Published in final edited form as:
Soc Dev. 2014 August 1; 23(3): 433–450. doi:10.1111/sode.12041.

Development of Ego-Resiliency: Relations to Observed Parenting and Polymorphisms in the Serotonin Transporter Gene During Early Childhood

Zoe E. Taylor[a], **Michael J. Sulik**[a], **Nancy Eisenberg**[a], **Tracy L. Spinrad**[b], **Kassondra M. Silva**[b], **Kathryn Lemery-Chalfant**[a], **Daryn A. Stover**[c], and **Brian C. Verrelli**[c]

[a] Department of Psychology, Arizona State University

[b] School of Social and Family Dynamics, Arizona State University

[c] Center for Evolutionary Medicine and Informatics & School of Life Sciences, Arizona State University

Abstract

We used observed parenting behaviors, along with genetic variants and haplotypes of the serotonin transporter gene (SLC6A4), as predictors of children's ego-resiliency during early childhood (N=153). Quality of mothers' parenting was observed at 18 months of age and mothers' reports of ego-resiliency were collected at six time points from 18 to 84 months. Genetic data were collected at 72 months. Observed parenting was positively associated with initial levels of children's ego-resiliency. Furthermore, although individual genetic variants of the serotonin transporter gene (LPR, STin2) were not associated with ego-resiliency, the S10 haplotype (that combines information from these two variants) was negatively associated with initial levels of ego-resiliency. Both parenting and serotonin genetic variation uniquely predicted children's ego-resiliency, suggesting an additive effect of genetic and parental factors.

Keywords

ego-resiliency; parenting; SLC6A4; serotonin transporter

Ego-resiliency is an individual characteristic that reflects adaptability to environmental stress and change (Block & Block, 1980). In general, children who have higher levels of ego-resiliency are better able to adapt to changing or stressful circumstances, shift behaviors as needed, and use problem-solving strategies flexibly. In contrast, children with relatively low ego-resiliency display little adaptive flexibility, are disquieted by new and changing circumstances, have the tendency to perseverate or become disorganized when dealing with stress, and have difficulty recouping after traumatic experiences (Block & Block, 2006). Block and Block (1980) suggested that individual differences in ego-resiliency can be observed early in life through examining how infants respond to environmental changes, how they can be comforted, and how they equilibrate physiological responses. At age 2,

Correspondence regarding this article should be addressed to Zoe E. Taylor, Department of Psychology, Arizona State University, Tempe, AZ 85287-3701. zetaylor@asu.edu.

characteristics such as, such as having a warm and positive relationship with caregivers, being effective at finding comfort when needed, confronting problems persistently and enthusiastically, and finding pleasure in mastery are considered reflective of children's ego-resiliency (Arend, Gove, & Sroufe, 1979). As children reach preschool age, ego-resiliency is evident in how a child is able to express affect in a situationally appropriate manner, and whether they are organized, persistent, and flexible when encountering challenges and stressors (Arend, Gove, & Sroufe, 1979; Block & Block, 1980). Ego-resiliency has been associated with a range of positive developmental outcomes across childhood including social competence (Block & Block, 1980; Cumberland-Li, Eisenberg, & Reiser, 2004), low externalizing and internalizing symptoms (e.g. Chuang, Lamb, & Hwang, 2006; Hofer, Eisenberg, & Reiser, 2010), and cognitive functioning and attentiveness (e.g. Martel et al., 2007). Given these demonstrated associations with positive development and emotional adjustment, it is important to examine factors that contribute to individual differences in children's ego-resiliency.

The Blocks (1980, 2006) hypothesized that both genetic and experiential influences contribute to children's ego-resiliency. In terms of environmental factors, it is believed that nurturant, supportive parenting fosters ego-resiliency in children (Block & Block, 1980). Supporting this assertion, Stams, Juffer, Van IJzendoorn, and Hoksbergen (2001) found that an intervention to improve maternal sensitivity and responsiveness when children were six and nine months of age predicted higher levels of ego-resiliency at age seven. In contrast, controlling, intrusive parenting has been negatively associated with ego-resiliency during early childhood (Taylor, Eisenberg, Spinrad & Widaman, in press). Lower levels of ego-resiliency have also been found in toddlers who display contradictory attachment behaviors when under stress (Smeekens, Riksen-Walraven, & Van-Bakel, 2009) and in maltreated school-age children (Shonk & Cicchetti, 2001). Sensitive parents most likely foster ego-resiliency by assisting their children in coping with stressful situations, modeling effective coping strategies, providing appropriate support when children are overwhelmed, and exposing them to manageable, age-appropriate stressors. In contrast, overprotective, intrusive or harsh parents are more likely to discourage children's independent behaviors and undermine the development of independent coping skills (Eisenberg, Cumberland, & Spinrad, 1998; Power, 2004).

Genetic influences are also likely to contribute to individual differences in children's ego-resiliency (Block & Block, 2006; Lemery-Chalfant, 2010). Some of the first sources of individual differences in personality are the result of biologically based temperamental characteristics (Derryberry & Rothbart, 1997). Genetic variation can be viewed as contributing to positive adaptation or to maladjustment (Rutter, 2006) and can also affect an individual's sensitivity to components of the environment (Shanahan & Hofer, 2005). In particular, it has been hypothesized that some individuals, for genetic or temperamental reasons, are more reactive than others to variation in both positive and negative environmental contexts, a concept referred to as differential susceptibility (Belsky & Pluess, 2009; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). However, although researchers are increasingly examining the role of genetic mechanisms in the development of successful adaptation to challenge and adversity (e.g. Kim-Cohen, & Gold, 2009; Lemery-Chalfant, 2010), thus far genetic contributions to the construct of ego-

resiliency have not been examined in children. The present study, therefore, examined the relations of children's ego-resiliency with the serotonin transporter (SLC6A4, or 5HTT) gene. It was plausible that this gene is linked to children's ego-resiliency because variation in SLC6A4 has been associated with differential reactivity to environmental stress exposure and, in turn, with psychological health such as elevated stress sensitivity, depression, and neurotism (for a review, see Caspi, et al., 2010). Thus, this gene appears linked to constructs that could be viewed as the inverse of ego-resiliency.

Serotonin Transporter Gene

Two polymorphisms of the serotonin transporter gene have usually been examined with respect to their relation with behavioral variation. The first polymorphism, the "linked polymorphic region" (LPR; or 5HTTLPR), has two common repeat alleles--short (S; 14 repeat) or long (L; 16 repeat)--that differentially modulate the transcription of SLC6A4, with the S allele demonstrated to have less transcriptional efficiency (Greenberg et al., 1999; Hanna et al., 1998; Heinz et al., 2000). Because the S allele is believed to be functionally dominant, SS and SL genotypes are often grouped together and compared with the LL genotype in analyses. The second, but less studied, polymorphism is the functionally relevant "serotonin transporter intron 2" (STin2; Ogilvie et al., 1996), which consists of a 17-base pair repeat in the second intron. Common repeats of STin2 are the 10 and 12 repeat alleles, the latter of which has been related to increased transcriptional effects (Fiskerstrand, Lovejoy, & Quinn, 1999).

In addition to considering LPR and STin2 individually, researchers have begun to examine SLC6A4 haplotypes, or combinations of these two polymorphisms that are inherited together (e.g., Sulik et al., 2012; Perea et al., 2012). Haplotypes can produce unique functional differences and responses not seen by either variant independently. For example, Ali et al. (2010) found that the LPR-S and STin2-12 (S12) haplotype possessed the highest SLC6A4 transcriptional expression compared with lower expression for the L10 haplotype. In addition, Claw, Tito, Stone, and Verrelli (2010) found specific S haplotypes differed in frequencies and combinations across ethnic populations, which together with work such as that of Ali et al. (2010), likely contributes to the inconsistencies of genotype-phenotype associations using single variants at this gene.

Serotonin and Resiliency

Although researchers have not specifically examined the relations between the serotonin transporter gene and the construct of ego-resiliency in children, one study found that college students with at least one S allele have relatively lower levels of self-reported emotional resilience, defined as an individual's ability to withstand and bounce back from stress (Stein, Campbell-Sills, & Gelernter, 2009). Furthermore, researchers have found that SLC6A4 is associated with other developmental outcomes that are likely linked to levels of children's ego-resiliency. Literature using the LPR polymorphism appears to demonstrate that children with at least one S allele are more susceptible to distress and are more vulnerable to maladjustment when exposed to stressful situations. For example, Lakotos and colleagues (2003) found that infants homozygous for the S allele were more anxious when approached

by a strange person. Researchers have also found that infants with an S allele have higher levels of irritability at 8 and 32 weeks (Ivorra et al., 2010). Psychological symptoms, such as depression and negative emotionality, are also more evident in children with at least one S allele (Hankin et al., 2011; Hayden et al., 2010; Kaufman et al., 2006; Pluess et al., 2011). Children with the S allele have also been found to have poorer self-control on a delay task (Sonuga-Barke et al., 2011), higher levels of conduct problems when perceiving high levels of discrimination (Brody et al., 2011), and are more likely to exhibit distress intolerance by choosing to quit a challenging task (Amstadter et al., 2012).

In addition, biological stress reactivity has been associated with the SLC6A4 gene. Girls with an S allele have higher waking cortisol levels than their L allele counterparts and also produce higher and more prolonged levels of cortisol in response to a stressor (Chen, Joormann, Hallmayer, & Gotlib, 2009; Gotlib, Joormann, Minor, & Hallmayer, 2008). On the other hand, children with the LL genotype had the highest salivary alpha-amylase response to a stressful task, but also the steepest recovery, compared to children with at least one S allele (Mueller et al., 2011). The latter investigators concluded that this difference could reflect a more dynamic system and might relate to higher resilience to stress for children with the LL genotype.

The second serotonin transporter polymorphism, STin2, has been less studied in regards to its relations with resiliency and stress and findings are mixed. Few researchers have examined these relations in children, although Ivorra and colleagues (2010) reported that infant irritability was not associated with variation of the STin2 polymorphism, although it was with the LPR. Studies with adults have found that having a 12 allele (as well as the S12 haplotype) was associated with higher symptoms of depression and anxiety in college students (Perea et al., 2012). However, other researchers have found that the association between having a 12 allele and negative affect depends on the environment. For example, Mitchell and colleagues (2011) found that new mothers with a 12 allele had more mental health problems in unfavorable environments (lower SES), whereas the genetic effect changed directions when the environment became more positive–mothers with a 12 allele had less symptoms in favorable environments, suggesting evidence of the STin2 reflecting a plasticity gene.

Parenting behaviors, such as responsiveness, have also been found to interact in combination with the serotonin transporter gene and differentiate developmental outcomes. For example, Kochanska, Kim, Barry, and Philibert (2011) reported that insecurely attached infants with an S allele developed poorer self-regulation skills in preschool, whereas genetic variation did not differentiate regulatory capacities for securely attached infants. Another study found that infants who were homozygous for the S allele had higher levels of fear and negative emotionality if they were insecurely attached to their caregiver (Pauli-Pott, Friedl, Hinney, & Hebebrand, 2009). Researchers also report that having an L allele is protective. Gilissen and colleagues (2008) found that 7-year-olds with a secure attachment as well as two L alleles were less stressed during a public speaking task than those with an insecure attachment or an S allele. In support of the differential susceptibility hypothesis, individuals with specific haplotypes may also demonstrate greater reactivity to both supportive and unsupportive environments. For example, Sulik and colleagues (2012) found that the quality

of early parenting was negatively associated with increases of noncompliance only for children with the S10 haplotype.

The Present Study

The aforementioned studies provide initial support for an association between genetic variation in SLC6A4 and children's ego-resiliency, a trait associated with adaptability to stress, regulation, as well as psychological flexibility and functioning. Prior research also suggests that parenting behaviors may interact with polymorphisms of SLC6A4 in predicting developmental outcomes. In the present study, we examined the prediction of ego-resiliency and its development in early childhood by observed parenting at 18 months of age and polymorphisms in the serotonin transporter gene (LPR and STin2). The LPR and STin2 polymorphisms were tested independently and as haplotypes, as well as in interaction with quality of observed maternal parenting, as predictors of children's initial level and growth in mother-reported ego-resiliency from 18 to 84 months of age. Child sex was also included in the analysis as a main effect and in interaction with genes. Although child sex was included as a potential moderator, we did not expect there to be sex differences in terms of children's ego-resiliency because these differences are typically evident in adolescence and not in early childhood (Block & Block, 2006). Other covariates included household income and years of mothers' and fathers' education, which we expected to be positively associated with parenting behaviors. Outcome measures were the 18-month initial level of ego-resiliency and the linear slope from 18 to 84 months.

We expected that supportive parenting to be positively associated with ego-resiliency and that genetic variants of the serotonin transporter gene (LPR, STin2), as well as the

haplotypes (that combine information from both polymorphisms), would be differentially associated with children's ego-resiliency. Additionally, it was expected that children with the lowest serotonin transporter gene transcriptional expression (the S and the 10 alleles) would have the lowest levels of ego-resiliency. Given the mixed findings previously regarding the STin2 polymorphism, it was less evident that the 10 allele alone would differentially be associated with ego-resiliency. However, given previous studies have shown haplotype combinations differ functionally for serotonin levels as well as with respect to measures of resiliency, we did expect that children with the S10 haplotype would differ from those with other haplotypes. We also hypothesized that parenting might moderate these relations and might provide evidence of differential susceptibility.

Method

Participants and Procedures

Participants were a subsample from a longitudinal study of children's emotions, regulation, and social functioning in a large metropolitan area. The larger sample collected data when the child was approximately 18, 30, 42, 54, 72, and 84 months old (*ns* = 256, 230, 210, 188, 162,153). Families completed questionnaires, and mothers and children participated in 1.5-2 hour laboratory visits at 18, 30, 42, and 54 months, a home visit at 72 months, and returned questionnaires by mail at 84 months (*N*s = 247, 223, 205, 189, 162, and 144). Samples for

genetic analyses were collected in the home when the children were 72 months old (see below). Children were included in the present study if they had SLC6A4 genetic data, observed parenting data at 18 months, and at least one mother-report index of ego-resiliency (N = 153, 55.6% boys). The sample was 86.3% White, 3.3% African American, 2.6% Native American, 1.3% Asian, and 6.5% other; with 20% reporting Hispanic/Latino ethnicity. The median annual family income ranged from $45,000 to $60,000. The majority of mothers had some college and most (83%) families were two-parent families.

We examined mean differences on study variables between children included in our analysis and children who were not included due to missing genetic or observed parenting data. Children in the present sample had near significantly higher family incomes (ranging from 1 = less than $15,000 to 7 = over $100,000; M = 4.26) compared to those without genetic data (M = 3.82), $t(226) = 1.93$, $p = .055$, and higher levels of mothers' education (ranging from 1 = grade school to 7 = Ph.D. or M.D.; M = 4.41) compared to those without genetic data (M = 4.10), $t(238) = 2.20$, $p < .05$. There were no significant differences for fathers' education or for the ego-resiliency and observed parenting variables.

Measures

Children's ego-resiliency—Ego-resiliency was measured using an 11-item adapted questionnaire version of Block and Block's (1980) Q-sort. This shortened scale was constructed by Eisenberg and colleagues (1996, 2003) in order to reflect a purer version of resiliency that did not overlap with other constructs, such as negative emotionality. After three experts eliminated some items that reflected constructs such as social competence (Eisenberg et al., 1996), six faculty and 5 graduate students with relevant expertise rated the resiliency items as to how much they reflected pure resiliency from 1 (not at all descriptive of resiliency) to 9 (most descriptive of resiliency). Resiliency was defined as flexible, adaptable behavior. Only the 11 items that obtained a mean score of 6.0 or above (absolute value) were retained (e.g., **"**Can bounce back or recover after a stressful or bad experience,**"** **"**Freezes up when things are stressful, or else keeps doing the same thing over and over again**"**), although a 7-item version has sometimes been used (Cumberland-Li et al., 2004). All correlations between the original scale and the 11-item scale were above .83 (mean r = . 91). This shortened scale has been used reliably in numerous studies with children ranging in age from toddlers to adolescents (e.g. Eisenberg et al., 2004; Martel et al., 2007; Taylor et al., in press).

For the present study, mothers rated children's ego-resiliency (1 = highly undescriptive to 9 = highly descriptive) at 18, 30, 42, 54, 72 and 84 months. Negative Items were reverse-coded so that values reflected higher levels of ego-resiliency. One item was dropped ("Can talk about unpleasant things that have happened to him/her) because it was not asked at 18 and 30 months due to being inappropriate for the ages assessed. This item was not included at the later ages in order to make the scales equivalent across our six time-points (αs ranged from .57 to .78; M = .71).

Observed parenting—Coders assessed mothers' behavior (maternal sensitivity, intrusiveness, warmth and verbal control) at 18 months from videotapes of mother-child

interactions during a lab teaching task, free-play task, and clean-up task, each lasting 3 minutes. In the free-play task, mothers were given a basket of toys and instructed to play with their child as they would at home. In the teaching task, participants were given a challenging puzzle, and mothers were asked to teach their child to complete the puzzle. During the clean-up task, mothers were instructed to have their child pick up toys and put them in a basket.

Maternal *sensitivity* and *intrusiveness* were assessed every 15 seconds during the free-play and teaching tasks. *Sensitivity* was rated as 1 = no evidence of sensitivity to 4 = mother was very aware of the toddler, contingently responsive to his or her interests and affect, and had an appropriate level of response/stimulation (interrater reliabilities [ICCs] = .81 and .82, for free-play and teaching tasks, respectively). Mothers' *overcontrolling, intrusive* behaviors included overstimulating the child with toys, employing intrusive physical interactions, or intervening to help the child when not required (1 = no overcontrolling behavior observed; 4 = extreme intrusive or overcontrolling behaviors; ICCs = .82 for free play and .81 for the teaching task). Maternal *warmth*, coded every 30 s during the teaching task, reflected mothers' displays of closeness, friendliness, physical affection, encouragement, and positive affect with the child (1 = no evidence of warmth; 5 = positive affect was predominant; ICC = .83). Maternal *verbal control*, rated every 15 s during the clean-up task, reflected verbal directives that were given by mothers in an assertive or controlling manner (1 = present, 0 = absent; ICC = .70). All parenting measures were correlated in expected directions. Absolute values of the *r*s ranged from .19 to .85, all *p*s < .05. We created a composite of parenting quality by reverse scoring maternal verbal control and intrusiveness, standardizing scores, and computing the average (as in blinded for review).

SLC6A4 genetic variants—Genetic data were collected at 72 months. Buccal cheek samples were collected from children with a cotton swab and DNA extractions were performed using standard isolation techniques (Sambrook & Russell, 2001). Two sets of PCR (Polymerase Chain Reaction) primers were designed to generate two DNA fragments. Primers designed for LPR were GGC GTT GCC GCT CTG AAT GC (Forward) and GAG GGA CTG AGC TGG ACA ACC AC (Reverse), which amplified fragments of 484-base pairs (S allele) to 528-base pairs (L allele). Primers designed for STin2 were TGG ATT TCC TTC TCT CAG TGA TTG G (Forward) and TCA TGT TCC TAG TCT TAC GCC AGT G (Reverse), which amplified fragments of 355- base pairs (10 allele) to 389-base pairs (12 allele). PCR fragments were evaluated and scored via direct gel electrophoresis visualization of 2% agarose gels in 1X Sodium Borate buffer with Ethidium Bromide staining. As previously documented (blinded for review), a set of 10 VNTRs (variable number of tandem repeats), sampled from across the genome, were genotyped for all individuals and showed no significant evidence of population admixture (using the STRUCTURE program; Pritchard, Stephens, & Donnelly, 2000). Thus, no subsequent statistical analyses to structure our data into different groups (i.e., by ethnicity) were necessary.

The polymorphisms LPR and STin2 were statistically analyzed independently and as haplotypes. As noted previously, because the S allele is believed to be functionally "dominant," SS ($N = 29$) and SL ($N = 70$) genotypes were grouped together ($N = 99$) and

compared with the LL genotype (N = 54) in analyses for LPR. For STin2, we compared children with the 10-10 (N = 15), 10-12 (N = 83), and 12-12 (N = 55) genotypes. Chi-squared tests of independence confirmed that genotype frequencies for both polymorphisms were consistent with Hardy– Weinberg equilibrium in the overall sample (LPR: χ^2 = 0.55, p = .46; STin2: χ^2 = 3.20, p = .09), which is an important assumption for all of our statistical analyses (e.g., Hedrick, 2010). The PHASE v. 2.1.1 program (Stephens, Smith, & Donnelly, 2001) was used to statistically estimate how the genotypes at the two variants LPR and STin2 within individuals form haplotypes. Haplotype frequencies of L10 = 32%, L12 = 26%, S10 = 6%, and S12 = 36% replicated those in global sampled populations (Claw et al., 2010).

As previously noted, the LPR-S and STin2-10 alleles have been related to reduced serotonin function, yet others have suggested that the S12 haplotype is associated with increased serotonin function (Ali et al., 2010). Given this functional evidence, we predicted that individuals with either the S10 and S12 haplotypes would be different compared to other haplotypes. Thus, as in our previous work (blinded for review), instead of comparing simply S vs. L haplotypes, we further divided the data into three groups based on this rationale. Group 1 (called “S10 group”) consisted of S10-S12, S10-L10; these individuals had the critical S10 haplotype (no children had the rare S10-S10 or S10-L12). Group 2 (called “S12 group”) included S12-S12, S12-L10, and S12-L12. Group 3 (called “L10-L12 group”) included haplotypes L10-L10, L10-L12, and L12-L12.

Covariates—Household income and mothers’ and fathers’ education were included as main effects. Child sex was included as a main effect and in interaction with the polymorphisms/haplotype.

Results

Descriptive Analysis

We first examined descriptive data and correlations among key variables. Means and standard deviations for variables are presented in Table 1. No variables needed to be transformed due to excessive skew or kurtosis. There were no sex differences in measures of parenting or ego-resiliency at any time point. Chi-squared tests examining the sex distribution across the SLC6A4 genotypes (LPR-SS/SL vs. LL; STin2-10-10 vs. 10-12 vs. 12-12) were not significant. Correlations among variables are shown in Table 2. Rated ego-resiliency was significantly interrelated across all time points, rs = .23 to .65, all ps < .05. Observed parenting at 18 months and ego-resiliency were concurrently associated at 18 months, and marginally significant at other time points. Being in the S10 haplotype group was negatively associated with ego-resiliency across time points.

We also examined whether variables differed between based on genetic groups using t-tests. No significant differences were found between ego-resiliency and parenting for either the LPR or STint2 polymorphisms. Observed parenting did not vary by the three haplotype groups; however, independent sample t-tests showed differences for ego-resiliency—children in the S10 haplotype group were significantly lower in ego-resiliency relative to children in the other two groups at 30 months, [t (146) = 2.33, p < .05; mean difference = .

66, standard error = .28]; and at 54 months [t (148) = 3.66, p < .01; mean difference = .93, standard error = .25], and at a trend level at 42 [t (148) = 1.73, p = .09; mean difference = . 43, standard error = .25] and 84 months [t (128) = 1.80, p = .08; mean difference = .53, standard error = .29].

We then used multilevel modeling (i.e., mixed effects or hierarchical linear modeling) to predict initial levels and change in mother-reported ego-resiliency from 18 to 84 months of age. First, we examined the best-fitting random effects model without substantive predictors. This model formed the basis of subsequent analyses and described the average trajectory of children's ego-resiliency in our sample. Then we tested the substantive models using the genetic variants and parenting, as well as their interactions, as predictors of children's levels and growth of ego-resiliency.

Random Effects Model

Adopting Singer and Willett's (2003) recommendation, we used a model-building approach. We began with a random intercept model with time centered at 18 months and added fixed and random effects for time and quadratic time until model fit could no longer be improved; likelihood ratio tests were used to compare the fit of nested models. The best fitting random effects model for ego-resiliency was a linear growth model with fixed and random effects for the intercept and linear slope.

Fixed and random effects

With time centered at 18 months, the intercept for ego-resiliency was 6.99 (SE = .06, p < .01). We also tested a model with the intercept centered at 84 months. The results were the same; thus, we choose to keep the intercept at 18 months. The fixed effect of time was nonsignificant, indicating that there was no average change in ego-resilience over time in our sample. The residual (within-person) variance was .41 (SE = .03, p < .01) for ego-resiliency. The variance of the random effects for ego-resiliency was .30 (SE = .06, p < .01) for the intercept and .01 (SE = .00, p < .01) for the linear slope, indicating that there were individual differences in the slope of children's ego-resiliency. The covariance between the intercept and linear slope was not significant.

Substantive Models

We next added predictors to the models in two steps. First, we examined the main effects of the genetic predictor(s), parenting, and child sex. In an additional model, we added the G x E interaction(s) and the interaction with child sex. For both of these models, we tested whether each set of predictors provided additional predictive power over the prior model. In addition, for each model, we calculated a pseudo r^2 value by computing the squared correlation between the model-predicted scores and the observed scores; we examined the increase in this statistic corresponding to the addition of each set of predictors (see Table 3).

In separate models for LPR and STin2, there were no significant main effects or interactions with parenting for either the 18-month or 84-month intercept or the slope of ego-resiliency (see Table 3). In the haplotype model, we found differences between the S10 group compared to the L10–L12 and S12 groups. Specifically, the S10 group negatively predicted

the intercept of ego-resiliency (β = −.52, *SE* = .19, *t* = −2.78, *p* < .01), but not the linear slope. This model fit the data better than the random effects models, χ^2 (2) = 7.92, *p* < .01. We then added observed parenting into the model. Both parenting (β = .22, *SE* = .09, *t* = 2.57, *p* < .01) and S10 status (β = −.54, *SE* = .19, *t* = −2.82, *p* < .01) uniquely and significantly predicted the 18-month intercept of children's ego-resiliency. Neither parenting nor the haplotypes predicted the linear slope. This model also fit the data better than the random effects models, χ^2 (3) = 14.3, p < .01. Interactions between the haplotype groups and parenting were nonsignificant. Child sex was not associated with ego-resiliency as a main effect or in interaction with genetic variables. Our other covariates (household income, mothers' education, and fathers' education, all reported at 18 months) also did not significantly predict ego-resiliency in the regression model. However, as shown in Table 2, income and both mothers' and fathers' education was significantly correlated with observed parenting as we expected.

Discussion

The present study supports the hypothesis that antecedents of the personality trait of ego-resiliency appear to include both socialization and genetic factors (Block & Block, 1980). We found that observed parenting at 18 months was positively related to children's ego-resiliency, consistent with prior studies (e.g. Stams et al., 2001; Taylor et al., in press). Furthermore, our study was, to the best of our knowledge, the first to find associations between ego-resiliency and the serotonin transporter gene (SLC6A4) in children (and the only one to examine serotonin haplotypes as predictors of ego-resiliency). Although the genetic variants in isolation did not predict ego-resiliency, haplotype combinations of the LPR and STin2 polymorphisms did. Specifically, children in the S10 haplotype group had lower initial levels of ego-resiliency than those in the other groups. Genetic variation does not always combine 'additively' when explaining phenotypic variance, in that functional combinations across polymorphisms, whether they be in the same gene or across genes, are much more likely to explain complex trait variation (Belsky & Beaver, 2011; Claw et al., 2010). This finding suggests that individual differences in children's ego-resiliency are partially due to variation in SLC6A4 haplotypes. Given the stability of ego-resiliency, it is likely that this haplotype predicts ego-resiliency well into childhood and perhaps beyond. Also of interest, the relation between genetic variants and ego-resiliency remained significant after including parenting in the model.

The present study did not find a gene x parenting interaction when predicting ego-resiliency; although prior research with this sample did detect a significant SLC6A4 haplotype x parenting interaction in regards to the development of compliance in children (Sulik et al, 2012). However, Stein and colleagues (2009) also did not find significant gene x environment interactions when predicting personality resilience. Because our sample was relatively low-risk, it could be the case that a more significant stressor (i.e., harsh parenting) would be needed in order to find a significant interaction that is associated with ego-resiliency, particularly because ego-resiliency might be most evident in adverse environmental contexts. However, the finding that both parenting and genetic variation provided significant and unique prediction of children's ego-resiliency supports an additive effect.

Although we did not find an individual genetic effect for the SLC6A4 LPR, our findings are generally consistent with Stein and colleagues (2009) who found that having an S allele was associated with lower levels of reported emotional resilience in college students. Importantly, we extended this prior work to early childhood. Furthermore, because we find significant results when examining haplotype groups, our observations provide more resolution and even stronger support for the role of the SLC6A4 gene and serotonin on ego-resiliency. Specifically, although associations with the S allele alone have been documented in previous studies (e.g., Caspi et al. 2010), this current work indicates that this allele alone may not always be the driving influence for behavior-related traits, but that only in combination with other genetic variants (e.g., the STin2-10 allele), a significant outcome is seen. This result is somewhat predicted by even previous functional work (e.g., Ali et al., 2010) with the S allele showing that in combination with the 10 allele, a significantly lower level of gene expression was possible. This functional association between haplotypes and gene expression predicts differences in serotonin expression (albeit possibly subtle associations for certain traits vs. others), even between the S10 and S12 groups, as is seen in the current study.

As ego-resiliency is assumed to reflect adaptability to environment stress and change, our findings are consistent with research that has found individuals with S alleles have higher sensitivity to stress and negative affect (Caspi et al., 2010; Gotlib et al, 2008; Pluess et al., 2011). A potential pathway linking SLC6A4 and ego-resiliency could be via the effect of the serotonin transporter gene on physiological functioning. Ellis and colleagues (2011) found that adults with short 5-HTTLPR alleles had significantly lower resting respiratory sinus arrhythmia (RSA) than those with long alleles. RSA is believed to reflect how individuals physiologically regulate emotion (Ellis, Beevers, Hixon, & McGeary, 2011). Our findings suggest that genetic variability in the serotonin transporter may also contribute to individual differences in the personality characteristic of ego-resiliency, although further study and replication need to be undertaken before firm conclusions can be made.

Lastly, although parenting and genetic variation predicted the intercept of ego-resiliency, neither of these factors predicted the slope. However, the same effects were found at 18 and 54 months of age. Ego-resiliency is fairly high in rank-order stability across early childhood (Block and Block, 2006), which suggests that individual differences in ego-resiliency may emerge at an early age and are relatively stable. Our findings are consistent with this hypothesis and underscore the importance of assessing antecedents of ego-resiliency, such as parenting, during early childhood. However, it is probable that the links between maternal parenting and children's ego-resiliency could result from shared genetic heredity. Future research that uses multi-method ways of examining ego-resiliency will be important.

Of note, there was significant variance around the slope that was not predicted by the variables in our study. Few researchers have examined trajectories of growth in ego-resiliency. Kim and colleagues (2009) found that for maltreated children, earlier onset of maltreatment was related to a low and decreasing trajectory of ego-resiliency and higher levels of internalizing symptoms compared to maltreated children with a later onset of maltreatment. However, slopes between nonmaltreated and maltreated children did not differ. It is possible that other temperamental characteristics contribute to the development

of ego-resiliency. For example, researchers have found that temperamental traits such as conscientiousness, openness to new experiences, extraversion and emotional stability explain variance in ego-resiliency in both children and adolescents (Asendorpf, & van Aken, 1999; Nakaya, Oshio, & Kaneko, 2006; Robins, John, Caspi, Moffitt, & Stouthamer-Loeber, 1996). More research is needed to determine what factors contribute to explaining variance in trajectories of ego-resiliency, especially in early childhood.

Strengths of the current study include the use of observed—rather than self-reported—parenting, application of a growth curve analysis, the inclusion of multiple genetic variants investigated separately and combined as haplotypes, and a longitudinal design. Our study is limited by the moderate sample size and our inability to examine some genotypes by themselves (e.g. the S10-S10). However, individuals with two S10 haplotypes are very rare (3% worldwide; Claw et al., 2010) and a larger sample is unlikely to have included significant numbers of this group. Focusing on predominantly single ethnic groups helps analyses from both statistical and interpretational views; however, we note that we do not necessarily expect our findings to generalize to children in other cultural or ethnic groups. In fact, these results support the need to conduct similar analyses in other diverse groups for comparison given the documented ethnic differences in parenting influences (e.g. Deater-Deckard et al., 2011; Martin, Fisher, & King, 2012), as well as in haplotype combinations (Claw et al. 2010). Also important, because associations between genes and developmental adjustment have a history of being inconsistent, the association between serotonin haplotypes and ego-resiliency should be replicated with other samples (Risch et al., 2009).

In general, our findings demonstrated the importance of looking at combinations of genetic variants as haplotypes in coordination with environmental variables when examining developmental characteristics in children; if we had examined only single genetic variants, we would not have found a relation between serotonin and ego-resiliency. Researchers are increasingly finding individual differences in adjustment are predicted by a combination of genetic variants and even multiple genes, whether this be by direct interaction or even by modification of one by another (Kim-Cohen & Gold, 2009). In particular, because resilience involves intrinsic and extrinsic processes of successful adaptation to adversity, it will be important to determine what additional genetic, individual, and environmental factors interact to predict ego-resiliency in children (Lemery-Chalfant, 2010). Future research should continue to address the ways in which genetic combinations contribute to development of children's personality traits such as ego-resiliency.

Acknowledgments

This project was funded by National Institute of Mental Health Grant 5 R01 MH60838 to Nancy Eisenberg and Tracy L. Spinrad. We would like to thank the children, parents, and teachers involved in the current study. In addition, we appreciate our undergraduate research assistants for their contributions.

References

Amstadter AB, Daughters SB, MacPherson L, Reynolds EK, Danielson CK, Wang F, Lejuez CW. Genetic associations with performance on a behavioral measure of distress intolerance. Journal of Psychiatric Research. 2012; 46:87–94. doi:10.1016/j.jpsychires.2011.09.017. [PubMed: 22024485]

Arend R, Gove FL, Sroufe LA. Continuity of individual adaptation from infancy to kindergarten: A predictive study of ego-resiliency and curiosity in preschoolers. Child Development. 1979; 50(4): 950–959. doi: http://dx.doi.org/10.2307/1129319. [PubMed: 535446]

Ali FR, Vasiliou SA, Haddley K, Paredes UM, Roberts JC, Miyajima F, Quinn JP. Combinatorial interaction between two human serotonin transporter gene variable number tandem repeats and their regulation by CTCF. Journal of Neurochemistry. 2010; 112:296–306. doi:10.1111/j.1471-4159.2009.06453.x. [PubMed: 19860858]

Asendorpf JB, van Aken MA. Resilient, overcontrolled, and undercon troled personality prototypes in childhood: Replicability, predictive power, and the trait-type issue. Journal of Personality and Social Psychology. 1999; 77(4):815–832. doi:10.1037/0022- 3514.77.4.815. [PubMed: 10531673]

Belsky J, Beaver KM. Cumulative-genetic plasticity, parenting and adolescent self-control/regulation. Journal of Child Psychology and Psychiatry. 2011; 52:619–626. doi: 10.1111/j.1469-7610.2010.02327.x. [PubMed: 21039487]

Belsky J, Pluess M. Beyond diathesis stress: Differential susceptibility to environmental influences. Psychological Bulletin. 2009; 135:885–908. doi:10.1037/a0017376. [PubMed: 19883141]

Block J, Block JH. Venturing a 30-year longitudinal study. American Psychologist. 2006; 61:315–327. doi:10.1037/0003-066X.61.4.315. [PubMed: 16719676]

Block, JH.; Block, J. The role of ego-control and ego-resiliency in the organization of behavior. In: Collins, WA., editor. Minnesota Symposia on Child Psychology. Vol. Vol. 13. Erlbaum; Hillsdale, NJ: 1980. p. 39-101.

Brody GH, Beach SRH, Chen Y, Obasi E, Philibert RA, Kogan SM, Simons RL. Perceived discrimination, serotonin transporter linked polymorphic region status, and the development of conduct problems. Development and Psychopathology. 2011; 23:617–627. doi:10.1017/S0954579411000046. [PubMed: 23786700]

Caspi A, Hariri AR, Holmes A, Uher R, Moffitt TE. Genetic sensitivity to the environment: The case of the serotonin transported gene and its implications for studying complex diseases and traits. American Journal of Psychiatry. 2010; 167:509–527. doi:10.1176/ appi.ajp.2010.09101452. [PubMed: 20231323]

Chen MC, Joormann J, Hallmayer J, Gotlib IH. Serotonin transporter polymorphism predicts waking cortisol in young girls. Psychoneuroendocrinology. 2009; 34:681–686. doi:10.1016/j.psyneuen.2008.11.006. [PubMed: 19128885]

Chuang SS, Lamb ME, Hwang CP. Personality development from childhood to adolescence: A longitudinal study of ego-control and ego-resiliency in Sweden. International Journal of Behavioral Development. 2006; 30:338–343. doi:10.1177/0165025406072795.

Claw KG, Tito RY, Stone AC, Verrelli BC. Haplotype structure and divergence at human and chimpanzee serotonin transporter and receptor genes: Implications for behavioral disorder association analyses. Molecular Biology and Evolution. 2010; 27:1518–1529. doi:10.1093/molbev/msq030. [PubMed: 20118193]

Cumberland-Li A, Eisenberg N, Reiser M. Relations of young children's agreeableness and resiliency to effortful control and impulsivity. Social Development. 2004; 13:193–212. doi:10.1111/j.1467-9507.2004.000263.x.

Deater-Deckard K, Lansford JE, Malone PS, Alampay LP, Sorbring E, Bacchini D, Al-Hassan S. The association between parental warmth and control in thirteen cultural groups. Journal of Family Psychology. 2011; 25(5):790–794. doi:10.1037/a0025120. [PubMed: 21875202]

Derryberry D, Rothbart MK. Reactive and effortful processes in the organization of temperament. Development and Psychopathology. 1997; 9(4):633–652. doi: http://dx.doi.org/10.1017/S0954579497001375. [PubMed: 9448999]

Eisenberg N, Fabes RA, Guthrie IK, Murphy BC, Maszk P, Holgren R, Suh K. The relations of regulation and emotionality to problem behavior in elementary school children. Development and Psychopathology. 1996; 8(1):141–162. doi .10.1017/S095457940000701x.

Eisenberg N, Spinrad TL, Fabes RA, Reiser M, Cumberland A, Shepard SA, Thompson M. The relations of effortful control and impulsivity to children's resiliency and adjustment. Child Development. 2004; 75(1):25–46. doi: http://dx.doi.org/10.1111/j.1467-8624.2004.00652.x. [PubMed: 15015673]

Eisenberg N, Valiente C, Fabes RA, Smith CL, Reiser M, Shepard SA. The relations of effortful control and ego control to children's resiliency and social functioning. Developmental Psychology. 2003; 39:761–776. doi:10.1037/0012-1649.39.4.761. [PubMed: 12859128]

Ellis AJ, Beevers CG, Hixon JG, McGeary JE. Serotonin transporter promoter region (5 HTTLPR) polymorphism predicts resting respiratory sinus arrhythmia. Psychophysiology. 2011; 48(7):923–926. doi: 10.1111/j.1469-8986.2010.01154.x. [PubMed: 21631517]

Ellis BJ, Boyce WT, Belsky J, Bakermans-Kranenburg MJ, van IJzendoorn MH. Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. Development and Psychopathology. 2011; 23:7–28. doi:10.1017/S0954579410000611. [PubMed: 21262036]

Fiskerstrand CE, Lovejoy EA, Quinn JP. An intronic polymorphic domain often associated with susceptibility to affective disorders has allele dependent differential enhancer activity in embryonic stem cells. FEBS Letters. 1999; 458:171–174. [PubMed: 10481059]

Gilissen R, Bakermans-Kranenburg M, IJzendoorn M. H. v. Linting M. Electrodermal reactivity during the trier social stress test for children: Interaction between the serotonin transporter polymorphism and children's attachment representation. Developmental Psychobiology. 2008; 50:615–625. doi:10.1002/dev.20314. [PubMed: 18683185]

Gotlib IH, Joormann J, Minor KL, Hallmayer J. HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. Biological Psychiatry. 2008; 63:847–851. doi:10.1016/j.biopsych.2007.10.008. [PubMed: 18005940]

Greenberg BD, Tolliver TJ, Huang SJ, Li Q, Bengel D, Murphy DL. Genetic variation in the serotonin transporter promoter region affects serotonin uptake in human blood platelets. American Journal of Medical Genetics. 1999; 88:83–87. doi:10.1002/ (SICI)1096-8628(19990205)88:1_83::AID-AJMG15_3.0.CO;2-0. [PubMed: 10050973]

Hankin BL, Jenness J, Abela JRZ, Smolen A. Interaction of 5-HTTLPR and idiographic stressors predicts prospective depressive symptoms specifically among youth in a multiwave design. Journal of Clinical Child and Adolescent Psychology. 2011; 40:572–585. doi: 10.1080/15374416.2011.581613. [PubMed: 21722029]

Hanna GL, Himle JA, Curtis GC, Koram DQ, VanderWeele J, Leventhal BL, Cook EH. Serotonin transporter and seasonal variation in blood serotonin in families with obsessive-compulsive disorder. Neuropsychopharmacology. 1998; 18:102–111. doi:10.1016/ S0893-133X(97)00097-3. [PubMed: 9430134]

Heinz A, Jones DW, Mazzanti C, Goldman D, Ragan P, Hommer D, Weinberger DR. A relationship between serotonin transporter genotype and in vivo protein expression and alcohol neurotoxicity. Biological Psychiatry. 2000; 47:643–649. doi:10.1016/S0006- 3223(99)00171-7. [PubMed: 10745057]

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; 10:696–702. doi:10.1037/a0019527. [PubMed: 21038952]

Hedrick, PW. Genetics of populations. 4th ed. Jones and Bartlett; Sudbury, MA: 2010.

Hofer C, Eisenberg N, Reiser M. The role of socialization, effortful control, and ego-resiliency in French adolescents' social functioning. Journal of Research on Adolescence. 2010; 20:555–582. doi:10.1111/j.1532-7795.2010.00650.x. [PubMed: 21228912]

Ivorra JL, Sanjuan J, Jover M, Carot JM, de Frutos R, Molto MD. Gene–environment interaction of child temperament. Journal of Developmental and Behavioral Pediatrics. 2010; 31(7):545–554. [PubMed: 20729761]

Kaufman J, Yang B, Douglas-Palumberi H, Grasso D, Lipschitz D, Houshyar S, Gelernter J. Brain-derived neurotrophic factor-5-HHTLPR gene interactions and environmental modifiers of depression in children. Biological Psychiatry. 2006; 59:673–680. doi:10.1016/j.biopsych. 2005.10.026. [PubMed: 16458264]

Kim J, Cicchetti D, Rogosch FA, Manly JT. Child maltreatment and trajectories of personality and behavioral functioning: Implications for the development of personality disorder. Development and Psychopathology. 2009; 21(3):889–912. doi:10.1017/S0954579409000480. [PubMed: 19583889]

Kim-Cohen J, Gold A. Measured gene-environment interactions and mechanisms promoting resilient development. Current Directions in Psychological Science. 2009; 18:138–142. doi:10.1111/j.1467-8721.2009.01624.x.

Kochanska G, Kim S, Barry RA, Philibert RA. Children's genotypes interact with maternal responsive care in predicting children's competence: Diathesis–stress or differential susceptibility? Development and Psychopathology. 2011; 23:605–616. doi:10.1017/S0954579411000071. [PubMed: 23786699]

Kremen AM, Block J. The roots of ego-control in young adulthood: Links with parenting in early childhood. Journal of Personality and Social Psychology. 1998; 75(4):1062–1075. doi:10.1037/0022-3514.75.4.1062. [PubMed: 9825535]

Lakatos K, Nemoda Z, Birkas E, Ronai A, Kovacs E, Ney K, Toth I, Sasvari-Szekely M, Gervai J. Association of D4 dopamine receptor gene and serotonin transporter promoter polymorphisms with infants' response to novelty. Molecular Psychiatry. 2003; 8(1):90–97. PMID:1255691. [PubMed: 12556912]

Lemery-Chalfant, K. Genes and environments: How they work together to promote resilience. In: Reich, JW.; Zautra, AJ.; Hall, JS., editors. Handbook of adult resilience. Guilford Press; New York, NY, US: 2010.

Martel MM, Nigg JT, Wong Maria M. Fitzgerald HE, Jester JM, Puttler LI. Childhood and adolescent resiliency, regulation, and executive functioning in relation to adolescent problems and competence in a high-risk sample. Development and Psychopathology. 2007; 19:541–563. doi:10.1017/S0954579407070265. [PubMed: 17459183]

Martin CG, Fisher PA, Kim HK. Risk for maternal harsh parenting in high-risk families from birth to age three: Does ethnicity matter? Prevention Science. 2012; 13(1):64–74. doi:10.1007/s11121-011-0247-0. [PubMed: 21935658]

Mitchell C, Notterman D, Brooks-Gunn J, Hobcraft J, Garfinkel I, Jaeger K, McLanahan S. Role of mother's genes and environment in postpartum depression. PNAS Proceedings of the National Academy of Sciences of the United States of America. 2011; 108(20):8189–8193. doi:10.1073/pnas.1014129108.

Mueller A, Strahler J, Armbruster D, Lesch K, Brocke B, Kirschbaum C. Genetic contributions to acute autonomic stress responsiveness in children. International Journal of Psychophysiology. 2011 doi:10.1016/j.ijpsycho.2011.11.007.

Nakaya M, Oshio A, Kaneko H. Correlations for adolescent resilience scale with big five personality traits. Psychological Reports. 2006; 98(3):927–930. doi:10.2466/pr0.98.3.927-930. [PubMed: 16933700]

Ogilvie AD, Battersby S, Bubb VJ, Fink G, Harmar AJ, Goodwim GM, Smith C. Polymorphism in serotonin transporter gene associated with susceptibility to major depression. Lancet. 1996; 347:731–733. doi.org/10.1016/S0140-6736(96)90079-3. [PubMed: 8602004]

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. doi: 10.1007/s00702-008-0171-z. [PubMed: 19137235]

Perea CS, Paternina AC, Gomez Y, Lattig MC. Negative affectivity moderated by BDNF and stress response. Journal of Affective Disorders. 2012; 136(3):767–774. doi:10.1016/j.jad.2011.09.043. [PubMed: 22044630]

Pluess M, Velders FP, Belsky J, van IJzendoorn MH, Bakermans-Kranenburg M, Jaddoe VWV, Tiemeier H. Serotonin transporter polymorphism moderates effects of prenatal maternal anxiety on infant negative emotionality. Biological Psychiatry. 2011; 69:520–525. doi:10.1016/j.biopsych.2010.10.006. [PubMed: 21126730]

Power TG. Stress and coping in childhood: The parents' role. Parenting: Science and Practice. 2004; 4(4):271–317. doi:10.1207/s15327922par0404_1.

Pritchard JK, Stephens M, Donnelly P. Inference of population structure using multilocus genotype data. Genetics. 2000; 155:945–959. Retrieved from http://www.genetics.org/content/155/2/945.full.pdf.html. [PubMed: 10835412]

Risch N, Herrell R, Lehner T, Liang K, Eaves L, Hoh J, Griem A, Kovacs M, et al. Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. Journal of the American Medical Association. 2009; 301(23):2462–2471. doi: 10.1001/jama.2009.878. PMC 2938776. PMID 19531786. [PubMed: 19531786]

Robins RW, John OP, Caspi A, Moffitt TE, Stouthamer-Loeber M. Resilient, overcontrolled, and undercontrolled boys: Three replicable personality types. Journal of Personality and Social Psychology. 1996; 70:157–171. [PubMed: 8558407]

Sambrook, J.; Russell, DW. Molecular cloning: A laboratory manual. Cold Spring Harbor Laboratory Press; Cold Spring Harbor, NY: 2001. Retrieved from http://www.gate2biotech.com/molecular-cloning-alaboratory-manual/

Shanahan MJ, Hofer SM. Social context in gene--environment interactions: Retrospect and prospect. The Journals of Gerontology: Series B: Psychological Sciences & Social Sciences. 2005; 60B(1): 65–76. doi:10.1093/geronb/60.Special_Issue_1.65.

Shonk SM, Cicchetti D. Maltreatment, competency deficits, and risk for academic and behavioral maladjustment. Developmental Psychology. 2001; 37:3–17. doi:10.1037/0012- 1649.37.1.3. [PubMed: 11206431]

Smeekens S, Riksen-Walraven J, van Bakel H. The predictive value of different infant attachment measures for socioemotional development at age 5 years. Infant Mental Health Journal. 2009; 30:366–383. doi:10.1002/imhj.20219.

Sonuga-Barke E, Kumsta R, Schlotz W, Lasky-Su J, Marco R, Miranda A, Faraone SV. A functional variant of the serotonin transporter gene (SLC6A4) moderates impulsive choice in attention-deficit/hyperactivity disorder boys and siblings. Biological Psychiatry. 2011; 70(3):230–236. doi: 10.1016/j.biopsych.2011.01.040. [PubMed: 21497794]

Stein MB, Campbell-Sills L, Gelernter J. Genetic variation in 5HTTLPR is associated with emotional resilience. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics. 2009; 150B: 900–906. doi: 10.1002/ajmg.b.30916.

Stephens M, Smith NJ, Donnelly P. A new statistical method for haplotype reconstruction from population data. American Journal of Human Genetics. 2001; 68:978–989. doi:10.1086/319501. [PubMed: 11254454]

Sulik MJ, Eisenberg N, Lemery-Chalfant K, Spinrad TL, Silva KM, Eggum ND, Verrelli BC. Interactions between serotonin transporter gene haplotypes and quality of mothers parenting predict the development of children's noncompliance. Developmental Psychology. 2012; 48(3): 740–754. doi:10.1037/a0025938. [PubMed: 22059451]

Taylor ZE, Eisenberg N, Spinrad TL, Widaman KF. Longitudinal relations of intrusive parenting and effortful control to ego-resiliency during early childhood. Child Development. in press.

Table 1

Means and Standard Deviations of Parenting and Mother-Reports of Children's Ego-Resiliency

	Parenting		Ego-Resiliency											
	18 months		18 months		30 months		42 months		54 months		72 months		84 months	
Variable	***N***	***M (SD)***	***N***	***M (SD)***	***N***	***M (SD)***	***N***	***M (SD)***	***N***	***M(SD)***	***N***	***M (SD)***	***N***	***M (SD)***
Total	146	.05 (.64)	143	6.88 (.78)	148	6.94(1.03)	150	7.01(.89)	145	6.86(.94)	146	6.81(.93)	130	6.92(1.04)
SLC6A4: LPR														
LL	52	−.06(.72)	49	6.83(.81)	54	7.00(.99)	54	7.06(.92)	50	6.95(.88)	50	6.84(1.02)	44	7.08(1.01)
SS/SL	94	.12(.58)	94	6.91(.77)	94	6.90(1.05)	96	6.97(.87)	95	6.81(.98)	96	6.80(.89)	86	6.84(1.06)
SLC6A4: STin2														
10-10	15	.02(.60)	14	6.81(.97)	14	6.78(1.03)	14	6.90(.86)	15	6.56(.98)	15	6.49(.85)	13	6.77(.86)
10-12	79	.03(.71)	78	6.82(.78)	81	6.95(.99)	82	6.96(.90)	78	6.85(.95)	80	6.81(1.01)	72	6.90(1.13)
12-12	52	.10(.52)	51	6.99(.73)	53	6.96(1.09)	54	7.11(.88)	52	6.96(.93)	51	6.91(.81)	45	7.00(.96)
SLC6A4: Haplotype														
S10	15	.06 (.73)	15	6.59 (.69)	14	6.34(1.05)	14	6.62 (.50)	14	6.02 (.84)	14	6.68 (.83)	14	6.46 (.76)
S12	79	.13 (.55)	79	6.97 (.77)	80	7.00(1.02)	82	7.04 (.91)	81	6.95 (.93)	82	6.82 (.90)	72	6.92(1.09)
L10/L12	52	-.06(.72)	49	6.83 (.81)	54	7.00 (.99)	54	7.06 (.92)	50	6.95 (.88)	50	6.84(1.02)	44	7.08(1.01)

Table 2

Correlations among variables, $N = 153$

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. ER 18 mo																		
2. ER 30 mo	.41**																	
3. ER 42 mo	.40**	.53**																
4. ER 54 mo	.42**	.47**	.56**															
5. ER 72 mo	.35**	.46**	.49**	.58**														
6. ER 84 mo	.23**	.49**	.56**	.58**	.65**													
7. Ob. Parenting	.27**	.08 .14†	.16†	.13	.02													
8. LPR (S vs. L)	−.05	.05	.05	.07	.02	.11	−.13											
9. STin2 (10-12)	−.09	.01	−.06	−.01	−.00	−.02	−.04	.16†										
10. STin2 (12-12)	.11	.02	.09	.08	.08	.06	.06	−.33**	−.82**									
11. STin2 (10-10)	−.03	−.05	−.04	−.11	−.12	−.05	−.02	.26**	−.36	−.25**								
12. S12 Hap	.12	.06	.04	.11	.01	−.01	.12	−.82**	−.23	.46**	−.36**							
13. L10-L12 Hap	−.05	.05	.05	.07	.02	.11	−.13	1.00	.16†	−.33**	.26**	−.82**						
14. S10 Hap	−.13	−.19*	−.14†	−.29**	−.05	−.16†	.01	−.24**	.13	−.25**	.19*	−.36**	−.24**					
15. Income	.04	.11	.07	.04	.11	.09	.38**	−.15†	.08	.02	−.17†	.10	−.15†	08				
16. Mo. Ed.	.10	.20*	.06	.01	.12	−.02	.48**	−.10	.05	−.06	.02	−.01	−.10	18*	.55**			
17.	Fa. Ed.	.02	.08	.08	.07	.11	.10	.35**	−.14	.11	.02	−.21*	.20*	−.14	−.12	.48**	.50**	
18. Child Sex	.08	−.01	.00	−.03	.07	.13	.01	−.03	−.10	.07	.06	.02	−.03	.02	−.17†	−.03	−.02	
M	6.88	6.94	7.01	6.86	6.81	6.92	.05	.35	.54	.36	.10	.55	.35	.10	4.27	4.41	4.29	.44
SD	(.78)	(1.03)	(.89)	(.94)	(.93)	(1.04)	(.64)	(.48)	(.50)	(.48)	(.30)	(.50)	(.48)	(.30)	(1.70)	(1.03)	(1.21)	(.50)

*Note: $p < .05$

**$p < .01$

†$p < .10$; Sex -1 = male, 1 = female; LPR short =0, long = 1

Table 3

Parenting and SLC6A4 Haplotypes as Predictors of Mother Reports of Ego-Resiliency

	Ego-Resiliency					
	Main Effects			Interactions		
Fixed Effects	***b***	***t***	***p***	***b***	***t***	***p***
Intercept [a] (18 months)	6.93			6.99		
S10 vs. S12	−0.49	−2.52	**	−.53	−2.78	**
S10 vs. L10-L12	−0.53	−2.82	**	−.50	−2.71	**
L10–L12 vs. S12	0.04	0.32		.03	.27	
Parenting	0.22	2.57	**	.30	2.37	*
Parenting × S10 vs. S12				−0.29	−1.07	
Parenting × S10 vs. L10-L12				−0.19	−0.68	
Parenting × L10-L12 vs. S12				0.10	0.55	
Linear slope [b]	−0.01	−0.81		0.01	0.41	
S10 vs. S12	−0.04	−1.13		−0.04	−1.11	
S10 vs. L10-L12	−0.01	−0.16		−0.01	−0.17	
L10–L12 vs. S12	0.03	0.55		0.03	0.54	
Parenting	−0.02	−1.00		−0.02	−0.97	
Parenting × S10 vs. S12				−0.10	−0.56	
Parenting × S10 vs. L10-L12				−0.29	−1.07	
Parenting × L10-L12 vs. S12				0.18	0.68	
Pseudo r^2 (%)	4.3%			>4.4%		
−2LL (deviance)	1932.92			1934.08		

[a] Note: This coefficient refers to the intercept for the S10 group at the mean level of parenting. The difference between the intercept for the S10 group and the other genetic groups at the mean level of parenting is represented by the genetic main effects

[b] This coefficient refers to the linear effect with time centered at 18 months. The genetic main effects on the slope represent the difference between this coefficient for the S10 group and the other genetic groups. Child sex was not associated with ego-resiliency as a main effect or as an interaction with genes. N =153.