HHS Public Access

Author manuscript

Soc Dev. Author manuscript; available in PMC 2018 April 04.

Published in final edited form as:

Soc Dev. 2017 November ; 26(4): 724–739. doi:10.1111/sode.12221.

Gene-environment correlations in the cross-generational transmission of parenting: Grandparenting moderates the effect of child 5-HTTLPR genotype on mothers' parenting

Daniel C. Kopala-Sibley,
Department of Psychology, Stony Brook University, Stony Brook, NY

Elizabeth P. Hayden,
Department of Psychology, Western University, London, Ontario, Canada

Shiva M. Singh,
Department of Psychology, Western University, London, Ontario, Canada

Haroon I. Sheikh,
Department of Psychology, Western University, London, Ontario, Canada

Katie R. Kryski, and
Department of Psychology, Western University, London, Ontario, Canada

Daniel N. Klein
Department of Psychology, Stony Brook University, Stony Brook, NY

Abstract

Evidence suggests that parenting is associated cross-generationally and that children's genes may elicit specific parenting styles (evocative gene-environment correlation). This study examined whether the effect of children's genotype, specifically 5-HTTLPR, on mothers' parenting behaviors was moderated by her own parenting experiences from her mother. Two independent samples of three-year-olds (N = 476 and 405) were genotyped for the serotonin transporter gene, and observational measures of parenting were collected. Mothers completed measures of the parenting they received as children. The child having a short allele on 5-HTTLPR was associated with more maternal hostility (sample 1 and 2) and with less maternal support (sample 1), but only if the mother reported lower quality grandmothers' parenting (abuse and indifference in Sample 1 and lower levels of grandmother care in Sample 2). Results support the possibility of a moderated evocative gene-environment correlation.

Keywords

Serotonin; 5-HTTLPR; gene-environment correlation; transmission of parenting

A core tenet of developmental psychology is that individuals are shaped in important ways by early experiences. One such experience, early caregiving, has lasting influences on

Correspondance: Daniel C. Kopala-Sibley, Ph.D., Department of Psychology, Stony Brook University, Stony Brook, NY, 11794-2500, daniel.kopala-sibley@mail.mcgill.ca.

children's development, including cognitive, linguistic, and academic functioning (see Bradley & Vandell, 2007), social competence (Crockenberg & Leerkes, 2005), and psychological disorders (McLeod et al., 2007), in part through the impact of parenting on children's development of vulnerable cognitive or personality styles (e.g., Hankin et al., 2009; Kopala-Sibley & Zuroff, 2014). Understanding biological and environmental factors that predict parenting behaviours in early childhood is therefore important.

A substantial literature shows that parenting styles or behaviours "run in families" or are transmitted cross-generationally (e.g., Beaver & Belsky, 2012; Belsky, Conger, & Capaldi, 2009). However, children also actively shape their environments, and a burgeoning literature is examining how children's genes may influence the environment around them via gene-environment correlations (rGE, Hayden et al., 2013; Jaffee & Price, 2007). However, no research of which we are aware has attempted to integrate the cross-generational transmission of parenting with rGEs to better understand the role of parents' own experiences of early care as well as children's genes in predicting parenting. To be clear, throughout this manuscript, any references to "grandparenting" or parents' own experiences of care refer to parents' retrospective reports of the parenting they received. As such, the goals of this study were to examine, in two independent samples of young children, whether the association between variants of the child's 5-HTTLPR genotype and mothers' behaviors towards their child was moderated by the mother's parenting experiences with her own parents (i.e., children's grandparents). If so, this would provide evidence for a moderated rGE, in that child genotype is related to maternal parenting behaviors only if the mother experienced certain parenting behaviors herself.

Cross-generational transmission of parenting and gene-environment correlations

Researchers have long been interested in factors that determine why parents parent the way they do (Belsky, 1984), and parents' experiences with their own parents have been one widely studied determinant (e.g., Serbin & Carp, 2003; Beaver & Belsky, 2012). For instance, multiple longitudinal studies have found that individuals whose own parents were harsh, rejecting, uncaring, or controlling are more likely to parent their own children in a similar manner (e.g., Belsky, Jaffee, Sligo, Woodward, & Silva, 2005; Conger, Schofield, Neppl, & Merrick, 2013; Friesen, Woodward, Horwood, & Fergusson, 2013; Kovan, Chung, & Sroufe, 2009; Neppl, Conger, Scaramella, & Ontai, 2009). However, despite the robustness of these findings, not all parents treat their children the way their own parents treated them. Indeed, only approximately 15% of the variance in parenting is attributable to the care that parents themselves received (Belsky et al., 2005; Capaldi, Pears, Patterson, & Owen, 2003; Conger et al., 2003), indicating that other factors may moderate the relationship between one's own care and the care one provides to offspring.

As Conger, Belsky, and Capaldi (2009) note, our understanding of mechanisms responsible for continuity or discontinuity in parenting across generations is limited. One possibility that has received relatively little attention is that children's genetic variants elicit parenting behaviours via rGE (Jaffee & Price, 2007; Hayden, Hanna, Sheikh, Laptook, Kim, Singh, &

Klein et al., 2013; Kryski, Smith, Sheikh, Singh, & Hayden, 2014). It is also possible that the strength of the association between children's genetic variants and the parenting behaviors the child receives is moderated by the parenting the parent received. More specifically, children's genetic variation may be related to the extent to which associations are found between the caregiving their parents provide and their parents' own early experiences of care. This possibility would be consistent with the growing rGE literature, although most studies have used quantitative genetic, rather than measured gene, approaches (see Kendler & Baker, 2007; Avinun & Knafo, 2014, for meta-analyses). However, a handful of studies examined variations of children's dopaminergic genes and found associations with negative parenting (Hayden et al., 2010; Hayden et al., 2013; Lucht et al., 2006; Mills-Koonce et al., 2007), and one study found associations with oxytonergic genes (Kryski et al., 2014). Other research has found associations between child catechol-O-methyltransferase genotype and positive parenting (Oppenheimer et al., 2013). Only one study of which we are aware tested a rGE between the child's serotonin transporter gene and the parenting they receive: Pener-Tessler et al. (2013) found that children with at least one short variant of 5-HTTLPR experienced lower levels of positive parenting and higher negative affect expressed by the mother during laboratory interaction tasks.

Given the work implicating variants of the serotonin promotor gene in emotions and interpersonal experiences (e.g., Steemer, Branchi, & Homberg, 2012; Pauli-Pott et al., 2009; Pluess et al., 2011), the role of 5-HTTLPR in eliciting aspects of early care merits further investigation. For instance, as several studies have associated a short allele with negative emotionality, fear, hostility, and anxiety (e.g., Pauli-Pott et al., 2009; Pluess et al., 2011; Zimmerman et al., 2009; Sen et al., 2004; Schinka et al., 2004), which elicit suboptimal patterns of care (Lengua & Kovacs, 2005).

Although work supports the possibility that children's genes elicit certain parenting behaviours, this research does not speak to whether children's genes may interact with their grandparents' behaviors towards their parents to predict their parents' behaviours. Such a possibility could occur if genetic variants shape the context of parental care in ways that render parents more likely to rely on parental care they themselves received as a model. Only a handful of studies have examined variables that interact with the intergenerational transmission of parenting, finding that the age at which the parents had children (Belsky, Hancox, Sligo, & Poulton, 2012), marital quality and the level of support and nurturance from the spouse toward the parent (Conger, Schofield, Neppl, & Merrick, 2013; Wang et al., 2014), and the spouse's parenting of the child (Conger, Schofield, & Neppl, 2012) interact with the parenting received by participants in predicting their own parenting behavior. Evidence also suggests the nurturing behaviours of others in the parent's life (Jaffee et al., 2013; Schofield et al., 2013), and the child's gender interact with the parenting received by participants in predicting their own parenting, although these findings are mixed (see Conger et al., 2009). However, no research of which we are aware has tested whether child genotype interacts with the parent's own parenting experiences to influence their parenting behaviors.

How might this interaction occur? Given evidence of evocative rGEs, it is possible that mothers may only show suboptimal parenting if they both experienced similar parenting themselves, and have a child with a genotype which elicits negative aspects of parenting.

This process implies a moderated gene-environment correlation. No research of which we are aware has tested this possibility. However, some evidence indirectly suggests that there may be interactions between the child's genotype and grandparent behaviors in predicting the parent's parenting behaviors. Beaver and Belsky (2012) examined genetic differential susceptibility (Belsky & Pluess, 2009) to the effects of participants' parenting experiences on their own parenting. Although they examined parental genes as the moderators in their analyses, rather than grandparenting behaviors, they found that adults carrying a greater number of alleles believed to render one more "plastic" to environmental experiences exhibited a stronger relationship between their parents' and their own parenting. Others have shown how specific genotypes are associated with more negative parenting behaviors, but only in certain contexts: mothers with a short allele at 5-HTTLPR show less sensitive parenting in the context of high levels of interparental conflict (Sturge-Apple et al., 2012). This suggests some mothers may be genetically susceptible to the effects of external influences on their parenting. However, Bakermans-Kranenberg & van Ijzendoorn (2008) reported contrary evidence, finding a main effect for mothers having a short allele on 5-HTTLPR, but no gene X marital discord interaction on their parenting behaviors. Thus, this literature is somewhat mixed. A possible explanation is that the child, who inherited their parents' genes, may in fact be the driving force influencing the transmission of parenting. Given that parents' genes appear to have main effects on how they parent, and may also interact with their parenting experiences to predict their own parenting, it possible the effect of the child's genes on their parents' behaviors may be moderated by the parents' own parenting experiences. This possibility is further supported by other evidence suggesting that child temperamental negative emotionality may elicit more negative parenting, but only if the parent experienced negative parenting behaviors in their childhood (Scaramella & Conger, 2003). Given that negative emotionality is associated with having a short allele on 5-HTTLPR (Hayden et al., 2010; Pauli-Pott et al., 2009), this further supports the possibility that children's genotype may influence parenting behaviors, but only if the parents experienced lower quality parenting themselves.

With this literature in mind, the overarching goal of this study was to test whether the influence of children's 5-HTTLPR genotype on their parents' behaviors towards them was moderated by the parenting their parents received. We expected that having the s/s or s/l variant of the 5-HTTLPR serotonin transporter gene would be associated with lower quality parenting during a lab-based mother-child interaction task, but only if the mother experienced high levels of negative grandmother parenting behaviors herself.

Method

Participants

Sample 1—Participants for Sample 1 consisted of a community sample of 405 children (208 girls) and their primary caregiver, recruited as part of a study of child temperament. For full methodological details, see Kryski et al. (2014). Children ranged between 36 and 47 months of age at baseline (M = 40.72, SD = 3.51). Families were recruited through a university's research participant pool and advertisements placed in local daycares, recreational facilities, and websites. Children with significant medical or psychological

problems were excluded from participation. Primary caregivers were usually the children's mothers (N = 380; 93%). Family income varied (5.5% < $20,000; 11% = $20,000–$40,000; 22.7% = $40,001–$70,000; 31.2% = $70,001–$100,000; 29.5% > $100,001). Children were mostly European American (90%).

Sample 2—Participants were 476 children (251 males) from a larger longitudinal study of 569 three-year-old children (see Olino et al., 2010 for details) and their mothers. The mean age of the children was 43.5 months (*SD* = 2.8).

Participants were recruited through a commercial mailing list and screened by phone. Eligible children had no significant medical problems or developmental disabilities, and had at least one English-speaking biological parent who could participate. Most children were from middle-class families, as measured by Hollingshead's four-factor index of social status (M = 44.4, SD = 10.7; Hollingshead, 1975). Most children were European American and non-hispanic (93.9%) and came from two-parent families (98.1%), middle-class families, as measured by the Four Factor Index of Social Status (*M* = 46.1; *SD* = 10.3; Hollingshead, 1975).

Procedure

Genotyping—Buccal samples were obtained and children were genotyped at age 3 in both samples using the same laboratory. We used the Qiagen DNA MicroKit (Qiagen, Valencia, CA) to extract genomic DNA from buccal epithelial cells. Purified genomic DNA was kept at 4°C while being analyzed and then at −80°C for long-term storage. Genotyping for the 5-HTTLPR variable number tandem repeat was conducted via polymerase chain reaction (PCR) following Sheikh and colleagues (2008) using the Applied Biosystems thermal cycler Gene Amp 9700 (Applied Biosystems, Foster City, California, USA). PCR amplicons were separated on polyacrylamide gels, stained with ethidium bromide, and visualized and documented by an ultraviolet imaging system (BioRad Labs, Mississauga, ON).

In the first sample, 127 participants had LL variants, whereas 193 had the SL variant, and 85 had the SS variant (total 283 in the SS/SL group). This distribution is in Hardy–Weinberg equilibrium ($\chi 2 = .54$, p = .46). In the second sample, 143 participants had LL variants, while 241 had the SL, and 92 had the SS variants (total N of 331 in the SS/SL group) of 5-HTTLPR. This distribution is also in Hardy–Weinberg equilibrium ($\chi 2 = .28$, p = .58). To insure accuracy of the genotyping data, a technician randomly selected and reanalyzed 10% of the DNA samples, but no discrepant results were found for 5-HTTLPR.

Sample 1—When children were age 3, mothers and children participated in the three-bag task (National Institute of Child Health and Human Development [NICHD] Early Child Care Research Network, 1997; Ispa et al., 2004) during a home visit, from which maternal support and hostility were coded. Approximately two years later, as part of the second phase of this study, mothers completed the Measure of Parenting Styles (MOPS; Parker et al., 1997) as a measure of their own parenting experiences as children. All 405 children provided buccal samples for genotyping.

At age 5, of the 405 participants who provided buccal samples at age 3, 99 parents had missing data on the MOPS, whereas there was no missing data on parenting variables. Participants were coded as having any missing data or no missing data. A series of independent t-tests showed that missingness was unrelated to any variable in the current study (all *p*s > .05).

Sample 2—Only participants who provided buccal samples are included in the current analyses, yielding an effective sample of 476 at age 6. Children included in the current study did not differ from those who did not give buccal samples on any variables in the current study (all *p*s > .05).

At age 3, children and their mother participated in a modified version of the Teaching Tasks battery (Egeland et al., 1995), from which maternal support and hostility were rated. At age 6, mothers completed self-report measures of their experiences with their own mothers (Parental Bonding Inventory; PBI, Parker et al., 1979).

At age 6, of the 476 children who were genotyped at age 3, 122 had missing data on maternal ratings of grandmother care and overprotection, and 33 had missing data on maternal hostility and support. As with Sample 1, missingness was unrelated to any variable in the current study (all *p*s > .05).

Materials

Grandmothers' Parenting—In the first sample, mothers reported on their experiences of being parented via the Measure of Parenting Styles (MOPS; Parker et al., 1997), a revision of the Parental Bonding Inventory (PBI; Parker et al., 1979) that includes three subscales: parental abuse (5 items), indifference (6 items), and overcontrol (4 items). Participants are asked to indicate how true each statement is for them, with regards to the first sixteen years of their life, with response options ranging from 0 ("not true at all") to 3 ("extremely true"). The MOPS shows similar psychometric properties to the widely used PBI (Parker et al., 1997), including good test-retest reliability (Parker et al., 1997), high internal consistency (Parker et al., 1997), and is independent of personality, mood, and psychopathological symptoms or diagnoses (Parker et al., 1997). Retrospective measures of parenting have been found to be fairly valid accounts of childhood experiences (Hardt & Rutter, 2004). In the current study, alphas for indifference (.84), abuse (.77), and overcontrol (.72) were adequate.

In Sample 2, mothers reported on their experiences of care and overprotection using Neale et al.'s (1994) 7-item revision of the Parental Bonding Inventory (PBI, Parker, Tupling, & Brown, 1979), a widely used self-report measure of recalled parenting experiences during the first 16 years of life. This version contains three items assessing care and four items assessing overprotection, each rated on a scale ranging from 1 (none) to 4 (a lot). PBI care and overprotection, conceptually, respectively assess similar constructs as MOPS indifference/abuse and overcontrol (Parker et al., 1997). The PBI also correlates well with other measures of reported parenting and with interviewers' judgments of the parent-child relationship as well as observers' ratings based on observation of their interactions (Parker, 1981; Parker et al., 1979). In the current study, alpha was .62 and .73 for maternal grandmother's overprotection and care, respectively.

Maternal support and hostility—In Sample 1, maternal support and hostility were coded from a task developed by the NICHD (1997; Ispa et al., 2004), in which the primary caregiver and child were instructed to play together with three bags of toys. The pair was told to play with the toys in order and to put away one set of toys before moving onto the next set. This free play paradigm lasted approximately 10 min. Video-recordings of the task were coded by trained graduate and undergraduate raters using a coding manual that was based on the Teaching Tasks coding manual (Weinfield, Egeland, & Ogawa, 1997) and the Qualitative Ratings for Parent–Child Interactions scale (Cox & Crnic, 2003), which include the subscales of maternal hostility and support. Raters coded a minimum of 10 consecutive tapes with an intraclass correlation of .80 with a master coder before coding independently. Once this standard was established, intermittent reliability checks were performed on 15% of all recordings, and coders periodically met and reviewed recordings together to prevent observer drift. Ratings of support were based on the mother's provision of emotional support and expression of positive regard. Ratings of hostility were based on mothers' expressions of anger, frustration, annoyance, and discounting or rejecting of the child. The average ICC for interrater reliability of both tasks, coded on 15% of the sample, was .86.

In Sample 2, maternal support and hostility were coded based on mother-child interactions during the Teaching Tasks battery (Egeland et al., 1995). The battery consisted of six standardized parent–child interaction tasks lasting a total of 25 to 30 minutes. The tasks, which occurred in the order listed here, were designed to elicit a variety of parenting styles and child behaviors, and consisted of book reading, naming objects with wheels, building with blocks, matching shapes, completing a maze, and opening a gift. An approach similar to that in sample 1 was used to train coders and establish inter-rater reliability. Maternal support ($\alpha = .86$) and hostility ($\alpha = .76$) were coded using a global approach to coding, with a single rating given for each of the six tasks. Ratings were subsequently averaged across tasks to yield total scores for each variable. The interrater intra-class correlation coefficient (ICC; n = 35) for support was .84, and .85 for hostility.

Data Analyses

Data were viewed as missing at random for analyses. Full Information Maximum Likelihood (FIML) procedures in AMOS 22.0 were used to estimate the means and intercepts in the presence of missing data for our analyses concerning the moderating effect of grandparent behaviors on the relationship between child genotype and their mother's support and hostility. This approach is generally acknowledged to be preferable to other methods for dealing with missing data, such as listwise deletion or mean imputation, as these latter approaches are more likely to yield biased estimates (see Schafer & Graham, 2002).

Primary analyses consisted of multiple regression models in AMOS 22.0. Given the strong correlation between MOPS abuse and indifference ($r = .63$, $p < .001$), they were averaged to create a composite negative parenting variable. This was done rather than creating a latent variable as latent variables with two indicators with correlated error variances typically result in poor model identification (Bollen & Davis, 2009). In each model, maternal support and hostility, which were covaried, and were simultaneously regressed on grandparent negative parenting and overcontrol (sample 1) or grandparent care and overprotection

(sample 2). Models then included a main effect of child genotype, as well as the interaction between child genotype and each of the two grandparent parenting behaviors. Standardized regression estimates are presented for all estimates. Results from both samples are presented in figure 1.

We then used Hayes' (2013) PROCSS macro in SAS 9.3, which implements the Johnson-Neyman test, also known as a regions of significance test (Johnson & Neyman, 1936; Bauer & Curran, 2005). This examines at which levels of the moderator (i.e., grandparent parenting) the predictor (i.e., child genotype) shows a significant effect on the dependent variable (i.e., maternal support or hostility). In this study, the effects of child genotype were examined at the 10^{th}, 25^{th}, 50^{th}, 75^{th}, and 90^{th} percentile levels of negative grandparent behaviors. This helps to overcome the somewhat arbitrary nature of only examining the effect of a predictor at two levels of the moderator (see Hayes & Matthes, 2009). Moreover, we examined these five levels of our moderator in case significant effects were not found at more commonly used levels of the moderator (i.e., +/− 1SD).

In both samples, analyses initially included the effect of maternal age when the child was age 3, as well as the effect of child gender on maternal support and hostility. However, neither of these effects was significant; thus, these variables were dropped. We also initially examined the effects of concurrent maternal depressive symptoms, assessed by the Inventory to Diagnose Depression (IDD; Zimmerman & Coryell, 1987) in sample 1 and by the Diagnostic Inventory for Depression (DID; Zimmerman et al., 2004) in sample 2. We additionally examined the effects of concurrent child negative and positive emotionality, assessed by the Laboratory-Temperament Assessment Battery (Goldsmith et al., 1995). In both samples, depressive symptoms were unrelated to maternal support or hostility, and did not alter the significance of effects reported below, and so were also dropped from our models. Similarly, the significance of results reported were unaltered after adjusting for child negative and positive emotionality, and so were dropped from our models. In order to control for effects of possible population stratification, all analyses were repeated after excluding non-European American participants (N = 40 in Sample 1, N = 29 in Sample 2). However, all results remained significant at levels reported, and so non-European American participants were retained in our analyses.

Results

Descriptive statistics and bivariate correlations

Descriptive statistics and bivariate correlations are presented in Table 1 for Samples 1 and 2. Apart from significant intercorrelations between grandparent parenting variables and between maternal support and hostility, higher levels of negative grandparent parenting were related to higher levels of maternal hostility in sample 1. Otherwise, variables were largely unrelated to one another at the zero-order level. A series of independent samples t-tests comparing levels of maternal hostility and support across child genotype showed no significant differences in sample 1 (both ps > .58) or sample 2 (both ps > .60), confirming no main effect of child genotype on observed maternal behaviors.

Moderation of rGEs by grandmother parenting behaviors – Sample 1

Results from all regression models are presented in Table 2. Maternal support and hostility were regressed upon the composite of grandmother indifference and abuse, as well as overcontrol, child genotype, and the interaction of child genotype with overcontrol and the composite negative grandparent behavior variable. Results showed no main effects of genotype or either grandparent variable, and no interaction between child genotype and overcontrol. However, there was a significant interaction between child genotype and negative grandparenting in predicting both maternal support and maternal hostility. Thus, moderation by child 5-HTTLPR genotype was found for the relationship between grandparent negative parenting and maternal support and hostility.

Regarding the shapes of these interactions (Figure 1), children having a short allele on 5-HTTLPR was associated with more maternal hostility when mothers' reports of negative grandparent behaviors were in the 90th percentile (standardized Mean = 1.12, $\beta = .26$, $t = 2.54$, $p = .01$). Similarly, children having a short allele on 5-HTTLPR was associated with less maternal support when mothers' reports of negative grandparent behaviors were in the 90th percentile ($\beta = -.44$, $t = -2.21$, $p = .027$). However, below this level of negative grandparent parenting behaviors, there was no significant effect of child genotype on maternal support or hostility (all ps > .16).

Sample 2

In our second sample, maternal support and hostility were regressed on grandmother care and control, as well as the main effect of child genotype, and interactions between genotype and grandmother care and control (Table 2). Results showed no main effects of any variable, and no interaction of child genotype and grandmother control in predicting either maternal support or hostility. There was, however, a significant interaction between child genotype and grandparent care in predicting maternal hostility, but not support. Thus, similar to Sample 1 findings, child 5-HTT genotype moderated the effect of grandparent care on maternal hostility.

Regarding the shape of this interaction (Figure 2), children having a short allele on 5-HTTLPR was associated with more maternal hostility when mothers reports of grandparent care were in the 10th percentile (standardized Mean = −1.3, $\beta = .15$, $t = 2.02$, $p = .04$). However, above this level of grandparent care, there was no significant effect of child genotype on maternal support or hostility (all ps > .16).

Ancillary analyses

One concern is that effects of children's genes are confounded with mothers' genes. That is, effects may be due to mothers' having a short allele at 5-HTTLPR. Although we cannot rule out this possibility without having genotyped mothers, we know that of the children with the SS genotype, all of their mothers must have a short allele. However, of the children with the SL genotype, approximately half of their mothers likely do not have a short allele. Thus, if effects of grandmothers' parenting on mothers' parenting are due to the mothers' having a short allele, effects may be stronger in children with the SS compared to the SL genotype. Thus, we re-analyzed our model, grouping children into either SS or SL genotypes. A multi-

group modeling approach was used to examine whether regression paths differ significantly across groups. When regression paths were constrained to be equal (i.e., no significant moderation), the model yielded $\chi 2$ (58) = 91.10. When regression paths were free to vary across groups (i.e., potentially significant moderation), this model yielded $\chi 2$ (53) = 85.90. A chi-square difference test showed that allowing paths to vary across groups, which would imply significant moderation, did not significantly improve model fit, delta$\chi 2$ (5) = 5.20, p = .39. Thus, regression paths do not significantly differ across the SS and SL groups. This finding diminishes the possibility that effects are due to mothers' having a short allele at 5-HTTLPR.

Discussion

This is, to our knowledge, the first study to examine whether the relationship between children's genotype and observations of their mothers' support and hostility towards their young children is moderated by the mothers' own parenting experiences. In two independent samples, mothers of children who had the S polymorphism of 5-HTTLPR showed less support and more hostility towards their child, but only if they had experienced elevated negative parenting themselves, although the effect on support did not replicate in our second sample. Overall, these results support a moderated rGE in the transmission of parenting, such that child genotype is more likely to be related to specific parenting behaviors, but only if the mother herself was parented in a certain way. However, results may also be consistent with the possibility that it is the mother's genotype which renders her more likely to parent her child in a similar manner to how she was parented. As such, we consider both possibilities.

rGEs and the cross-generational transmission of parenting

Our results are broadly consistent with the substantial literature demonstrating the relationship between parents' and their offspring's parenting (Beaver & Belsky, 2012), as well as a smaller literature on children's genes and their parenting experiences (e.g., Hayden et al., 2013), although we note that the lack of main effects of child genotype across the samples is inconsistent with this prior research. However, the current findings may provide a framework for integrating these literatures in that we provide evidence that the influence of children's genotype on parenting may be moderated by the parenting behaviors experienced by the mother. Although consistent with a moderated rGE, there are alternative explanations for our findings, as discussed below.

Our results indicate that the child having a short allele of the 5-HTTLPR gene may elicit more hostility (Samples 1 and 2) and less support (Sample 1) from their mother, but only if that mother experienced high levels of indifference or abuse or low levels of care from her own mother. Although mothers who experienced low levels of care as children may be already prone to parent their child with less support and more hostility, children with a short allele on 5-HTTLPR may also tend to elicit more negative parenting from their mothers. Although our results remained significant after adjusting for child negative and positive emotionality, this does not preclude the possibility that children with a short allele on 5-HTTLPR exhibit temperamental or behavioural features that result in the mother being more

likely to parent the child the way she was parented in her early life. This possibility would be consistent with Hayden et al. (2013), who found that the effects of variations in dopaminergic genes on parenting were partially mediated by the child's negative affectivity, while Kryski et al. (2014) found a similar indirect effect of oxytonergic variations on parenting via child temperament. In addition, Penner-Tessler et al., (2013) found that the effect of the child having one short allele at 5-HTTLPR on their parents' behaviors was mediated by the child's self-control. Other evidence also suggests that children with a short allele show more negative emotionality and fear (Pauli-Pott et al., 2009; Pluess et al., 2011) and more hostility towards their mothers, especially if they are insecurely attached (Zimmerman et al., 2009). However, an in-depth examination of the role of child temperament was beyond the scope of this paper, but this is a question we hope to examine in the future.

That child characteristics may mediate the effect of children's genotype on parenting behaviors as a function of the parent's own parenting experiences is speculative given that we did not examine child characteristics in depth in the current study. Nevertheless, it is possible that mothers who experienced less caring parenting themselves may find it particularly challenging to parent children who show genetically influenced temperamental or behavioral styles that may be perceived as more difficult or less reinforcing (e.g., Scaramella & Conger, 2003; Mathis & Bierman, 2015). Our findings suggest that the child's genotype may elicit sub-optimal patterns of parenting, but only if the parent internalized or learned specific parenting behaviors based on their early experiences with their own parents. In this context, grandparenting may influence to what extent children's 5-HTTLPR genotype affects to what extent mothers who experienced less optimal care themselves rely on such negative early care as a template for parenting. To be clear, however, we are not suggesting that one specific temperamental characteristic in particular mediates the effects we found. As just discussed, several distinct temperamental variables have been shown to mediate rGEs, and future research would benefit from investigating these possibilities.

We also note that the effects of child genotype on parenting were only significant at either very high (90^{th} percentile) levels of negative grandparenting or very low (10^{th} percentile) levels of grandparent care, suggesting the shape of this interaction does not follow a linear pattern. Instead, it appears that relatively extreme forms of negative grandparenting are necessary for child genotype to be related to parenting behaviors. Additionally, figure 1 may suggest a cross-over pattern, such that when mothers experienced low levels of negative grandparent behaviors, the SS/SL allele was associated with less maternal hostility and more support. However, these effects were not statistically significant, so should be interpreted very cautiously.

Finally, results showed only a small effect of child genotype, even in interaction with grandparenting behaviors. As such, there are undoubtedly many other factors that influence parenting behaviors, and these should continue to be examined in future research. It is also possible that grandparents' behaviors may have been influenced parents' behaviors, or by the parent's genotype, which would be partially shared with the children's in our sample.

Alternatively, mothers' genotype may render them more likely to parent their children in a manner more similar to how they were parented. Without having genotyped mothers, we cannot test this question, but it is a plausible alternative explanation for our results. Although our ancillary analyses comparing effects across the SS and SL groups attempted to mitigate this concern, these analyses may have been underpowered to detect the relatively small effects found in our primary analyses. It is therefore possible that mothers with a short allele at 5-HTTLPR may be more susceptible to the effect of their own experiences of being parented on their parenting. This possibility is consistent with recent findings from Beaver and Belsky (2012), who found that mothers who had several specific dopaminergic and serotonergic polymorphisms showed differential susceptibility to the effects of their mother's behavior on their own parenting behavior. Along a similar vein, mothers with a short allele show less sensitivity to toddlers (Bakermans-Kranenberg & van Ijzendoorn, 2008) and report poorer quality attachment to their infant (Mileva-Seitz et al., 2011). Mothers with a short allele at 5-HTTLPR also show less sensitive parenting in the context of high levels of interparental conflict (Sturge-Apple et al., 2012). If mothers in the current study had a short allele, their own predisposition towards being less sensitive to their children may be exacerbated by having had less caring mothers themselves. Thus, rather than children's genotype eliciting schemas or templates that were developed via mothers' own experiences, mothers with a short allele may instead be genetically susceptible to internalizing the parenting behaviors they received as children as a model for how to parent their own children. Thus, effects of child genotype may be due, in part, to genes shared between the child and mother. These two possible explanations should be explored in future research.

Regardless of which possibility is supported in future research, it raises the further question of why child or mother genotype would be associated with mothers' parenting in a similar manner to how they were parented themselves. We have suggested effects may be due to a relationship between child genotype and child characteristics which results in the activation of schemas or heuristics which were laid in place during the mothers' own childhoods. Alternatively, results may be due to mothers carrying a short allele on 5-HTTLPR, which rendered them more likely to internalize the parenting behaviors they received as templates or models for parenting. We suspect there will likely be multiple mediators linking child or mother genotype to the transmission of parenting, and know of no research that could inform hypotheses on the topic. However, there will also likely be both unconscious, automatic processes as well as more controlled, cognitive-affective mediators in the mother which account for the effects of child or mother genotype on the likelihood of parenting in a manner similar to their parents. For instance, mothers who, as children, acted in a non-rewarding or difficult manner similar to their own children's behaviour, may have experienced a withdrawal of support or increased hostility from their own mothers. If it is the mother's genotype driving results, some cognitive phenotype associated with a short allele at 5-HTT may mediate this effect and lead to these mothers being more likely to internalize the parenting behaviors they received. In turn, they may model these behaviours in their own parenting. Alternatively, if the child possessing a short allele increased their more challenging behaviors or traits, mothers who received negative parenting themselves may be more likely to resort to hostile or unsupportive behaviors towards their children.

Both possibilities, admittedly, are highly speculative and go well beyond our data. However, future research will likely benefit from examining maternal cognitive-affective processes that mediate the effect of child genotype and temperament on the transmission of parenting.

Limitations and future directions

Two important limitations other than those discussed above should be acknowledged. First, for a candidate gene study, our sample is relatively small, although we did test our models in two independent samples. Second, mothers' reports of their parenting experiences were assessed retrospectively. Although the PBI and MOPS appear to measure actual, not perceived parenting (Parker, 1987), show good test-retest stability over several decades (Lizardi & Klein, 2005; Parker et al., 1979), and are independent of personality (Duggan, Sham, Minne, Lee, & Murray, 1998) and mood and depressive symptoms (Parker, 1981; Parker, 1983), we cannot discount possible recall biases, although our results were not influenced by maternal depressive symptoms. It is also unclear whether results would generalize to observational or interview-based assessments of mothers' experiences of parenting.

As ongoing longitudinal studies mature, it may eventually be possible to examine the parenting experiences of youth via a variety of methods, follow them until adulthood, and assess their own parenting and their offspring's behaviour in multi-generational samples that have all been genotyped. This will permit investigators to more precisely disentangle the complex mechanisms that contribute to relationship between child or parent genotype and parental parenting behaviors. Future research should also examine whether our results hold for fathers as well as mothers.

Conclusion

This paper examined the associations between child genotype and mothers' support and hostility toward their children, and whether this relationship was moderated by the mothers' own parenting experiences. Results showed that, in children with a short allele on 5-HTTLPR, mothers displayed more hostility (Samples 1 and 2) and less support (Sample 1), but only if they experienced high levels of negative parenting themselves. These results further our understanding of the predictors of parenting, and take a step towards integrating the disparate literatures on the transmission of parenting and evocative gene-environment correlations, which has the potential to help elucidate the complex web of relationships involved in the transmission of parenting.

Acknowledgments

Supported by NIMH grant RO1 MH45757 (Klein); This research was supported by grants from the Canadian Institutes of Health Research (CIHR MOP86458; Hayden), and a NARSAD New Investigator Award (Hayden)

References

Avinun R, Knafo A. Parenting as a reaction evoked by children's genotype: A meta-analysis of children-as-twins studies. Personality and Social Psychology Review. 2014; 18:87–102. [PubMed: 23940232]

Bakermans-Kranenburg MJ, van IJzendoorn MH. Oxytocin receptor (OXTR) and serotonin transporter (5-HTT) genes associated with observed parenting. Social cognitive and Affective Neuroscience. 2008; 3:128–134. [PubMed: 19015103]

Bakermans-Kranenburg MJ, van IJzendoorn MH. Parenting matters: Family science in the genomic era. Family Science. 2010; 1:26–36.

Bauer DJ, Curran PJ. Probing interactions in fixed and multilevel regression: Inferential and graphical techniques. Multivariate Behavioral Research. 2005; 40:373–400. [PubMed: 26794689]

Beaver KM, Belsky J. Gene-environment interaction and the intergenerational transmission of parenting: testing the differential-susceptibility hypothesis. Psychiatric Quarterly. 2012; 83:29–40. [PubMed: 21553075]

Belsky J. The determinants of parenting: A process model. Child Development. 1984; 55:83–96. [PubMed: 6705636]

Belsky J, Conger R, Capaldi DM. The intergenerational transmission of parenting: introduction to the special section. Developmental Psychology. 2009; 45:1201–1204. [PubMed: 19702385]

Belsky J, Hancox RJ, Sligo J, Poulton R. Does being an older parent attenuate the intergenerational transmission of parenting? Developmental Psychology. 2012; 48:1570–1574. [PubMed: 22429005]

Belsky J, Jaffee SR, Sligo J, Woodward L, Silva PA. Intergenerational transmission of warm-sensitive-stimulating parenting: A prospective study of mothers and fathers of 3-year-olds. Child Development. 2005; 76:384–396. [PubMed: 15784089]

Belsky J, Pluess M. Beyond diathesis stress: differential susceptibility to environmental influences. Psychological Bulletin. 2009; 135:885. [PubMed: 19883141]

Bollen KA, Davis WR. Causal indicator models: identification, estimation, and testing. Structural Equation Modeling: A Multidisciplinary Journal. 2009; 16:498–522.

Bradley RH, Vandell DL. Child care and the well-being of children. Archives of Pediatrics & Adolescent Medicine. 2007; 161:669–676. [PubMed: 17606830]

Capaldi DM, Pears KC, Patterson GR, Owen LD. Continuity of parenting practices across generations in an at-risk sample: A prospective comparison of direct and mediated associations. Journal of Abnormal Child Psychology. 2003; 31:127–142. [PubMed: 12735396]

Conger RD, Neppl T, Kim KJ, Scaramella L. Angry and aggressive behavior across three generations: A prospective, longitudinal study of parents and children. Journal of Abnormal Child Psychology. 2003; 31:143–160. [PubMed: 12735397]

Conger RD, Belsky J, Capaldi DM. The intergenerational transmission of parenting: Closing comments for the special section. Developmental Psychology. 2009; 45:1276. [PubMed: 19702391]

Scaramella LV, Conger RD. Intergenerational continuity of hostile parenting and its consequences: The moderating influence of children's negative emotional reactivity. Social Development. 2003; 12:420–439.

Conger RD, Schofield TJ, Neppl TK. Intergenerational continuity and discontinuity in harsh parenting. Parenting. 2012; 12:222–231. [PubMed: 22754400]

Conger RD, Schofield TJ, Neppl TK, Merrick MT. Disrupting intergenerational continuity in harsh and abusive parenting: The importance of a nurturing relationship with a romantic partner. Journal of Adolescent Health. 2013; 53:S11–S17. [PubMed: 24059934]

Crockenberg SC, Leerkes EM. Infant temperament moderates associations between childcare type and quantity and externalizing and internalizing behaviors at years. Infant Behavior and Development. 2005; 28:20–35.

Cox M, Crnic K. Qualitative Ratings for Parent-Child Interaction at 3–15 months. 2003 Unpublished coding manual.

Duggan C, Sham P, Minne C, Lee A, Murray R. Quality of parenting and vulnerability to depression: results from a family study. Psychological Medicine. 1998; 28:185–191. [PubMed: 9483695]

Egeland, B., Weinfield, N., Hiester, M., Lawrence, C., Pierce, S., et al. Teaching tasks administration and scoring manual. Minneapolis, MN: University of Minnesota; 1995.

Friesen MD, Woodward LJ, Horwood LJ, Fergusson DM. Quality of parent–child relations in adolescence and later adult parenting outcomes. Social Development. 2013; 22:539–554.

Hankin BL, Oppenheimer C, Jenness J, Barrocas A, Shapero BG, Goldband J. Developmental origins of cognitive vulnerabilities to depression: review of processes contributing to stability and change across time. Journal of Clinical Psychology. 2009; 65:1327–1338. [PubMed: 19827008]

Hardt J, Rutter M. Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. Journal of Child Psychology and Psychiatry. 2004; 45:260–273. [PubMed: 14982240]

Hayden EP, Klein DN, Dougherty LR, Olino TM, Laptook RS, Dyson MW, … Singh SM. The dopamine D2 receptor gene and depressive and anxious symptoms in childhood: associations and evidence for gene–environment correlation and gene–environment interaction. Psychiatric Genetics. 2010; 20:304. [PubMed: 20526230]

Hayden EP, Klein DN, Sheikh HI, Olino TM, Dougherty LR, Dyson MW, … Singh SM. The serotonin transporter promoter polymorphism and childhood positive and negative emotionality. Emotion. 2010; 10:696. [PubMed: 21038952]

Hayden EP, Hanna B, Sheikh HI, Laptook RS, Kim J, Singh SM, Klein DN. Child dopamine active transporter 1 genotype and parenting: evidence for evocative gene–environment correlations. Development and Psychopathology. 2013; 25:163–173. [PubMed: 23398760]

Hayes, AF. PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling [White paper]. 2013. Retrieved from http://www.afhayes.com/public/process2013.pdf

Hayes AF, Matthes J. Computational procedures for probing interactions in OLS and logistic regression: SPSS and SAS implementations. Behavior research methods. 2009; 41:924–936. [PubMed: 19587209]

Hollingshead, AB. Unpublished manuscript. Department of Sociology, Yale University; New Haven, CT: 1975. Four factor index of social status.

Ispa JM, Fine MA, Halgunseth LC, Harper S, Robinson J, Boyce L, et al. Maternal intrusiveness, maternal warmth, and mother-toddler relationship outcomes: variations across low-income ethnic and acculturation groups. Child Development. 2004; 75:1613–1631. [PubMed: 15566369]

Jaffee SR, Price TS. Gene–environment correlations: a review of the evidence and implications for prevention of mental illness. Molecular Psychiatry. 2007; 12:432–442. [PubMed: 17453060]

Jaffee SR, Bowes L, Ouellet-Morin I, Fisher HL, Moffitt TE, Merrick MT, Arseneault L. Safe, stable, nurturing relationships break the intergenerational cycle of abuse: A prospective nationally representative cohort of children in the United Kingdom. Journal of Adolescent Health. 2013; 53:S4–S10.

Johnson PO, Neyman J. Tests of certain linear hypotheses and their application to some educational problems. Statistical Research Memoirs. 1936; 1:57–93.

Kendler KS, Baker JH. Genetic influences on measures of the environment: a systematic review. Psychological Medicine. 2007; 37:615–626. [PubMed: 17176502]

Kiser D, Steemer SB, Branchi I, Homberg JR. The reciprocal interaction between serotonin and social behaviour. Neuroscience & Biobehavioral Reviews. 2012; 36:786–798. [PubMed: 22206901]

Kopala-Sibley DC, Zuroff DC. The developmental origins of personality factors from the self-definitional and relatedness domains: A review of theory and research. Review of General Psychology. 2014; 18(3):137.

Kovan NM, Chung AL, Sroufe LA. The intergenerational continuity of observed early parenting: a prospective, longitudinal study. Developmental Psychology. 2009; 45:1205. [PubMed: 19702386]

Kryski KR, Smith HJ, Sheikh HI, Singh SM, Hayden EP. Evidence for evocative gene–environment correlation between child oxytocin receptor (OXTR) genotype and caregiver behavior. Personality and Individual Differences. 2014; 64:107–110.

Lengua LJ, Kovacs EA. Bidirectional associations between temperament and parenting and the prediction of adjustment problems in middle childhood. Journal of Applied Developmental Psychology. 2005; 26:21–38.

Lizardi H, Klein DN. Long term stability of parental representations in depressed outpatients utilizing the Parental Bonding Instrument. Journal of Nervous and Mental Disease. 2005; 193:183–188. [PubMed: 15729108]

Lucht M, Barnow S, Schroeder W, Grabe HJ, Finckh U, John U, … Herrmann FH. Negative perceived paternal parenting is associated with dopamine D2 receptor exon 8 and GABA (A) alpha 6 receptor variants: an explorative study. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics. 2006; 141:167–172.

Mathis ET, Bierman KL. Dimensions of Parenting Associated with Child Prekindergarten Emotion Regulation and Attention Control in Low-income Families. Social Development. 2015; 24:601–620. [PubMed: 26195853]

McLeod BD, Weisz JR, Wood JJ. Examining the association between parenting and childhood depression: a meta-analysis. Clinical Psychology Review. 2007; 27:986–1003. [PubMed: 17449154]

Mileva-Seitz V, Kennedy J, Atkinson L, Steiner M, Levitan R, Matthews SG, … Fleming AS. Serotonin transporter allelic variation in mothers predicts maternal sensitivity, behavior and attitudes toward 6-month-old infants. Genes, Brain and Behavior. 2011; 10:325–333.

Mills-Koonce WR, Propper CB, Gariepy JL, Blair C, Garrett-Peters P, Cox MJ. Bidirectional genetic and environmental influences on mother and child behavior: the family system as the unit of analyses. Development and Psychopathology. 2007; 19:1073–1087. [PubMed: 17931435]

Munafò MR, Freimer NB, Ng W, Ophoff R, Veijola J, Miettunen J, … Flint J. 5-HTTLPR genotype and anxiety-related personality traits: A meta-analysis and new data. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics. 2009; 150:271–281.

Neale MC, Walters E, Heath AC, Kessler RC, Pérusse D, Eaves LJ, Kendler KS. Depression and parental bonding: cause, consequence, or genetic covariance? Genetic Epidemiology. 1994; 11:503–522. [PubMed: 7713392]

Neppl TK, Conger RD, Scaramella LV, Ontai LL. Intergenerational continuity in parenting behavior: Mediating pathways and child effects. Developmental Psychology. 2009; 45:1241. [PubMed: 19702389]

NICHD Early Child Care Research Network. The effects of infant child care on infant-mother attachment security: Results of the NICHD study of early child care. Child Development. 1997; 68:860–879. [PubMed: 29106728]

Olino TM, Klein DN, Dyson MW, Rose SA, Durbin CE. Temperamental emotionality in preschool-aged children and depressive disorders in parents: associations in a large community sample. Journal of Abnormal Psychology. 2010; 119:468. [PubMed: 20677836]

Oppenheimer CW, Hankin BL, Jenness JL, Young JF, Smolen A. Observed positive parenting behaviors and youth genotype: Evidence for gene–environment correlations and moderation by parent personality traits. Development and Psychopathology. 2013; 25:175–191. [PubMed: 23398761]

Parker G. Parental reports of depressives: An investigation of several explanations. Journal of Affective Disorders. 1981; 3:131–140. [PubMed: 6454707]

Parker G. Parental affectionless control as an antecedent to adult depression: a risk factor delineated. Archives of General Psychiatry. 1983; 40:956–960. [PubMed: 6615158]

Parker G, Tupling H, Brown LB. Parental bonding instrument (PBI). British Journal of Medical Psychology. 1979; 52:1–10.

Parker G, Roussos J, Hadzi-Pavlovic D, Mitchell P, Wilhelm K, Austin MP. The development of a refined measure of dysfunctional parenting and assessment of its relevance in patients with affective disorders. Psychological Medicine. 1997; 27:1193–1203. [PubMed: 9300523]

Pauli-Pott U, Friedl S, Hinney A, Hebebrand J. Serotonin transporter gene polymorphism (5-HTTLPR), environmental conditions, and developing negative emotionality and fear in early childhood. Journal of Neural Transmission. 2009; 116:503–512. [PubMed: 19137235]

Pener-Tessler R, Avinun R, Uzefovsky F, Edelman S, Ebstein RP, Knafo A. Boys' serotonin transporter genotype affects maternal behavior through self-control: A case of evocative gene–environment correlation. Development and Psychopathology. 2013; 25:151–162. [PubMed: 23398759]

Pluess M, Velders FP, Belsky J, van IJzendoorn MH, Bakermans-Kranenburg MJ, Jaddoe VW, … Tiemeier H. Serotonin transporter polymorphism moderates effects of prenatal maternal anxiety on infant negative emotionality. Biological Psychiatry. 2011; 69:520–525. [PubMed: 21126730]

Schafer JL, Graham JW. Missing data: our view of the state of the art. Psychological Methods. 2002; 7:147. [PubMed: 12090408]

Schinka JA, Busch RM, Robichaux-Keene N. A meta-analysis of the association between the serotonin transporter gene polymorphism (5-HTTLPR) and trait anxiety. Molecular Psychiatry. 2004; 9:197–202. [PubMed: 14966478]

Sen S, Burmeister M, Ghosh D. Meta-analysis of the association between a serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety-related personality traits. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics. 2004; 127:85–89.

Sheikh HI, Hayden EP, Singh SM, Dougherty LR, Olino TM, Durbin CE, Klein DN. An examination of the association between the 5-HTT promoter region polymorphism and depressogenic attributional styles in childhood. Personality & Individual Differences. 2008; 45:425–428. [PubMed: 19122845]

Schofield TJ, Lee RD, Merrick MT. Safe, stable, nurturing relationships as a moderator of intergenerational continuity of child maltreatment: A meta-analysis. Journal of Adolescent Health. 2013; 53:S32–S38.

Serbin L, Karp J. Intergenerational studies of parenting and the transfer of risk from parent to child. Current Directions in Psychological Science. 2003; 12:138–142.

Sturge-Apple ML, Cicchetti D, Davies PT, Suor JH. Differential susceptibility in spillover between interparental conflict and maternal parenting practices: evidence for OXTR and 5-HTT genes. Journal of Family Psychology. 2012; 26:431. [PubMed: 22563705]

Wang M, Xing X, Zhao J. Intergenerational transmission of corporal punishment in china: the moderating role of marital satisfaction and gender. Journal of Abnormal Child Psychology. 2014; 42:1263–1274. [PubMed: 24915779]

Watson KK, Ghodasra JH, Platt ML. Serotonin transporter genotype modulates social reward and punishment in rhesus macaques. PLoS One. 2009; 4:e4156. [PubMed: 19142220]

Weinfield, NS., Egeland, B., Ogawa, JR. Promises to keep: Assessing affective and behavioral qualities of mother-child relationships in the New Chance Observational Study. Joint Center for Poverty Research Working Paper: Child Development. 1997. Retrieved from http://www.northwestern.edu/ipr/jcpr/workingpapers/childdev.html

Zimmerman M, Coryell W. The Inventory to Diagnose Depression (IDD): A self-report scale to diagnose major depressive disorder. Journal of Consulting and Clinical Psychology. 1987; 55:55. [PubMed: 3571659]

Zimmerman M, Sheeran T, Young D. The Diagnostic Inventory for Depression: a self-report scale to diagnose DSM-IV major depressive disorder. Journal of Clinical Psychology. 2004; 60:87–110. [PubMed: 14692011]

Zimmermann P, Mohr C, Spangler G. Genetic and attachment influences on adolescents' regulation of autonomy and aggressiveness. Journal of Child Psychology and Psychiatry. 2009; 50:1339–1347. [PubMed: 19769585]

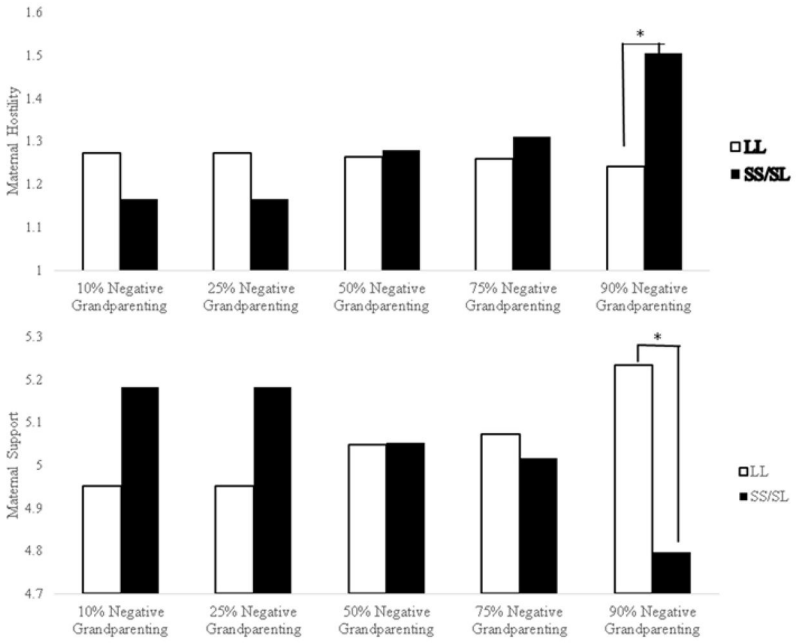

Figure 1.

* $p < .05$. Interaction between grandmother negative parenting and child genotype predicting maternal support and hostility in sample 1. Note: % refers to the Nth percentile (e.g., scores at the 10^{th} percentile).

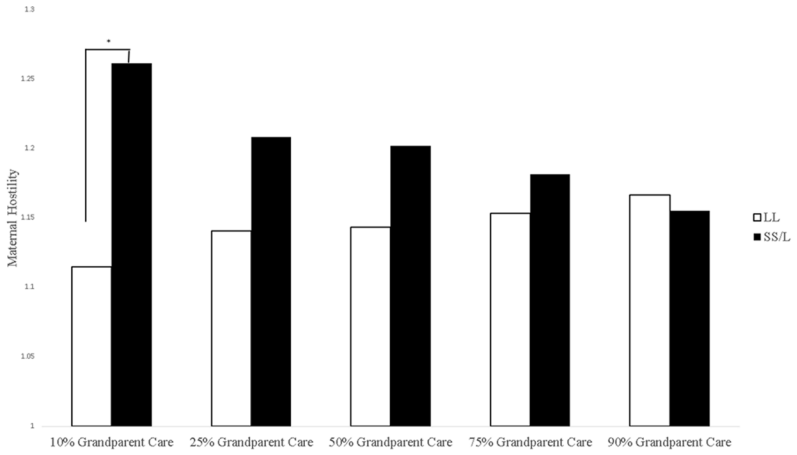

Figure 2.

* $p < .05$. Interaction between grandmother care and child genotype predicting maternal hostility in sample 2. Note: % refers to the Nth percentile (e.g., scores at the 10^{th} percentile).

Table 1

Bivariate correlations and descriptive statistics for all observed variables.

	1	**2**	**3**	**4**	**5**
1. 5-HTTLPR	--	−.02	−.06	−.01	.02
2. MOPS Negative Parenting		--	.43**	−.07	.17**
3. MOPS Overcontrol			--	.02	.01
4. Maternal Support				--	−.45**
5. Maternal Hostility					--
Mean	.69	2.76	3.40	5.04	1.29
SD	.46	4.33	2.80	1.26	166
	1	2	3	4	5
1. 5-HTTLPR	--	−.01	.02	.01	−.02
2. PBI Care		--	−.33**	−.08	.08
3. PBI Control			--	.02	−.03
4. Maternal Hostility				--	−.63**
5. Maternal Support					--
Mean	.70	10.98	7.47	4.47	1.63
SD	.46	1.95	2.45	.57	.23

Note:

** p < .01,

* p < .05,

+ p < .10.

Top half represents correlations for sample 1, bottom half for sample 2. PBI = Parental Bonding Inventory; MOPS = Measure of Parenting Styles; 5-HTTLPR coded as 1 = LL group, 2 = SS/SL group.

Table 2

Results of regression models predicting maternal hostility and support.

	b	SE	*t*	*p*	R^2
Sample 1 – Maternal hostility					.05
Serotonin	.04	.08	.52	.60	
Negative Parenting	−.01	.07	−.06	.95	
Over-control	−.04	.08	−.54	.59	
Serotonin * Negative Parenting	.19 *	.08	2.24	.03	
Serotonin * Over-control	−.02	.09	−.21	.83	
Sample 1 – Maternal Support					.03
Serotonin	−.11	.15	−.74	.46	
Negative Parenting	.13	.13	.61	.54	
Over-control	.15	.15	1.18	.26	
Serotonin * Negative Parenting	−.31 *	.16	−1.98	.048	
Serotonin * Over-control	−.12	.18	−.67	.50	
Sample 2 - Maternal Hostility					.02
Serotonin	.01	.04	−.14	.89	
PBI Care	.04	.03	1.05	.29	
PBI Control	.04	.03	1.27	.20	
Serotonin * Negative Parenting	.08 *	.04	−2.04	0.04	
Serotonin * Over-control	−.06	.04	−1.57	.11	
Sample 2 – Maternal Support					.01
Serotonin	.04	.06	.65	.51	
PBI Care	−.01	.06	−.23	.81	
PBI Control	−.06	.06	−1.09	.28	
Serotonin * Negative Parenting	.08	.07	1.08	.28	
Serotonin * Over-control	.08	.07	1.19	.24	

** $p < .01$,

* $p < .05$.

Note: PBI = Parental Bonding Inventory. Serotonin coded as 1= SS or SL genotype, 0 = LL genotype.