (van Goozen et al., 2007). In illustrating the first link in this hypothesis, Path 3 in Figure 1 proposes that exposure to early adversity is associated with greater negative emotional reactions for children who exhibit diminished serotonergic functioning (Carver, Johnson, & Joormann, 2008; van Goozen et al., 2007). In support of this prediction, the S allele of 5-HTTLPR has been associated with greater physiological indices of fear and stronger reactivity to aversive stimuli (see Bevilacqua & Goldman, 2011). Moreover, although the relation between 5-HT and externalizing problems is inconsistent (Li & Lee, 2010), some studies have reported that depletion of amino acid precursors of 5-HT are related to greater negative emotional responses to stress (see Carver et al., 2008).

In the second proposed link in the mediational cascade (van Goozen et al., 2007), increasingly intense and prolonged forms of negative emotionality resulting from the interaction between 5-HTTLPR and maternal unresponsiveness are proposed to crystallize into broader forms of externalizing problems over time. Although the theory does not offer precise guidance on the nature and consequences of specific forms of negative emotion, other work highlights the value of distinguishing between angry and distressful forms of emotional reactivity in the context of family interactions. For example, the integrative model of emotion specifically distinguishes between angry and distress responses in understanding the development of children’s externalizing problems (Snyder, Scherpfman, McEachern, & DeLeeuw, 2010). Angry, hostile responses in family interactions are specifically regarded

as markers of a broader coercive process that serves as a training ground for the development of a protracted antisocial pattern characterized by aggression, antagonism, and defiance. In contrast, vulnerable affect characterized by fear and distress are proposed to reduce children's subsequent tendencies to exhibit impulsive, reflexive forms of aggression and overt defiance. Consistent with this hypothesis, Snyder and colleagues (2010) reported that children's angry reactivity in parent-child interactions predicted growth in oppositional forms of externalizing symptoms during the early elementary school years. Conversely, fearful reactivity during parent-child interactions was associated with diminished externalizing problems over time. In drawing on this earlier work, we specifically examine the relative viability of angry and distress responses to maternal negative affect as potential mediators of the hypothesized moderating role of 5-HTTLPR in the prospective association between maternal unresponsiveness and child externalizing symptoms.

Despite some evidence indicating that the S allele may confer susceptibility to negative emotional reactivity and psychopathology in the face of maternal unresponsiveness, models have increasingly underscored that the functional properties of 5-HTTLPR may vary across forms of psychological adjustment, racial characteristics, and developmental periods (e.g., Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). First, studies of whether the vulnerability of S allele carriers to early life adversity extends beyond the prediction of internalizing symptoms have yielded contradictory results. For example, research has reported that childhood adversity is a significant predictor of externalizing problems (e.g., conduct problems, binge drinking) only for homozygous L carriers of the 5-HTTLPR gene (e.g., Laucht et al., 2009; Sadeh et al., 2010; Sulik et al., 2012). Second, G $\times$ E research has primarily examined the 5-HTTLPR gene in samples of adolescents and adults. Highlighting the potential differences in the operation of the serotonin system in the first part of the life span, documented associations between markers of serotonergic functioning and aggression identified during later periods of the life span do not readily hold in juvenile samples (e.g., Anderson, 2003). Third, relying on predominantly White samples has hindered progress in identifying the generalizability and specificity of 5-HTTLPR as a moderator. For example, a meta-analysis revealed that S allele served as a plasticity moderator only in samples that were comprised primarily (> 80%) of White children (van IJzendoorn et al., 2012). In highlighting the LL genotype as a potential plasticity allele, other research findings suggest that the LL allele for Black samples may function in a similar way to the S allele in White samples by virtue of its association with diminished serotonin activity (e.g., Williams et al., 2003), heightened physiological reactivity to stress (Williams et al., 2008), and vulnerability to internalizing symptoms (e.g., Anderson & Mayes, 2010). Collectively, these results raise the possibility that the LL allele may actually confer psychobiological plasticity in the development of externalizing problems in samples of younger, predominantly Black children.

In summary, our study is designed to test a process model of why the 5-HTTLPR gene moderates associations between maternal unresponsiveness and childhood externalizing problems (see Figure 1). Drawing from a literature examining internalizing symptoms as outcomes in primarily White samples of adolescents or adults, one plausible hypothesis is that the S allele magnifies associations between maternal unresponsiveness and child externalizing problems by increasing children's emotional reactivity in stressful parental

events. However, potential differences in effects across child race, psychological sequelae, and developmental periods raise the possibility that the 5-HTTLPR gene may moderate developmental pathways among maternal unresponsiveness, children's emotional reactivity, and their behavior problems in a qualitatively different manner. In our mostly Black sample of preschool children, an alternative plausible hypothesis is that the maternal unresponsiveness may be a particularly strong predictor of externalizing symptoms for children possessing the 5-HTTLPR-LL genotype. In addressing the "mediational" component of our mediated-moderation model, further support for this prediction would be garnered if children's angry or distress reactivity partially explained why LL carriers evidenced greater susceptibility to maternal unresponsiveness. To rigorously test the relative utility of these complementary hypotheses, we utilized a multi-method, prospective approach to examine whether the interaction between maternal unresponsiveness and the 5-HTTLPR genotype predicted change in preschooler externalizing problems over a two year period by increasing their emotional reactivity to maternal anger.

Method

Participants

Participants included 201 two-year-old children and their mothers who were part of a project on children's coping and functioning in the context of family stress. Due to the focus of the project on understanding differences in how children cope with significant family and social adversity, a two-step recruitment procedure was implemented. In the first step, we recruited participants through agencies serving disadvantaged families in a moderately-sized metropolitan area in the Northeast, including Women, Infants, and Children, Temporary Assistance to Needy Families rosters, and the county court system. In the second step, we administered screening procedures to insure that roughly equal proportions of participating mothers experienced (a) no violence, (b) mild to moderate physical violence, and (c) severe physical violence in the interpartner relationship. Median annual income for households was \$18,300 (US) per year and a substantial minority of mothers (30%) did not complete high school. Most families were impoverished according to the US Federal Guidelines (99.5%). The mean age of the children was 26 months ($SD = 1.69$), with 44% of the sample consisting of girls ($n = 92$). Most participants were Black (56%), followed by smaller numbers of family members who identified as White (23%), Latino (11%), Multi-Racial (7%), and "Other" (3%). The retention rate across the three annual waves was 87%. To test for selective attrition, we conducted statistical comparisons between the mother-child dyads that participated through the third measurement occasion and dyads that dropped out during the course of the study along 22 primary variables, covariates, and demographic factors at Wave 1. No significant differences were identified in the analyses.

Procedures and Measures

Mothers and children visited the laboratory at a research center on multiple occasions at each of three annual waves beginning when children were two years old. The Institutional Review Board at the research site approved all research procedures prior to conducting the study.

Diminished maternal responsiveness—Maternal unresponsiveness was assessed using maternal questionnaires and observational ratings of mother-child interactions during Wave 1. For the observational procedures, coders rated unresponsiveness within a parent-child problem-solving paradigm (Sroufe, Egeland, Carlson, & Collins, 2005). The procedural battery consisted of four tasks (e.g., puzzles, shape sorters) that were each five minutes in duration and designed to be difficult for the children to solve without the assistance of an adult. Coders rated maternal unresponsiveness in the interaction episode using the Harsh, Insensitive Parent-Centered, and Warmth-Support scales from the Iowa Family Interaction Rating Scales (IFIRS; Melby & Conger, 2001). Global ratings for each scale ranged from (1) “Not at all characteristic” to (9) “Mainly characteristic.” The Harsh scale assessed maternal hostile, rejecting, and antagonistic behaviors directed toward the child during the interaction, while the Insensitive Parent-Centered code captured maternal difficulties identifying and attending to child needs, psychological states, and competencies. As the final code, Warmth was reverse scored so that higher ratings reflected lower maternal affirmation, encouragement, and caring. To evaluate interrater reliabilities, a second coder independently rated a subsample (24%) of the mother-child interactions. One-way random intraclass correlation coefficients (ICC) for the three codes ranged from .83 and .90.

For the questionnaire assessment, mothers completed the Lack of Empathy Toward Children’s Needs Scale from the Adult-Adolescent Parenting Inventory (AAPI; Bavolek & Keene, 1984). The scale captures parental unresponsiveness to the developmental needs of young children (e.g., eight items; “Young children who feel secure often grow up expecting too much.”). Internal consistency for the Lack of Empathy scale was good ($\alpha = .82$). The three observational ratings and the AAPI scale were standardized and aggregated into a single composite of maternal unresponsiveness for the analyses (composite $\alpha = .82$).

Serotonin genotype—Trained research assistants obtained DNA samples from children by collecting buccal cells with Epicentre Catch-All Collection Swabs. Using the conventional method, DNA was prepared for polymerase chain reaction (PCR) amplification through extraction with the Epicentre Buccal Amp DNA Extraction Kit. DNA was whole-genome amplified using the Repli-g kit (Qiagen, Chatsworth, CA., Catalog No. 150043) per the kit instructions to ensure availability of data over the long term for this sample. Amplified samples were then diluted to a working concentration. If a genotype for a gene could not be determined after the first run, then it was repeated up to four times. If the null result persisted, then a genotype was not assigned to that individual. The call rate, based on the 192 children who provided DNA samples, was 99.5% for 5-HTTLPR. All DNA samples were genotyped in duplicate for quality control. Human DNA from cell lines was purchased from Coriell Cell Repositories for all representative genotypes in duplicate and genotypes were confirmed by sequencing using DTCS chemistry on an ABI 3130×1. These and a no template control were run alongside study samples representing 9% of the total data output. Any samples that were not able to be genotyped to a 95% or greater confidence level were repeated under the same conditions.

Samples were genotyped for fragment length polymorphisms of 5-HTTLPR with Hot Star Taq PCR Mix (Qiagen, Catalog No. 203205) and previously described primers (Gelernter, Kranzler, & Cubells, 1997), followed by fragment analysis using a CEQ8000 (Beckman-

Coulter, Inc., Fullerton, CA). Although genotypes with one or two short (S) alleles of the 5-HTTLPR gene are generally associated with lower transcription and function of 5-HTT (Bevilacqua & Goldman, 2011) than genotypes with two long (L) alleles, research identifying an A>G substitution in a snp upstream from the promoter region has shown that L_G functions more similarly to the S allele than the L_A in its regulation of serotonin (Reimold et al., 2007). Therefore, following prior procedures (Mileva-Seitz et al., 2011), we compared children with genotypes containing at least one copy of S or L_G with the homozygote L genotype consisting of two copies of L_A. Consequently, the L_AL_A genotype (28%; $n = 54$) was contrasted with a single group of genotypes that function in a similar way to the S allele (23% L_AL_G, 26% L_AS, 3% L_GL_G, 10% L_GS, 9% SS). For quantification in analyses, the functional heterozygote S and homozygote L carriers were assigned values of -1 and +1, respectively. The genotype distribution for 5-HTTLPR was in Hardy-Weinberg equilibrium $\chi^2(1, N = 191) = 1.94, p = .16$.

Child emotional reactivity—During the first wave, children and their mothers participated in the Simulated Phone Argument Task (SPAT) to assess child emotional reactivity to angry maternal expressions (Davies, Sturge-Apple, Winter, Cummings, & Farrell, 2006). In this procedure, children witnessed live simulation in which the mother engaged in an angry exchange and subsequent amicable resolution with her intimate partner over the telephone. Each exchange lasted one minute and was followed by three-minute free periods. The conflict script revolved around a relatively trivial disagreement regarding in which the mother conveys mild frustration and anger toward the partner for failing to complete a requested chore. For the simulated resolution, mothers were asked to communicate a moderate level of understanding and caring in her tone of voice. Although the simulations indicated to the child that the mothers' partner was on the other end of the phone, an experimenter was actually on the phone feeding the mother the lines from the script. To insure displays of desired emotion, mothers listened to a standard, audiotaped sample of the script and practiced the lines until they were able to accurately convey the content and affective tone of the exchanges. In conveying script over the phone, the experimenter also simulated the emotional tone and intensity for mothers to emulate. Supporting its validity, emotional reactivity codes from the SPAT have been linked with children's experiences with family discord and psychological problems (e.g., Davies et al., 2006).

SPAT video records were subsequently coded by trained raters to obtain three indicators of angry and distress reactivity to maternal angry expressiveness. First, judges coded for the presence (1) and absence (0) of behavioral dimensions of child distress and anger during four intervals of the SPAT: (a) angry exchange, (b) post-anger, (c) resolution, and (d) post-resolution periods. Specific dimensions of distress reactivity included: (a) *anxious facial expressions* – facial expressions indicating anxiety or worry (e.g., eyebrows raised and drawn together, staring wide-eyed); (b) *anxious postures or gestures* – apprehensive gestures or postures (e.g., biting fingernails, repeated grooming gestures); (c) *sad or dysphoric facial expression* – facial signs of sadness (e.g., pouting lips, drawing up the inner corner of eyebrows); (d) *dysphoric posture* – dejected postural displays (e.g., slumping); (e) *sulking* – sulking or whining. Angry reactivity codes consisted of: (a) *angry facial*

expressions: facial signs of anger and frustration (e.g., eyebrows pushed downward and together); (b) *dominant posturing*: angry, threatening postural displays (e.g., up-tilted chin); (c) *controlling behavior*: protesting in an angry, coercive manner; (d) *behavioral dysregulation* – behaviors with a disorganized quality indicative of dysregulated anger (e.g., uncontrolled screaming); (e) *triangulation* – behaviors reflecting an alliance with one parent against the other parent. Indicators for the two forms of negative emotional reactivity were summed across the four SPAT intervals to generate behavioral *tallies* of distress and anger reactivity. Two coders independently rated 21% of the videos to assess interrater reliability. ICCs were .86 for distress reactivity tally and .96 for the angry reactivity tally.

Second, coders provided *specific ratings* of children's distress and hostility along seven point scales based on the multiplicity, intensity, quality, and organization of child negative affect during the entire SPAT exchange. Distress was defined as child expressions of fear, anxiety, and emotional upset, whereas hostility was characterized by anger, aggression, and irritation. At one extreme, ratings of "none" (0) were designated for children who displayed no indications of the specific form of emotional reactivity. Conversely, "high" ratings (6) reflected that children were having substantial problems regulating their negative reactivity. ICCs, reflecting reliability between the same two raters, were .85 for distress reactivity and .91 for hostile reactivity.

Third, *molar ratings* of children's distress and angry reactivity were coded based on the course and nature of child behaviors during the SPAT. Ratings reflected the correspondence between the children's behaviors and prototypical descriptions of distress and anger reactivity to the maternal exchange along five-point scale ranging from "not at all characteristic" (1) to "highly characteristic" (5). High distress reactivity was characterized by dysregulated and blatant forms of distress coupled, in many cases, with involvement or avoidance (e.g., comfort seeking) during the SPAT. Angry reactivity reflected the enactment of angry, demanding, controlling, and aggressive behaviors. ICCs between the coders were .88 for distress and .91 for anger.

Children's psychological symptoms—Two primary experimenters who were responsible for keeping in contact with the families, visiting families at their homes, providing transportation to our laboratory, and overseeing activities and tasks during the visits completed ratings of child adjustment at the first and third waves. Ratings were based on close observations of the children for approximately eight hours during each wave, encompassing multiple visits to our laboratory and, in most cases, transportation of families to and from the research center. To increase the validity of the assessments, experimenters retained a written record of their observations of the children after each visit using a form organized around dimensions of psychopathology.

Both experimenters for the visit completed q-set ratings using the California Child Q-Set (CCQ; Block, 2008). The CCQ requires raters to sort 100 descriptors of children's behaviors and psychological symptoms into nine different piles ranging from "extremely uncharacteristic" to "extremely characteristic." Following guidelines for scoring the CCQ (Block, 2008; Shields & Cicchetti, 1997), we selected q-set ratings for the assessment of child externalizing symptoms based on the items placed in the two most characteristic piles

for the CCQ aggression prototype. The resulting externalizing scale consisted of 20 items indexing aggression and defiance (i.e., “Is aggressive physically or verbally”). As the third indicator, the experimenter who completed the CCQ and was primarily responsible for overseeing child activities during family visits filled out the Externalizing scale of the Caregiver-Teacher Report Form (C-TRF; Achenbach & Rescorla, 2006). Internal consistencies across the three measures and two waves ranged from .85 to .94.

Covariates—Children’s sex (1 = boys; 2 = girls), race (1 = Black; 2 = White), family socioeconomic status, and interparental aggression were assessed as covariates. For SES, three indicators were derived from maternal reports on a demographic interview at the first wave: (1) education level, consisting of the mean of maternal and partner educational level (1 = less than 7th grade; 7 = graduate degree); (2) parental occupational strata as indexed by the Hollingshead Four Factor Index (1 = “Farm laborers or Menial Service Workers” to 9 = “Higher executives, proprietors of large businesses, and major professionals”) (Hollingshead, 1975); and (3) household income per capita, calculated as the ratio of total annual family income relative to the number of individuals living in the home. Finally, given the aim of the larger project was to achieve a balanced representation of families experiencing different degrees of interparental aggression, interparental aggression was also included as a covariate. Mothers completed the 24-item Physical Assault Subscale of the Revised Conflict Tactics Scale (CTS2; Straus et al., 1996) to capture a relatively comprehensive range of maternal and partner acts of violence in the interparental relationship over the previous year (e.g., “pushed or shoved my partner,” “choked my partner”). Following scoring guidelines, prevalence scores were calculated for each of the two scales based on the sum of the occurrences of specific aggressive acts (1 = act occurred one or more times; 0 = specific act did not occur). Reliability was satisfactory for the CTS2 ($\alpha = .92$).

Results

Table 1 provides the means, standard deviations, and intercorrelations for the target constructs in this study. As a continuous predictor, maternal unresponsiveness was centered to reduce problems with multicollinearity in the moderator analyses. Skewness in the distress and angry reactivity tallies and the C-TRF measure of externalizing symptoms were log transformed to increase normality in their distributions. Missing data for the primary variables and covariates ranged from 0% to 22% (*Median* = 8%). Therefore, to retain the full sample for primary analyses, structural equation models (SEM) were estimated using full-information maximum likelihood (FIML) with the primary variables and covariates in Amos 19.0 (Enders, 2001).

Primary Analysis I: 5-HTTLPR Genotype as a Moderator of Maternal Unresponsiveness

In analyses testing the moderational role of the 5-HTTLPR genotype in associations between maternal sensitivity and preschooler’s problems, we examined changes in children’s externalizing symptoms across measurement occasions through latent difference score (LDS) models (McArdle, 2009). LDS offers a powerful way of testing whether the interaction between maternal unresponsiveness and the 5-HTTLPR genotype predicts

interindividual differences in intraindividual changes in externalizing symptoms over time through specification of latent difference scores (McArdle, 2009). As a first analytic step, we examined whether the cross product of the 5-HTTLPR genotype and maternal unresponsiveness predicted latent change in child externalizing symptoms over a two year period prior to the inclusion of child distress and angry reactivity as mediators of the moderating effects of 5-HTTLPR. We specified all pathways running from the three predictors (i.e., maternal unresponsiveness, 5-HTTLPR genotype, and their multiplicative interaction) to the latent change in child externalizing problems. Because preliminary correlational analyses revealed that children's sex, race, interparental aggression, and SES were significantly associated with variables in the primary analyses, they were included as covariates in the analyses. To maximize measurement equivalence, loadings of each of the indicators of externalizing symptoms were constrained to be equal across the measurement occasions. We also specified correlations among all the predictors in the model. Finally, although not a primary focus of the study, we also estimated structural paths among the predictors and Wave 1 intercept of child externalizing symptoms. For the sake of clarity, only significant structural paths and correlations are depicted in the figure.

The results of the model, which are shown in Figure 2, provided a good fit with the data, $\chi^2(62, N = 201) = 78.41, p = .08, RMSEA = .04, CFI = .98$. Significant paths involving covariates indicated that: (a) boys exhibited more W1 externalizing problems than did girls, $\beta = -.25, p < .01$; and (b) White children displayed greater increases in externalizing problems from Wave 1 to Wave 3 than did Black children, $\beta = .16, p < .05$. Maternal unresponsiveness was also associated with higher concurrent levels of externalizing problems, $\beta = .21, p < .05$, and changes in externalizing symptoms from Wave 1 to Wave 3, $\beta = .28, p < .01$. Of relevance to the aims of the study, the maternal unresponsiveness $\times$ 5-HTTLPR interaction predicted latent change in children's externalizing symptoms over the two year period even after inclusion of the structural paths involving the other predictors and covariates, $\beta = -.21, p < .01$.

The moderating effect of the 5-HTTLPR genotype for children's externalizing symptoms was first clarified by graphically plotting and calculating the simple slopes of maternal sensitivity separately for the homozygote L and functional S carriers. To portray the interaction across a relatively comprehensive range of the proposed predictor (i.e., 95%), we conducted simple slope plots and calculations at $-2 SD$ and $+2 SD$ from the mean of maternal unresponsiveness (Roisman et al., 2012). As depicted in Figure 3, the graphical plot revealed a disordinal interaction characterized by a cross-over of the two regression lines. Simple slope analyses further showed that maternal unresponsiveness significantly predicted greater increases in children's externalizing symptoms for children with the homozygote L genotype, $b = 13.91, p < .001$, but not for their counterparts carrying the functional S genotype, $b = 2.03, p = .44$.

Following recommendations for examining differential susceptibility (Roisman et al., 2012), two additional analyses were conducted to test the correspondence of the moderating effect with the diathesis-stress and differential susceptibility models. First, in calculating the proportion of the interaction (PoI) index, we examined the ratio of improved functioning for children with the homozygous L allele relative to the overall aggregate of improved and

poor outcomes within the + and – 2 SD range of parental unresponsiveness. Second, because PoI indices can vary depending on the range of interest specified for the predictor variable, we also calculated a proportion affected (PA) index, a coefficient that does not rely on designation of boundaries for parental unresponsiveness. The PA index is defined as the proportion of children within the hypothesized “for better” region in differential susceptibility theory or, more precisely, children who were below the point along the parental unresponsiveness variable in Figure 2 where the two regression slopes cross. PoI and PA indices approximating .50 and exceeding .16 are regarded as yielding support for differential susceptibility, whereas values between .00 and .16 provide stronger support for diathesis stress (see Roisman et al., 2012). In support of differential susceptibility, PoI and PA for the LL allele were .32 and .46, respectively.

Primary Analysis II: Child Emotional Reactivity as a Mediator of 5-HTTLPR Gene Moderation

In the second analytic step, we examined whether children’s emotional reactivity to maternal anger mediated the moderating role of the 5-HTTLPR gene in the prospective links between maternal unresponsiveness and child externalizing symptoms. Therefore, in addition to estimating the same set of structural paths in the analyses depicted in Figure 2, we specified paths between all the predictors and covariates and children’s distress and angry reactivity to maternal anger. In turn, structural paths between child distress and angry reactivity were estimated as predictors of prospective changes in externalizing symptoms. As additional model specifications, we estimated correlations between: (1) child externalizing symptoms at Wave 1 and Wave 2 angry and distress reactivity to maternal anger, and (2) all predictors and covariates.

To maximize clarity, only significant structural paths and correlations are depicted in Figure 4. The overall model fit the data well, $\chi^2(142, N = 201) = 213.28, p < .001, RMSEA = .05, CFI = .95$. Inspection of the structural paths revealed that children’s distress reactivity did not mediate the moderating effects of 5-HTTLPR due to its negligible associations with the maternal unresponsiveness $\times$ 5-HTTLPR interaction term and their externalizing symptoms. However, the interaction between Wave 1 maternal unresponsiveness and the 5-HTTLPR genotype predicted children’s angry reactivity to maternal negativity at Wave 2, $\beta = -.25, p < .01$. Children’s angry reactivity, in turn, predicted increases in their externalizing problems from Wave 1 to Wave 3, $\beta = .28, p < .01$. In addition, the significant direct path between the maternal unresponsiveness $\times$ 5-HTTLPR interaction and externalizing symptoms dropped by 29% from $\beta = -.21, p < .01$ in our original analysis to $\beta = -.15, p < .05$, following inclusion of the mediational pathways. Further supporting the operation of mediated moderation, bootstrapping tests utilizing the PRODCLIN software program indicated that the indirect paths between the moderating role of 5-HTTLPR, children’s angry reactivity, and changes in their externalizing symptoms were significantly different from 0, 95% CI = $-.047$ to $-.391$ (MacKinnon, Fritz, Williams, & Lockwood, 2007).

A stronger case for mediation would be garnered if the moderating effects of 5-HTTLPR for child angry reactivity and externalizing symptoms were comparable in form. Therefore, our next step was to characterize the moderating role of 5-HTTLPR in the association between

Wave 1 maternal unresponsiveness and children's angry reactivity through the approach for dissecting the moderation of 5-HTTLPR in the model of externalizing symptoms. First, as Figure 5 shows, the graphical plot of the relation between maternal unresponsiveness (+ and - 2 SDs around the mean) and angry reactivity for children in the two 5-HTTLPR genotype groups revealed a disordinal interaction that is similar in form to the results for externalizing symptoms. Second, consistent with the findings for externalizing symptoms, simple slope analyses indicated that maternal unresponsiveness was significantly associated with greater angry reactivity at Wave 2 for children carrying the LL allele, $b = 0.75$, $p < .001$, but not for children carrying the functional S allele, $b = 0.09$, $p = .37$. Third, PoI and PA indices yielded additional evidence supporting the 5-HTTLPR-LL genotype as a differential susceptibility marker for children's angry reactivity. The PoI value, indexing the ratio of improved outcomes of children with the LL allele relative to the sum of improved and harmful outcomes, was .50. Likewise, the PA, defined by the proportion of children within the "for better" region of the disordinal interaction, was .54. Both values approximated the .50 range that is interpreted as yielding strong support for differential susceptibility. Thus, the comparability of the graphical plots, simple slope analyses, and PoI calculations across externalizing symptoms and angry reactivity models provide additional evidence for angry reactivity as a mediator of the moderating effects of 5-HTTLPR.

Post Hoc Analyses: Variability in the Moderating Role of 5-HTTLPR across Child Race

Our identification of the LL genotype as a plasticity attribute may largely be the result of variability in the role of 5-HTTLPR as a function of child race. To address this issue, we conducted selective post hoc tests to examine whether the moderational role of 5-HTTLPR in predicting of angry reactivity and change in externalizing symptoms varied significantly between Black and White children. We specifically utilized multiple group comparisons in which the data were split into groups of Black and White children. Given the small sample sizes resulting from dividing the data, we examined the 5-HTTLPR as a moderator in separate models for angry reactivity and change in externalizing symptoms. Multiple group comparisons for each model consisted of comparing a model in which all structural path parameters were allowed to vary freely with a model in which comparable paths across race were constrained to equality.

Comparing the fully constrained and free-to-vary models in the prediction of children's angry reactivity revealed no difference in fit, $\chi^2 (6, N = 201) = 1.56$, *ns*. Thus, race did not moderate pathways to child angry reactivity. In contrast, the free-to-vary model in the prediction of child externalizing symptoms did produce a significantly better fit than the fully constrained model $\chi^2 (13, N = 201) = 24.66$, $p < .05$; thereby indicating structural paths varied significantly as a function of child race. Follow up tests identified race differences for three predictors of change in child externalizing symptoms: (a) parental unresponsiveness, $z = 2.97$, $p < .01$, (b) the 5-HTTLPR gene, $z = 2.13$, $p < .05$; and (c) the maternal unresponsiveness $\times$ 5-HTTLPR interaction, $z = 3.02$, $p < .01$. Parental unresponsiveness predicted increases in externalizing problems for Black ($\beta = .32$, $p < .01$), but not White ($\beta = -.25$, *ns*), children. Conversely, the functional S allele was associated with subsequent decreases in externalizing symptoms for White ($\beta = -.50$, $p < .01$), but not Black ($\beta = -.05$, *ns*) children. Finally, the parenting - 5HTTLPR interaction was a significant predictor of

externalizing symptoms for Black ($\beta = -.32, p < .01$), but not White ($\beta = .15, ns$) children. Consistent with the results for the whole sample, simple slope analyses revealed that maternal unresponsiveness predicted greater externalizing symptoms for Black children with the homozygous L genotype, $b = 18.94, p < .01$, but not the functional S genotype, $b = -0.08, ns$. Consistent with the disordinal (i.e., cross-over of the simple slopes) form of the interaction in the graphical plot (not shown), the respective PoI and PA values of .41 and .50 supported differential susceptibility theory (Roisman et al., 2012). Thus, in keeping with the findings from the larger sample, the results supported the designation of the LL genotype as a plasticity allele for Black children in the sample.

Discussion

Although some studies have identified the S allele as a plasticity characteristic in associations between early adversity and developmental psychopathology, they largely focus on the prediction of internalizing symptoms in predominantly White samples of adolescents or adults (Belsky, Jonassaint, Pluess, Brummett, & Williams, 2009). Accordingly, the first goal of this study was to examine whether the S allele of the gene served a comparable plasticity function in the prospective link between maternal unresponsiveness and externalizing symptoms in an impoverished sample of preschool children who were primarily Black. In highlighting specificity in the operation of 5-HTTLPR, our findings revealed that maternal unresponsiveness predicted greater externalizing symptoms over two-year period for children with the LL allele but not for children carrying the functional S allele. As a second objective, we utilized mediated-moderation analyses to address the process-oriented question of why LL carriers evidenced plasticity. The results indicated that children with the LL allele were more susceptible to developing externalizing symptoms in the context of maternal unresponsiveness by virtue of their tendency to exhibit heightened angry reactivity to maternal negativity.

In the first step of our analyses, follow up tests of the 5-HTTLPR gene as a moderator in the prediction of externalizing problems provided strong support for designating the LL allele as a plasticity characteristic rather than a diathesis. Results showed that mother unresponsiveness predicted increases in externalizing symptoms only for children possessing the LL allele. The graphical plot and the quantitative tests of differential susceptibility (i.e., PoI, PA) revealed that children with the LL genotype experienced outcomes in a “for better” or “for worse” manner across conditions of maternal unresponsiveness. In comparison to their counterparts with the functional S genotype, children with the LL genotype specifically evidenced greater externalizing problems in the context of maternal unresponsiveness but lower externalizing problems when maternal responsiveness was high. Supporting the viability of differential susceptibility framework over the diathesis-stress model, our findings are consistent with the notion that the LL allele confers significant mental health benefits in supportive environments but disproportionate costs in the form of disruptive behavior problems in unsupportive socialization contexts (Belsky & Pluess, 2009).

To advance a process analysis of why 5-HTTLPR moderates early adversity, we also aimed to delineate the cascade of mechanisms underlying the greater susceptibility of children with

the LL allele. Guided by models identifying forms of negative emotional reactivity as mechanisms of variation in serotonergic functioning (van Goozen et al., 2007), we examined distress and angry responses to maternal negativity as two putative processes mediating the moderating role of the 5-HTTLPR gene. Integrated with family process conceptualizations (Dodge, 2006; Snyder, Cramer, AfFrank, & Patterson, 2005), we proposed that genetic sensitivity to maternal unresponsiveness would first emerge in the form of child negative emotionality in the parent-child dyad and, in turn, set the stage for the intensification and proliferation into persistent and intractable displays of externalizing problems. The results indicated that children's angry reactivity partially mediated the association between maternal unresponsiveness and changes in their externalizing symptoms even after estimating the negligible mediational role of distress reactivity. Supporting the first proposed link in the mediational chain, the interaction between maternal unresponsiveness and the 5-HTTLPR gene significantly predicted child angry reactivity to standardized expressions of maternal anger one year later. Consistent with the pattern of plasticity identified in the prediction of externalizing symptoms, associations between maternal unresponsiveness and children's angry reactivity were only significant for LL carriers. Moreover, in comparison to children with the functional S alleles, the graphical plots and differential susceptibility indices (i.e., PoI, PA) supported the hypothesis that LL carriers evinced disproportionately greater anger reactivity in contexts of maternal unresponsiveness but also substantially lower anger reactivity under conditions of maternal responsiveness. These findings decidedly favored predictions of differential susceptibility theory over diathesis-stress models.

In support of the second link in the mediational chain, angry reactivity to maternal negative affect, in turn, was a significant predictor of subsequent increases in preschooler externalizing problems over a two year period. Interpreted in social learning theory (Snyder et al., 2010), child reciprocation of anger may be part of transactional tendency for the dyad to engage in the aggressogenic cycle of mutually escalating negativity. In this coercive process, parents are theorized to capitulate to increasing child hostility without successfully enforcing negative consequences for children (e.g., no discipline). Through negative reinforcement contingencies of eliminating parental threats, the children may increasingly adopt a strategy of escalating aggression and misbehavior across multiple contexts that catalyzes the development of externalizing problems (Snyder et al., 2005). In drawing from coping models, child angry reactivity may also signify the acquisition of negative scripts characterized by highly automatic rules for responding to stressful family events (Johnston, Roseby, & Kuehnle, 2009; Pollak & Tolley-Schell, 2004). To the extent that angry reactivity serves as a blueprint for responding to new social challenges, it may reflexively and intransigently be applied in a wider diversity of settings and, in the process, develop into externalizing problems (Johnston et al., 2009).

Although confidence in the validity of the moderating effects for 5-HTTLPR are bolstered by coherency in the findings across levels of process (i.e., angry reactivity) and outcome (i.e., externalizing symptoms), interpretation of the findings in the context of mixed findings in the literature is warranted. Against the backdrop of some earlier studies identifying that S carriers evidenced heightened environmental susceptibility (e.g., Belsky & Plouss, 2009), our identification of the LL genotype as the plasticity allele may be a product of the

moderating effects of race. Comparing our sample characteristics with the predominantly White samples utilized in previous research, the present findings may be the result of a relatively large proportion (66%) of Black children in our study. In attempting to delineate the mechanisms underlying documented racial differences in the moderating role of 5-HTTLPR (van IJzendoorn et al., 2012), studies suggest that the LL allele for Black individuals may function in a similar way to the S allele in White participants (e.g., Anderson & Mayes, 2010; Williams et al., 2003).

Given evidence that racial differences may underlie our designation of the LL genotype as a plasticity factor, we conducted post hoc, exploratory tests to determine whether the moderating role of 5-HTTLPR varied significantly as a function of child race. Multi-group SEM analyses revealed that the association between the maternal unresponsiveness $\times$ 5-HTTLPR interaction and changes in externalizing symptoms differed significantly for Black and White children. Consistent with research on race differences in the role of 5-HTTLPR in the regulation of serotonin (Anderson & Mayes, 2010; Williams et al., 2003), follow up tests indicated that documentation of LL genotype as the differential susceptibility factor was specific to the Black children in our sample. Conversely, no race differences were found in the moderating effects of 5-HTTLPR in predicting children's angry reactivity. Although it is important to note that our relatively small sample size reduced power to identify racial differences, these results suggest that role of children's angry reactivity to family stress as a mediator of the moderating effects of 5-HTTLPR are comparable for Black and White children. Therefore, racial differences in 5-HTTLPR as a moderator may be one source of our findings, but they may not fully explain why children's angry reactivity accounts for the greater susceptibility of LL carriers to maternal unresponsiveness.

Accordingly, the question remains as to what other factors may account for our findings. Our relatively distinctive focus on externalizing symptoms may be another possible source for our empirical documentation of LL as a plasticity factor. Reviews identifying the S allele as a susceptibility factor have largely examined internalizing problems as outcomes (e.g., Karg et al., 2011; Skelton, Ressler, Norrholm, Jovanovic, & Bradley-Davino, 2012). Rare efforts to expand the scope of outcomes to externalizing symptoms have yielded a different pattern of results. Consistent with our findings, Sulik and colleagues (2012) reported that poorer parenting quality predicted externalizing problems only for children with the LL genotype. Moreover, research has linked the LL allele with externalizing symptoms and its precursors of hostility, impulsivity, and risk-taking (Glenn, 2011; Laucht et al., 2009). According to a recent process model, the interplay between early family adversity and the LL genotype may engender bold, hostile, impulsive behaviors underlying externalizing symptoms by producing serotonin deficits in brain stem regions that regulate mood (Laucht et al., 2009). Supporting this thesis, higher 5-HTT density in the raphe nuclei was associated with lower metabolite markers of serotonin turnover in primates exposed to early adversity. Markers of diminished serotonin turnover, in turn, were associated with social isolation and impulsive aggression. Thus, efficient reuptake of serotonin may reduce the serotonin availability in brain regions implicated in mood regulation (Heinz et al., 2001).

In comparison to a 5-HTTLPR literature that has almost exclusively targeted periods of adolescence and adulthood, our findings may also differ from some previous results by

virtue of our focus on early childhood. In documenting dynamic changes in serotonergic functioning in early childhood, peak serotonin levels during toddlerhood progressively decrease to adult levels by five years of age (Whitaker-Azmitia, 2001). Thus, early childhood is commonly regarded as a sensitive period for the regulation of serotonin (Anderson, 2003; Embree et al. 2013; Whitaker-Azmitia, 2001), with neurobiological models proposing that serotonin has different implications early in life (e.g., Anderson, 2003; Uher & McGuffin, 2007). In accord with these models, links between markers of high serotonin levels and diminished aggression in adult animals do not generalize to young organisms (Sodhi & Sanders-Bush, 2004; Taravosh-Lahn, Bastida, & Delville, 2006). Likewise, in research on humans, relations between 5-HTTLPR and infant negativity reversed during infancy such that S carriers showed greater distress at two months of age but lower distress at one year of age (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2003).

Fully interpreting the results requires consideration of limitations of our study. First, although the prospective prediction of changes in child psychological problems within a multi-method, multi-informant design increases methodological rigor, our approach does not permit a full prospective analysis of change at each link in the mediational chain. Because child emotion reactivity to maternal anger was only observed at the second measurement occasion, we were unable to test whether parenting $\times$ 5-HTTLPR genotype interaction predicted changes in their angry emotional reactivity. Thus, despite the time lag between the constructs, it is also possible that our findings may reflect the operation of child effects of angry reactivity on parenting, reciprocity between child angry reactivity and maternal unresponsiveness, or third variables (e.g., temperament). Second, due to the impoverished, primarily Black, and high risk (e.g., family violence) nature of our sample, replicating our findings in samples with different sociodemographic, family, and developmental characteristics is an important future direction. Given our relatively small sample size (particularly our White subsample), use of larger samples of children who are specifically selected for racial homogeneity (e.g., Black, White) will permit more powerful empirical tests of whether the moderating role of 5-HTTLPR varies significantly across race. Third, although the consistency of the 5-HTTLPR genotype as a moderator in predicting externalizing symptoms and the mediating role of angry reactivity bolsters the fidelity of our results, the documented associations were generally modest to moderate in magnitude.

In conclusion, commentaries on the state of the literature have emphasized the value of delineating the specificity and generalizability of 5-HTTLPR S genotype as a susceptibility allele across racial characteristics (Ellis et al., 2011), form of psychological adjustment (Glenn, 2011), and developmental period (Uher & McGuffin, 2007). Testifying to the significance of this research direction, our findings with a sample of racially diverse preschool children indicated that the homozygous L genotype served as plasticity characteristic that was associated with significantly lower and higher levels of externalizing symptoms under respective conditions of maternal responsiveness and unresponsiveness. In further advancing a process-oriented approach to $G \times E$ research, results of mediated moderation analyses revealed that children's angry reactivity to maternal negativity explained, in part, the greater susceptibility of homozygous L carriers to developing externalizing problems in the face of maternal unresponsiveness. Therefore, our findings suggest that the proposed plasticity properties of 5-HTTLPR alleles may reverse under some

specific developmental and contextual conditions. At a translational level, identifying how the implications of parenting for children's externalizing symptoms may differ depending on children's genetic make-up may not only help to identify the biological sources of individual differences in developmental outcomes, it also highlights the importance of carefully tailoring early prevention and intervention approaches to the specific profile of intrapersonal and family attributes experienced by young children (Cicchetti & Toth, 2009). Moreover, identifying angry reactivity to maternal negative affect as a behavioral carrier of the genetic susceptibility experienced by children carrying the LL allele may also be helpful in developing targets of treatment and therapeutic tools for these children (e.g., emotion regulation techniques).

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Figure 1.

A conceptual model of the interplay between maternal unresponsiveness and the 5-HTTLPR genotype in predicting a developmental cascade of children's disinhibited emotional reactivity and externalizing problems.

Figure 2.

Structural equation model examining the interaction between maternal responsiveness and the 5-HTTLPR gene in predicting latent difference score derived changes in children's externalizing symptoms over a two year period. Parameter estimates for the structural paths are standardized path coefficients. ** $p < .01$; * $p < .05$.

Figure 3.

A graphical plot of the interaction between maternal responsiveness and the 5-HTTLPR gene in predicting changes in children's externalizing symptoms over a two year period.

Figure 4.

Structural equation model examining children's emotional reactivity as a mediator of the interaction between maternal responsiveness and the 5-HTTLPR gene in predicting changes in children's externalizing symptoms over a two year period. Parameter estimates for the structural paths are standardized path coefficients. $^{**}p < .01$; $^*p < .05$.

Figure 5.

A graphical plot of the interaction between maternal responsiveness and the 5-HTTLPR gene in predicting children's angry reactivity to maternal anger one year later.

Table 1

Means, Standard Deviations, and Intercorrelations Among the Primary Variables.

	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Wave 1 Primary Predictors																
1. Mother unresponsiveness	0.00	0.82	–													
2. 5-HTTLPR	0.00	0.45	–.14	–												
3. Unresponsiveness $\times$ 5-HTTLPR	–0.05	0.36	.05	.15*	–											
Wave 2 Child Angry Reactivity																
4. Tally	0.85	1.64	.22*	–.03	–.11	–										
5. Specific Rating	1.31	1.52	.24*	–.03	–.12	.66*	–									
6. Molar Rating	1.76	1.12	.27*	–.04	–.17*	.76*	.84*	–								
Wave 2 Child Distress Reactivity																
7. Tally	1.96	2.29	–.06	–.06	–.04	.15	.11	.04	–							
8. Specific Rating	2.19	1.26	–.02	–.08	–.05	.27*	.28*	.21*	.75*	–						
9. Molar Rating	2.33	1.12	.08	.03	–.08	.38*	.56*	.42*	.51*	.62*	–					
Wave 1 Externalizing Problems																
10. Exp. 1 Q-set	89.17	20.42	.22*	–.01	–.03	.09	.12	.11	–.06	.07	.09	–				
11. Exp. 2 Q-set	92.81	22.45	.21*	.00	–.10	.11	.13	.16*	–.09	.04	.12	.70*	–			
12. Exp 1 CTRF	5.72	7.17	.25*	–.06	.03	.17*	.16*	.14	–.09	–.03	.02	.75*	.55*	–		
Wave 3 Externalizing Problems																
13. Exp. 1 Q-set	87.89	21.39	.31*	–.11	–.18*	.24*	.23*	.36*	–.09	.01	.12	.27*	.28*	.21*	–	
14. Exp. 2 Q-set	91.52	21.22	.24*	–.15	–.13	.24*	.25*	.32*	–.10	–.04	.03	.24*	.20*	.25*	.73*	–
15. Exp 1 CTRF	9.29	10.64	.28*	–.12	–.14	.25*	.29*	.39*	–.07	.03	.13	.27*	.25*	.28*	.88*	.73*

Note.

*
 $p < .05$.