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Authors

Noroña, Amanda N
Tung, Irene
Lee, Steve S
et al.

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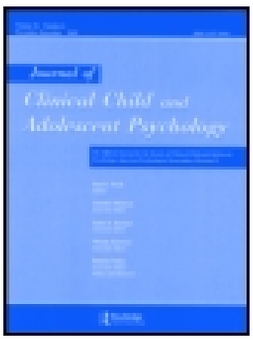

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Developmental Patterns of Child Emotion Dysregulation as Predicted by Serotonin Transporter Genotype and Parenting

Amanda N. Noroña, Irene Tung, and Steve S. Lee
Department of Psychology, University of California, Los Angeles

Jan Blacher
School of Education, University of California, Riverside

Keith A. Crnic
Department of Psychology, Arizona State University

Bruce L. Baker
Department of Psychology, University of California, Los Angeles

Individual differences in emotion regulation are central to social, academic, occupational, and psychological development, and emotion dysregulation (ED) in childhood is a risk factor for numerous developmental outcomes. The present study aimed to (a) describe the developmental trajectory of ED across early childhood (3–6 years) and (b) examine its sensitivity to youth serotonin transporter genotype, positive and negative parenting behaviors, and their interaction. Participants were 99 families in the Collaborative Family Study, a longitudinal study of children with or without developmental delays. Child ED and early parenting were coded from parent–child interactions.

To examine serotonin transporter genotype as a moderator between parenting and child emotion dysregulation (ED), children with the homozygous short (SS) genotype were compared to children with the homozygous long (LL) or heterozygous (SL) genotype. We used latent growth curve modeling (LGCM) to model yearly change in ED from child age 3 to 6 years. LGCM revealed that ED decreased overall across early childhood. In addition, we observed separate Genotype × Positive and Genotype × Negative parenting behavior interactions in predictions of ED growth curves. Children with the SL/LL genotype had ED trajectories that were minimally related to positive and negative parenting behavior, whereas ED decreased more precipitously among children with the SS genotype when exposed to low negative parenting or high positive parenting. These findings provide evidence for Gene × Environment interactions (G×Es) in the development of ED in a manner that is conceptually consistent with vantage sensitivity, and they improve inferences afforded by prospective designs.

Individual differences in emotion regulation reliably predict numerous long-term psychological, emotional, and physical outcomes. Emotion regulation, defined as "behaviors, skills, and strategies, whether conscious or unconscious, automatic or effortful, that serve to modulate, inhibit, and enhance emotional experiences and expressions," (Calkins & Hill, 2007, p. 229) is a broad construct that can range from very poor regulation (i.e., emotion dysregulation [ED]) to very

Correspondence should be addressed to Amanda N. Noroña, M.A., Department of Psychology, University of California, Los Angeles, 1285 Franz Hall, Box 951563, Los Angeles, CA 90095-1563. E-mail: amandanorona@ucla.edu

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high regulation. ED, defined by emotional expression that is inappropriate to the context in terms of intensity and duration, and interferes with functioning (Hoffman, Crnic, & Baker, 2006), is central to heuristic models of psychopathology, including major depressive disorder, bipolar disorder, anxiety disorders, eating disorders, and personality disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Cole & Deater-Deckard, 2009). Of note, although not precisely opposite constructs, emotion regulation has been found to increase as ED and related constructs decrease (Blandon, Calkins, Keane, & O'Brien, 2008).

The majority of research on ED and psychopathology, however, is cross-sectional and thus compromises directional inferences. However, in one short-term (7 months) longitudinal study, McLaughlin, Hatzenbuehler, Mennin, and Nolen-Hoeksema (2011) found that ED predicted increased anxiety, aggression, and eating pathology, even with control of baseline psychopathology. Thus, as ED has been identified as a key cross-diagnostic factor for psychopathology, understanding the development of ED is critical for understanding the etiology, maintenance, and treatment of psychological disorders. The current study focuses on predictors of change in ED during early childhood, toward a better understanding of the processes that underlie the development of this crucial risk factor.

EMOTION REGULATION DEVELOPMENT

Given associations with several domains of long-term functioning, the independent regulation of emotional reactions is considered a major developmental milestone of childhood (Cicchetti, Ganiban, & Barnett, 1991; Kopp, 1982). Surprisingly then, although many cross-sectional studies have examined emotion regulation in children, relatively few studies have studied the development of emotion regulation longitudinally. Understanding longitudinal growth in emotion regulation is critical, given rapid changes in socioemotional, linguistic, cognitive, and biological development in early childhood (Blandon et al., 2008; Fox, 1994). Prior prospective examinations of developmental change in emotion regulation and ED-related constructs suggest that, in early childhood, emotion regulation behaviors increase (Blandon et al., 2008), and ED behaviors decrease (Fabes, Hanish, Martin, & Eisenberg, 2002; Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999). Of note, only one of these studies examined change in observed behavior and focused on change over only a 3-month period (Fabes et al., 2002). These findings provide support for theoretical assertions and cross-sectional findings in this developmental stage, including findings that preschoolers demonstrate emerging abilities to use problem-solving skills to directly address their source of distress, positively reframe upsetting situations, and flexibly employ different regulation strategies (Kalpidou, Power, Cherry, & Gottfried, 2004; Stansbury & Sigman, 2000). Together, these studies highlight the importance of further studying behaviorally observed regulation and dysregulation in early childhood and suggest that early childhood may be an especially formative period for emotional development.

In studies examining predictors of emotion regulation development, two broad factors have been found to be central to the development and maintenance of emotion regulation: influences external to the individual, and influences internal to the individual (for the seminal review, see Calkins, 1994). Extensive research has been done on environmental (i.e., external) influences on emotion regulation, with the vast majority focusing on the family system. Positive parenting behaviors, such as sensitivity (Feldman, 2007; Fogel, 1993; Leerkes, Blankson, & O'Brien, 2009), scaffolding (Bernier, Carlson, & Whipple, 2010; Gulsrud, Jahromi, & Kasari, 2009; Hoffman et al., 2006; Lengua, Honorado, & Bush, 2007), and expression of positive affect (Cumberland-Li, Eisenberg, Champion, Gershoff, & Fabes, 2003; Eisenberg et al., 2001) influence emergent youth emotion regulation, such that the presence of positive parenting behaviors facilitates emotion regulation development, whereas the absence hinders it (NICHD Early Child Care Research Network, 2004).

Conversely, ED in childhood has been predicted by negative parenting behaviors, such as intrusiveness (Cabrera, Shannon, & Tamis-LeMonda, 2007; Graziano, Keane, & Calkins, 2010; Stevenson & Crnic, 2013) and parental expression of negative emotions (Morris, Silk, Steinberg, Myers, & Robinson, 2007). The majority of previous research has focused on either positive *or* negative parenting behavior. The present study examined and compared the contributions of broad measures of positive and negative parenting to trajectories of ED across childhood.

In addition to research on the environmental contributors to ED, characteristics within the individual have been studied. Researchers have theorized and provided empirical evidence for the effects of neuroregulatory reactivity (Dennis & Hajcak, 2009; Fox, 1994; Stansbury & Gunnar, 1994), behavioral traits (e.g., temperament; Calkins, 1994; Gunnar, Porter, Wolf, Rigatuso, & Larson, 1995; Stifter & Braungart, 1995), and cognitive ability (Crnic, Hoffman, Gaze, & Edelbrock, 2004; Norona & Baker, 2016) on ED. In a different line of study, researchers have examined the influence of genes on ED (see Hariri & Holmes, 2006, for a review).

GENETICS AND ED

Among genetic variants linked to ED, the promoter polymorphism in the serotonin transporter gene is the most common variant studied. As serotonin is a key neurotransmitter in mood regulating systems, such as the hypothalamic-pituitary-adrenal axis, it is not surprising that the serotonin transporter genotype is associated with mood, attention, and psychopathology (Auerbach,

Faroy, Ebstein, Kahana, & Levine, 2001; Champoux et al., 2002; Lucki, 1998; Soubrie, 1986; Van Goozen, Fairchild, Snoek, & Harold, 2007). 5-HTTLPR has two allelic forms, a short (S) variant and a long (L) variant. The S variant has been associated with reduced serotonin transporter transcription, lower serotonin transporter protein levels, and diminished serotonin reuptake (Lesch et al., 1996). Further, the S allele has been identified as a potential genetic "risk" factor, as having the SS (short–short) or SL (short–long) genotype has been linked with outcomes closely related to ED, such as higher levels of anxiety (Hariri et al., 2005; Lesch et al., 1996), vulnerability to life stress (Caspi et al., 2003; Champoux et al., 2002), and amygdala hyperreactivity (Hariri et al., 2002; Heinz et al., 2005). Thus, the S allele may represent a risk factor for ED, with individuals with the SS genotype at highest risk (Kendler, Kuhn, Vittum, Prescott, & Riley, 2005). Some researchers, however, have failed to replicate these genetic "risk" effects (e.g., Gillespie, Whitfield, Williams, Heath, & Martin, 2005; Surtees et al., 2006; Willis-Owen et al., 2005). Researchers have suggested that the inconsistency in findings is due to complex relationships between genes and phenotypes, the difficulty of operationalizing complex phenotypes such as ED, and/or small effect sizes (Canli & Lesch, 2007; Hariri & Holmes, 2006).

One area of research that has provided some clarity in regards to inconsistent findings for genetic "risk" effects examines G×E. This line of research directly answers questions regarding the complexity between genetic predisposition and phenotypic expression. It asserts that inconsistency in risk findings is due to interplay between genotypes and the amount of adversity or nurturance in the individual's environment; this has yielded meaningful findings in investigations of psychopathological outcomes associated with ED (e.g., depression, externalizing behaviors). Thus, investigating the development of ED using the G×E framework is a necessary follow-up.

G×E AND ED

The effects of early caregiving environments on ED trajectories may be moderated by serotonin transporter genotype through G×E. Two studies have found support for a serotonin transporter Genotype × Attachment interaction in predicting ED, one in preschool children (Kochanska, Philibert, & Barry, 2009) and the other in adolescents (Zimmermann, Mohr, & Spangler, 2009), with secure attachment conceptualized as an indicator of early positive and sensitive parenting. Kochanska et al. (2009) found that preschool children with insecure attachment and an S allele developed increased ED, whereas securely attached children with an S allele exhibited ED similar to LL genotype children. These findings suggest that serotonin transporter genotype may increase a child's vulnerability to insecure attachment in the preschool years. Results from the adolescent study tell a somewhat different story. Similar to the preschool findings, S allele carriers with insecure attachment displayed less agreeable and more hostile behaviors with parents, compared to S allele carriers with secure attachment. When compared to adolescents with the LL genotype, however, the S allele carriers appeared to benefit more from secure attachment (i.e., displayed more agreeable behavior) and exhibit more impairment from insecure attachment (i.e., displayed more hostile behavior) (Zimmermann et al., 2009).

These two studies highlight an area of inconsistency in G×E research in developmental psychopathology. Although convergent evidence seems to support that individuals differ in genetic plasticity to environmental influences, researchers have yet to identify the mechanism of this plasticity. Some studies' findings (e.g., Kochanska et al., 2009) support the *diathesis-stress* (a.k.a. *dual risk*) model, which asserts that certain characteristics (e.g., genotype, temperament) predispose certain individuals to be more vulnerable to the effects of adverse environments (Monroe & Simons, 1991; Sameroff, 1983; Zuckerman, 1999). Other studies, however (e.g., Zimmermann et al., 2009), provide support for the *differential susceptibility* hypothesis, which states that these individuals who under the diathesis-stress framework are more vulnerable to negative environments are also more sensitive to the positive effects of enriched environments (Belsky, 1997; Belsky & Pluess, 2009; Boyce & Ellis, 2005). *Vantage sensitivity* is another proposed variation of G×E, by which certain individuals are disproportionately likely to be solely positively affected by positive contextual conditions. Emotion regulation and/or dysregulation have not yet been studied in the context of vantage sensitivity, though 5-HTTLPR has been identified as a genetic marker of this type of plasticity for related constructs, such as positive emotionality (Hankin et al., 2011) and anxiety disorders (Eley et al., 2012). Although a literature base is still accumulating for G×E and ED, studies that have examined the effects of the S allele within developmental psychopathology and temperament have consistently demonstrated that, as compared to individuals with LL (and oftentimes SL) genotype, individuals with the SS genotype evidence greater plasticity. Further research will elucidate the nature of the plasticity, whether in conferring dual risk, differential susceptibility, or vantage sensitivity.

The present study extends the literature base by exploratory analysis of G×E effects on the trajectory of ED. To our knowledge, no study to date has tested G×E in predicting ED trajectories. It is critical to address this empirical gap, given that ED processes are fundamentally dynamic, particularly in early childhood, and that genetic plasticity to the environment is likely effective across the lifespan, as opposed to a single time point (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011). Further,

identifying G×E underlying individual differences in ED trajectories has the potential to inform identification, prevention, and intervention efforts in childhood psychopathology. Although researchers have yet to specifically examine G×E effects on emotion regulation or dysregulation trajectories, evidence has been presented on G×E effects on trajectories of constructs theoretically tied to ED, such as negative emotionality (Lipscomb et al., 2012) and externalizing behavior problems (Brett et al., 2015; Trucco, Villafuerte, Heitzeg, Burmeister, & Zucker, 2016; Tung & Lee, 2016).

THE CURRENT STUDY

The current study investigated the development of ED, with two primary aims: (a) to identify the developmental trajectory of ED across early childhood, from 3 to 6 years of age, and (b) to examine how serotonin transporter genotype and positive and negative parenting behaviors independently and interactively affect individual differences in trajectories of ED.

We hypothesized that ED would decrease across early childhood. Further, we hypothesized that genotype and parenting would have independent effects on ED and that serotonin transporter genotype would moderate the association between parenting and ED. In terms of age 3 (intercept) ED, we expected effects of positive and negative parenting but not of genotype or G×E, such that positive parenting would be associated with lower initial ED, and negative parenting with higher ED. For yearly change in ED from age 3 to 6 (slope), we predicted a continued main effect of parenting and that genotype would moderate its effects, such that children with the SS genotype, as compared to children with the SL/LL genotype, would demonstrate more sensitivity (i.e., faster decline in ED for high positive and low negative parenting and increase in ED for low positive and high negative parenting).

This study addresses critical limitations in the literature. First, the majority of studies examine the deleterious effects of the negative early environment. We have taken a holistic approach to the environment, using observationally obtained measures of positive *and* negative parenting behaviors. This approach has the potential to provide important nuance to the current literature on G×E processes. Second, the field lacks an empirical examination of longitudinal effects of G×E in the context of ED. It is critical to take this developmental approach, considering the dynamic nature of ED across childhood. Third, most G×E investigations have focused on complex clinical phenotypes such as depression, antisocial behavior, and attention deficit/hyperactivity disorder. These findings may be confounded because these disorders share underlying traits, including ED. Thus we directly targeted intermediate phenotypes, herein ED, that may be closer to the G×E process.

METHOD

Participants

Participants were 99 families enrolled in the Collaborative Family Study, a longitudinal study of children with and without developmental delays (DD) and their families, conducted by University of California, Los Angeles (UCLA), University of California, Riverside (UCR), and the Pennsylvania State University (PSU). The samples, drawn from Southern California and Central Pennsylvania, were followed from child age 3 to 15 years. Informed consent was obtained from participating parents and assent from the children. The present sample comprised all families for whom data were available on at least one of the primary measures (i.e., ED, positive parenting, negative parenting) at child ages 3–6, as well as genetic data, which was collected at age 13. The initial sample comprised 238 families. Our combination of measure criteria reduced the sample to 99 families, who had consistent participation in the study for a decade. In addition to families dropping out of the study over time, other reasons for missing data included youth declining to continue with participation, video/audio recording malfunctions, and poor saliva sample for genotyping. The present sample did not differ from families who were no longer available on demographic factors (i.e., ethnicity, marital status, socioeconomic status, parental education, parent age) or any measure of interest (i.e., genotype or sex distribution, IQ, positive parenting, negative parenting, ED at any age). Among the 99 participants in the current sample, 83 participants (83.8%) had complete data, 15 participants (15.2%) had missing data for one key variables (with 13 of those missing age 6 ED), and one participant (1%) had missing data for two key variables.

Families were recruited at child age 3 years. Families of children with DD came primarily from agencies that provide diagnostic and intervention services for this population. Children with autism were excluded from the study. Families of children with typical development were recruited through local preschools and day care programs. Selection criteria were that the child scored in the range of normal cognitive development and had not been born prematurely or had any known developmental disability.

Table 1 shows the sample demographic characteristics at child age 3. Among the children, 43.4% were female and about half were Caucasian, non-Hispanic (56.6%), followed by Hispanic (18.2%), “Other” (15.2%), African American (8.1%), and Asian (2.0%). Mothers’ race/ethnicity was primarily Caucasian, non-Hispanic (60.6%) or Hispanic

TABLE 1
Correlations Among Key Variables

Variable	M	SD	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*
1. Genotype[a]	.23	.42	1								
2. Sex[b]	.43	.49	.15	1							
3. DD Status[c]	.19	.40	.10	.01	1						
4. IQ	94.50	21.3	−.11	.03	−.79**	1					
5. Positive Parenting	12.00	1.93	−.08	.11	−.14	.34**	1				
6. Negative Parenting	3.31	3.20	−.05	−.16	.19	−.41**	−.43**	1			
7. Age 3 ED	.96	.80	−.03	−.24*	.15	−.23*	−.17	.35**	1		
8. Age 4 ED	.87	.71	.10	.05	.29**	−.32**	.02	.19	.28**	1	
9. Age 5 ED	.53	.58	.07	−.02	.24*	−.31**	.05	−.00	.33**	.38**	1
10. Age 6 ED	.29	.58	−.00	−.01	.09	−.12	−.02	.26*	.18	.25*	.18

Note: DD = developmental delay; ED = emotion dysregulation.
[a]0 = heterozygous or homozygous long genotype; 1 = homozygous short genotype.
[b]0 = boy; 1 = girl.
[c]0 = typically developing; 1 = developmentally delayed.
*$p < .05$. **$p < .01$.

(24.2%), with others African American (9.1%), Asian (3.0%), Native American (2.0%) or self-identified as other (1.0%). The vast majority of mothers were married (87.9%), as recruitment initially focused on married parents. Family socioeconomic status was generally high; 59.6% of families had an annual income above $50,000 (in year 2000 U.S. dollars), and mothers' and fathers' average years of schooling was 2 years of college.

Procedures

In recruiting participants, school and agency personnel mailed brochures describing the study to families who met selection criteria and interested parents contacted the research center closest to them. The family was visited at age 3 for an in-home confirmation of the child's development. Then the primary parent and child were assessed in the research center and/or home setting at ages 3–6, and demographic assessment and observational measures of the child's behavior and parent–child interactions were completed. Children's cognitive abilities were measured again at age 5, using the Stanford-Binet IV (Thorndike, Hagen, & Sattler, 1986), and we used this measure of IQ as a covariate in our analyses.[1] At each time point, families were monetarily compensated with $50 for their participation. To maintain study engagement among the participant families, annual holiday and birthday cards were mailed to the families.

Saliva samples were collected from the participants at the age 13 visit and genotyped. Adolescents deposited their saliva into a vial, which was then transported to the UCLA Genotyping and Sequencing Core Facility for genotyping. Technicians were masked to diagnostic status, and confidentiality was protected by labeling each sample with a unique case identifier known only to the authors. Genomic DNA was isolated from buccal cells using standard methods.

For the observations of ED and parenting in the research center, parents and children were guided by an experimenter through a series of activities, which were videotaped for later coding. Three of the activities were problem-solving tasks ranging from easy to difficult. The easy task was designed to be easily completed by the child within a short time (e.g., inset foam puzzle), with little or no help from the mother; the medium-level task was designed to be complex enough to warrant most children needing at least some assistance from their mothers (e.g., constructing a LEGO tower); and the difficult task was designed to be sufficiently difficult that it could not be solved by the child alone and always required the mother's assistance (e.g., manipulating two metal rods to get a metal ball to roll up an incline and drop into a designated hole). Developmental psychologists with extensive experience with young children were involved in the selection of the tasks, and the materials were adjusted for the children based on age and cognitive status to keep the difficulty level consistent across ages and groups.

Measures

Serotonin Transporter Genotype

The serotonin transporter genotype variable was a composite of 5-HTTLPR and rs25531. 5-HTTLPR encodes two allelic variants, a short (S) variant and a long (L) variant,

[1] Although we obtained a measure of IQ at age 3 and acknowledge that a covariate before or at the baseline measure is ideal, we elected to use the age 5 measure given the variability in earlier measures. Within our research center, we have found that the age 5 Stanford Binet index is more consistent with assessment of DD at older ages. In addition, previous researchers have urged caution in drawing predictive conclusions regarding delay using the Bayley Scales (Crowe, Deitz, & Bennett, 1987; Hack et al., 2005).

and the S variant has been associated with lower levels of serotonin transcription, expression, and function (Lesch et al., 1996). In addition, rs25531 encodes two variants as well, an A nucleotide and a G nucleotide. Researchers have found that the G variant of rs25531 functionally transforms a 5-HTTLPR L allele into an S. In accordance with previous studies (Cervilla et al., 2007; Kendler et al., 2005), we compared individuals with two-low expression alleles (i.e., SS, SL_G, L_GLG) with all others. In all analyses, children with the SS genotype were coded as 1, and children with the LL or SL genotype were coded as 0.

Emotion Dysregulation Codes (Hoffman et al., 2006)

ED was coded at child ages 3, 4, 5, and 6 years, from observations of the child's behavior during the three parent–child problem-solving tasks (the age 6 visit consisted of only two problem-solving tasks) of increasing difficulty, using the Dysregulation Coding System. This consists of Behavior Dysregulation and Emotion Dysregulation subscales, though the present study examines only the Emotion Dysregulation subscale, as trajectories of ED is the focus of this article. Each child was rated on ED for each problem-solving task, and the task ratings were averaged together to create a composite score of ED at each age. The ED ratings were significantly correlated within each time point (range = .30–.45). The coding, described next, was held consistent across the time points. The ED subscale was adapted from the parameters presented by Cole, Michel, and Teti (1994). This scale was designed to measure the appropriateness of the type, duration, and intensity of emotional expressions, as well as the lability and soothability exhibited by the child. ED ratings, therefore, involved emotional expressions exhibited by the children, but as Cole et al. suggested, ratings also captured more process-level features of the expressions and their relationship to the context, rather than simply considering the valence of the emotional expression.

The children were assigned scores ranging from 0 (*no evidence of dysregulation*) to 4 (*significant dysregulation*). A score of 1 reflected a low degree of ED and described children who (a) displayed only one or two brief emotional expressions that were inappropriate to the situation and who were able to regroup on their own or (b) displayed one or two brief instances of emotional lability and/or variability in intensity of emotional expression and usually recovered quickly from inappropriate emotional experiences. In contrast, a child receiving a score of 4 showed significant dysregulation in that he or she displayed several intense emotional expressions or displayed less intense but frequent emotional expressions for the majority of the segment; was virtually unable to regroup without the help of the parent; and was very labile, showing extreme variability in the intensity of emotion and/or very slow recovery from emotional experiences. For the purposes of this study, dysregulation of various emotions (i.e., negative vs. positive) were not examined separately, as we were interested in assessing ED at a more macrolevel. At each time point, six coders who were blind to the study hypotheses coded in pairs, first coding independently and then coming to consensus. Twenty percent of a pair's codes were compared against a master coder, and reliability was achieved and maintained when agreement was exact for 70% of the codes and within 1 point for the remaining 30%. The overall reliability of the Dysregulation Coding System was quite high (r = .90). Analyses indicated that the ED subscale also had good reliability (r = .79).

Parent–Child Interaction Rating Scale

Parenting was coded from research center and home observations of mother and child, at child ages 3 and 4. The Parent–Child Interaction Rating Scale (Belsky, Crnic, & Woodworth, 1995) measures six dimensions of parenting: positive affect, negative affect, sensitivity, stimulation of cognition, intrusiveness, and detachment. Coding teams rated each mother in each dimension using a 5-point Likert scale, from 1 (*not at all characteristic*) to 5 (*highly characteristic*), for all dyadic interaction activities. The sum of these scores were converted into z scores and combined into two composites, positive parenting and negative parenting, using a previously derived factor structure (Fenning, Baker, Baker, & Crnic, 2007). Positive parenting consisted of positive affect + sensitivity + stimulation of cognition—detachment. Negative parenting consisted of intrusiveness + negative affect. Each mother–child dyad was assigned scores for the research center observations and home observations at child ages 3 and 4, and the final scores used for analysis were the average across ages 3 and 4 scores from the research center and home.

Research assistants were trained by watching videotaped lab observations until reliability was established, defined as reaching a criterion over 70% exact agreement and 95% agreement within 1 scale point with the criterion coder. Once reliable, two research assistants were paired to code the tapes as a team. To maintain interreliability within and across contexts, a master coder, usually an advanced graduate student, was designated. Reliability was collected for 30% of the tapes. Kappa for interrater reliability was 0.71 (range = .68–.77), which is considered moderate (McHugh, 2012).

The home observations, on the other hand, were rated live during observation of family interactions. This coding included six 15-min segments, for a total of 90 min of coded interaction. Coders observed for 10 min, followed by a 5-min coding period; ratings were averaged across observation periods. Coders were trained on videotapes of home observations and attended live home observations with an experienced coder until reliability met the criterion of over 70% exact agreement with the master coder and 95%

agreement within 1 scale point. To maintain reliability within and across the two project sites, a primary coder was designated at each site. Reliability was regularly redetermined through videotapes and live home observations. Kappa coefficients were .61 and .60 for within-site reliability at the California and Pennsylvania sites, respectively, and .64 for cross-site reliability, which are all in the moderate range (McHugh, 2012).

Data Analytic Plan

LGCM was used to examine (a) yearly change in ED from ages 3 to 6 and (b) individual and interactive effects between serotonin-transporter genotype (G) and parenting behaviors (positive and negative) in predicting ED change. To this end, we implemented three LGCMs. The first model was an unconditional model which examined growth in ED, without any predictors or covariates. The second model examined the effects of G × *Negative Parenting* on growth curves of ED, and the third examined the effects of G × *Positive Parenting* on growth curves of ED.

LGCM allows for examining individual differences in change over time and exploring what factors are associated with, or potentially causal to, these (Cheong, MacKinnon, & Khoo, 2003; Krull & Arruda, 2015; Raudenbush, 2001). LGCM uses the structural equation modeling framework, with repeated measures of the outcome construct (i.e., ED) serving as indicators of latent growth factors. Several latent growth factors were estimated, including mean ED at each time point (i.e., intercept), the linear change in ED across time points (i.e., slope), as well as a quadratic trend of this growth. In contrast to multilevel modeling growth models, separate error variances are estimated for each observation of the outcome construct (Krull & Arruda, 2015). Mplus software (Muthén & Muthén, 2009) was used to estimate LGCM. All models initially included only the intercept and linear slope latent factors, and then a quadratic factor was added to examine whether it significantly improved the fit of the model. Three criteria were employed to evaluate model fit: the chi-square test, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). A nonsignificant chi-square value indicates adequate model fit, as do CFI values above .95 (range = 0–1.00) and RMSEA values below .06.

To examine ED change over time, regardless of genotype and parenting, we fit an unconditional LGCM. Then, to examine how change in ED over time is affected by serotonin transporter genotype, parenting, and their interaction, we fit conditional LGCMs, and these factors were included as predictors, controlling for child sex, IQ, DD status, and the other type of parenting (e.g., positive parenting in the negative parenting model). Sex was included as a covariate due to prior research finding significant differences in sensitivity to context by sex (Yates, Obradović, & Egeland, 2010). A significant path coefficient from serotonin transporter genotype (G), parenting (E), or the G×E interaction term to the latent intercept would indicate effects on baseline (age 3) ED, whereas a significant path coefficient leading to the growth factor(s) would reveal change in the trajectories of ED over time (Simons-Morton, Chen, Abroms, & Haynie, 2004). Multiple interaction terms were not included simultaneously because inclusion of multiple higher order terms introduces multicollinearity and instability in equations (Cohen, Cohen, West, & Aiken, 2003). Last, as Little's test determined data missingness to be Missing Completely at Random, $\chi^2(19, N = 99) = 20.08$, *ns*, Full Information Maximum Likelihood was used to estimate missing data. Compared to using listwise deletion, Full Information Maximum Likelihood leads to less biased estimates for coefficients and standard errors and decreases the likelihood of Type I error.

To aid the interpretation and presentation of our findings, graphs were made in Microsoft Excel. We constructed growth curves of ED for the positive and negative parenting models within each genotype group. Coefficients for intercept, linear slope, and quadratic slope generated in each LGCM were used to calculate expected values of ED at the mean, ±1 *SD*, and ±2 *SD*s of positive and negative parenting.

RESULTS

Descriptive Statistics

Of the 99 participants, 18 (18.2%) were homozygous for the high-expression allele, 58 (58.6%) had the heterozygous genotype, and 23 (23.2%) were homozygous for the low-expression allele (S or Lg). This genotype distribution did not deviate from the Hardy-Weinberg equilibrium ($p > .05$). Thus 23 participants were in the SS genotype group, and 76 were in the SL/LL genotype group. In addition, 80 participants were designated as typically developing (IQ > 75), and 19 as DD (IQ < 76). Table 1 shows means, standard deviations, and Pearson correlations among model variables. The average level of ED appeared to decrease across the four time points, from .96 to .29. The ED scores ranged similarly across time, from 0 to 3.5 overall. Sex was dummy-coded (0 = boys, 1 = girls) so that the average score for sex in Table 1 reflects the proportion of girls in the sample; likewise with DD status, with DD coded as 1, and with genotype, with the SS genotype coded as 1. Serotonin transporter genotype was not correlated with ED, and child sex correlated only with age 3 ED (with girls having lower ED than boys). IQ was negatively associated with ED at all time points, though not significantly at age 6. Positive parenting was not correlated with ED, whereas negative parenting positively correlated with ED at two of the time points.

LGCM Results

Change Over Time

The unconditional (without covariates) model indicated that ED significantly decreased from age 3 to 6. Without genotype and parenting in the model, a negative linear slope was found to best capture yearly change in ED across this developmental period (Intercept: B = 1.03, SE = .07, p < .001; Slope: B = –.24, SE = .03, p < .001). The unconditional model was determined to have excellent fit with the data, $\chi^2(7, N = 99) = 8.59$, *ns*, CFI = .95, RMSEA = 0.048.

TABLE 2
Negative Parenting Latent Growth Model

	Intercept[a]		*Linear Slope*[b]		*Quadratic Slope*[c]	
	B	SE	B	SE	B	SE
Intercept	−.652	1.048	2.404	1.323	−.827*	.413
Effect of						
Child DD Status	.097	.301	−.179	.384	.057	.121
Child IQ	−.002	.006	−.016*	.008	.006*	.002
Child Sex	.261	.146	−.219	.185	.047	.057
Positive Parenting	.007	.043	.067	.054	−.019	.017
SS Genotype (G)	−.072	1.036	2.870*	1.337	−1.006*	.432
Negative Parenting (E)	.402**	.138	−.442*	.173	.127*	.054
G×E	.031	.314	−.857*	.408	.320*	.133

Note: DD = developmental delay; SS = homozygous short; G×E = Gene × Environment interaction between SS Genotype and Negative Parenting.
[a]At age 3.
[b]Linear change per year from 3 to 6 years of age.
[c]Quadratic change per year from 3 to 6 years of age.
*p < .05. **p < .01.

Negative Parenting Model

In the negative parenting model, a negative quadratic slope best captured change in ED. A model with linear slope only was determined to have poor fit with the data, $\chi^2(21, N = 99) = 36.29$, p = .02, CFI = .74, RMSEA = 0.09; thus a quadratic slope was added to the model. The final negative parenting model, with quadratic slope, was determined to have excellent fit with the data, $\chi^2(13, N = 99) = 13.85$, *ns*, CFI = .99, RMSEA = 0.03. Negative parenting was the only predictor of initial levels (age 3) of ED, such that higher levels of negative parenting were associated with higher ED (Table 2). The intercept was not predicted by child IQ, DD status, sex, genotype, or positive parenting. Change over time (linear and quadratic slope) was significantly predicted by child IQ, negative parenting, child genotype, and G×E. In addition, linear and quadratic slope were significantly correlated (r = –.99) and thus should not be interpreted independently of one another. In examining the generated graphs, for children with the SL/LL genotype, lower levels of negative parenting appeared to be related to a slightly steeper decrease (Figure 1). In contrast, for children with the SS genotype, high negative parenting appeared to predict stable ED over time, whereas lower negative parenting predicted decreases in ED (Figure 2). Further, this decrease appeared to be steeper as compared to children with the SL/LL genotype.

Positive Parenting Model

The positive parenting model was largely consistent with the negative parenting model. Again, change in ED was best captured by a negative quadratic slope. A model with linear slope only was determined to have poor fit with the data, $\chi^2(21, N = 99) = 32.29$, p = .05, CFI = .81, RMSEA = 0.07; thus a quadratic slope was added to the model. The final positive parenting model, with quadratic slope, had excellent fit with the data, $\chi^2(13, N = 99) = 12.66$, *ns*, CFI = 1.00,

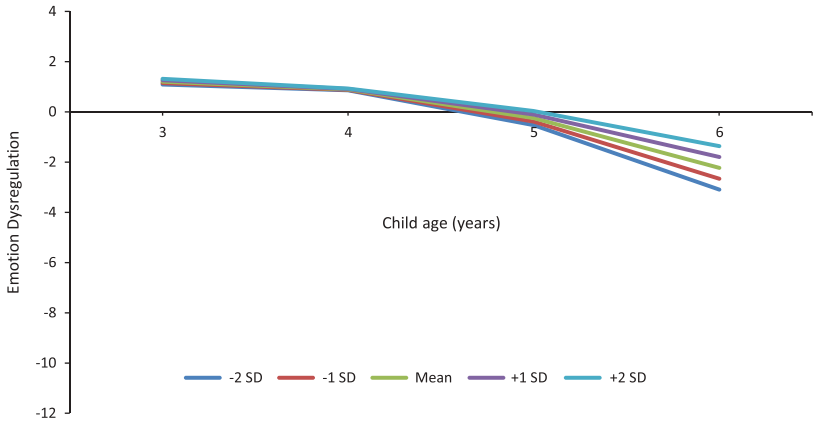

FIGURE 1 Negative parenting in predicting change in emotion dysregulation among children in the heterozygous/homozygous long (SL/LL) genotype group.

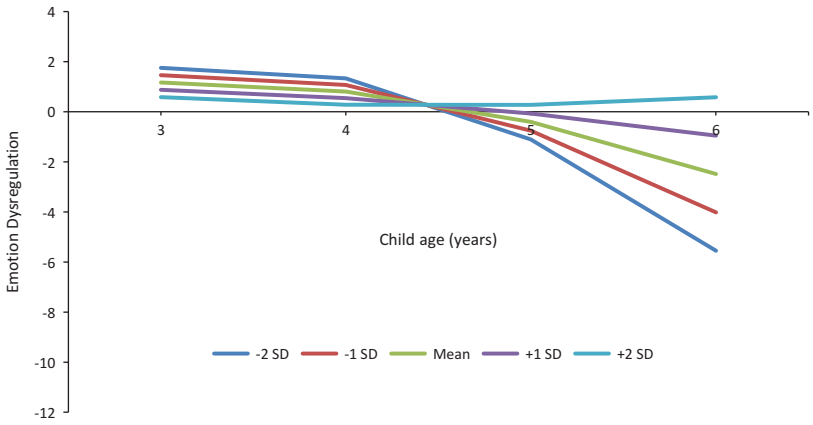

FIGURE 2 Negative parenting in predicting change in emotion dysregulation among children with the homozygous short (SS) genotype.

TABLE 3
Positive Parenting Latent Growth Model

	Intercept[a]		*Linear Slope*[b]		*Quadratic* Slope[c]	
	B	SE	B	SE	B	SE
Intercept	−.501	1.052	3.079*	1.328	1.103**	.413
Effect of						
Child DD Status	.167	.308	−.051	.392	.005	.124
Child IQ	−.002	.006	−.013	.008	.005	.002
Child Sex	.267	.145	−.239	.183	.052	.057
Negative Parenting	.395**	.132	−.561**	.167	.171**	.052
SS Genotype (G)	−1.118	1.232	−2.331	1.576	1.020*	.501
Positive Parenting (E)	−.010	.046	.020	.059	.000	.018
G×E	.097	.104	.205	.132	−.089*	.042

Note: DD = developmental delay; SS = homozygous short; G×E = Gene × Environment interaction between SS Genotype and Positive Parenting.

[a]At age 3.

[b]Linear change per year from 3 to 6 years of age.

[c]Quadratic change per year from 3 to 6 years of age.

*$p < .05$. **$p < .01$.

RMSEA = 0.00. In addition, negative parenting was the only predictor of initial levels (age 3) of ED, such that higher levels of negative parenting were associated with higher ED (Table 3). The intercept was not predicted by child IQ, DD status, sex, genotype, or positive parenting. Change over time (linear and/or quadratic slope) was significantly predicted by negative parenting, child genotype, and G×E. In addition, linear and quadratic slope were again significantly correlated ($r = –.98$) and thus should not be interpreted independently of one another. Upon examination of the graphs, for children with the SL/LL genotype, positive parenting did not appear to have an effect on the trajectory of ED (Figure 3). For children with the SS genotype, higher levels of positive parenting seemed to be associated with steeper decrease in ED (Figure 4).

DISCUSSION

We examined the effects of serotonin transporter genotype and parenting behaviors on the trajectory of ED across early childhood. We addressed important gaps in the literature by directly modeling change over time in ED and

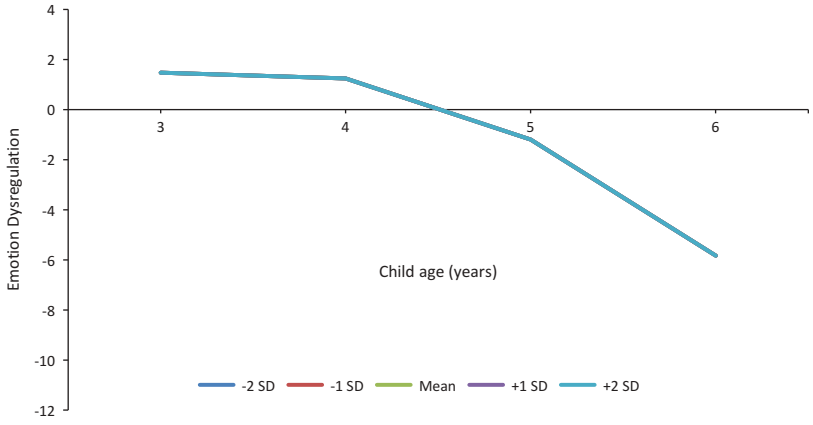

FIGURE 3 Positive parenting in predicting change in emotion dysregulation among children with the heterozygous/homozygous long (SL/LL) genotype. *Note*. Only one line is visible because all lines are overlapping.

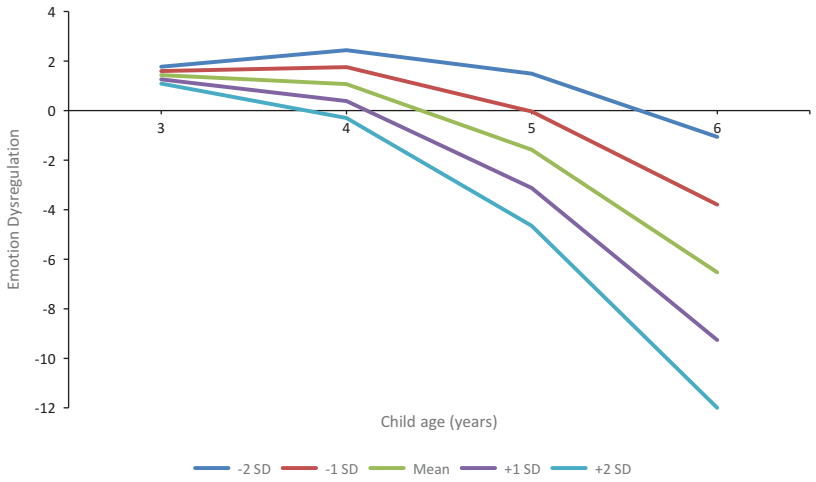

FIGURE 4 Positive parenting in predicting change in emotion dysregulation among children with the homozygous short (SS) genotype.

examining differential contributions of positive *and* negative parenting. The study extends the research on genetic plasticity to the environment by examining ED, a factor critically underlying many poor outcomes commonly studied in this framework.

Trajectory of ED

Our first aim was to identify the developmental trajectory of ED across early childhood. Using LGCM, on average, ED decreased from age 3–6 years. A negative slope best fit this trajectory, which is consistent with previous studies that examined change in regulation and related constructs over time (Blandon et al., 2008; Fabes et al., 2002; Murphy et al., 1999) and with theoretical propositions set forth by researchers in the emotion regulation field (Blair, 2002; Kopp, 1982). Decreases in ED have been proposed to reflect neurobiological and social developmental milestones. For example, brain regions (i.e., prefrontal cortex, anterior cingulate cortex, amygdala) associated with higher order cognitive functioning undergo marked maturation across childhood (Fox, 1994; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner & Gross, 2005). Maturation of these brain regions affords increased ability to recognize emotional states, positively reappraise distressing situations, and plan for decreased exposure to distressing stimuli (Blandon et al., 2008; Larsen & Prizmic, 2004). Simultaneously, from age 3 to 6, children experience changes in their social environments. At school, children are more frequently exposed to emotionally charged situations but also a plethora of strategies utilized by peers and coached by teachers for managing those emotions. Further, there is an increase in demands on children in school to independently regulate their behavior and emotions with larger class sizes and decreased supervision (Bronson, Tivnan, & Seppanen, 1995; Rimm-Kaufman & Pianta, 2000). Thus, due to brain maturation, exposure to emotionally charged but safe peer interactions, social modeling/coaching of emotion regulation strategies, and more opportunities to practice regulatory behaviors, early childhood seems to be structured to optimally support emotion regulation development. Our empirical evidence of change in ED over time underscores the dynamic nature of this construct and thus the continued need for longitudinal investigations of ED.

An important note regarding our trajectory results is that predicted values of ED at certain levels of parenting at ages 5 and 6 fell below zero, whereas the range of the ED measure was 0–4. Statistically, it is not uncommon for linear growth models to predict values that are out of the bounds of the actual outcome measure. As long as model assumptions hold (as is the case in the current study), estimates are believed to be unbiased, consistent, and efficient (Suen, Lei, & Li, 2011). In terms of real-world implications of negative predicted values, it appears that the children who went on to have the lowest (most negative) predicted dysregulation at ages 5 and 6 were predicted to score a zero at an earlier age in comparison to children with higher predicted dysregulation at ages 5 and 6. This means that these children, during a frustrating task, evidenced no emotional reactions that were inappropriate to the situation (e.g., tantrums, outbursts) at age 4, for example, whereas other children continued to display inappropriate behaviors after age 5. Beyond this interpretation, we can speculate that negative scores potentially indicate the development and implementation of positive emotion regulation strategies/skills. Our measure was of dysregulation, and thus we cannot definitively make this assertion, but displaying no tantrums or outbursts during a frustrating task likely requires some degree of conscious or automatic efforts to regulate. Future studies that include a measure that ranges from dysregulated to regulated emotional behavior are encouraged to examine this shift.

Effects of G×E on ED Trajectories

Next we set out to examine the effects of serotonin transporter genotype (G), positive and negative parenting behaviors (E), and G×E on initial levels of ED and change in ED over time.

Negative parenting emerged as the only predictor of initial levels of ED in the expected direction, such that higher negative parenting scores were associated with higher ED at age 3. When looking at yearly change in ED from age 3 to 6 years, main effects for serotonin transporter genotype and negative parenting emerged, and significant interactions emerged for G × Negative Parenting and G × Positive Parenting. For children with the SL/LL genotype, negative parenting affected the trajectory of ED to a small degree, and positive parenting appeared to have *no* effect on the trajectory of ED. However, for children with the SS genotype, negative and positive parenting behaviors predicted change in ED over this developmental period, above and beyond the effect of the other type of parenting behavior. That is, in the SS genotype group, when examining the effect of negative parenting while controlling for positive parenting, lower negative parenting predicted faster declines in ED over time, whereas high negative parenting predicted no improvement in ED from ages 3 to 6. Similarly, when examining positive parenting, children in the SS group appeared to demonstrate a steeper decrease in ED with higher levels of positive parenting.

Drawing from the G×E literature, these interactive effects provide conceptual support for the recently proposed *vantage sensitivity* framework, which asserts that certain individuals demonstrate increased sensitivity to the beneficial effects of positive environments and experiences (Pluess & Belsky, 2013; Sweitzer et al., 2013). This theory serves as a complement to dominant G×E frameworks, as vantage sensitivity focuses solely on individual differences in sensitivity (vs. resistance) to positive environments, whereas diathesis-stress focuses solely on negative experiences and differential-susceptibility examines the positive and negative in tandem. Our results suggest that individuals with the SS genotype exhibited the fastest decline in ED under conditions of low levels of negative parenting and high levels of positive parenting. They did not, however, seem to evidence comparatively worse functioning under conditions of high negative and low positive parenting; thus, individuals with the SS genotype appeared to have heightened sensitivity primarily to positive, nurturing environmental conditions. These findings emphasize the importance for researchers to continue to incorporate measures of negative *and* positive environments. In addition, most G×E studies have examined outcomes at one time point, even though biological sensitivity to the environment likely extends across time to continuously affect phenotypic outcomes across development (Ellis et al., 2011). Our study shows that for children with the SS genotype, the environment was particularly influential in shaping the developmental *pattern* of ED from age 3 to age 6, a critical period of change for ED. This supports the idea that G×E effects are developmentally meaningful with respect to how outcomes change across time. It will be important for future studies to examine G×E in the context of development and growth.

Our findings also suggest that, though modestly inversely correlated, negative and positive parenting behaviors are not simply opposite ends of the same spectrum but have unique effects. Specifically, after controlling for the effect of one type of parenting (e.g., positive) on ED, the other type of parenting (e.g., negative) continued to interact with genotype to predict ED trajectories. This is consistent with previous findings showing that positive and negative parenting are orthogonal constructs that can have separable effects on child psychopathology (Dallaire et al., 2006; Eamon, 2002) and ED (Eisenberg et al., 2001). In the context of intervention programs, these results support the use of a "two-pronged" approach to treatment, in which therapists help parents simultaneously increase their use of positive parenting strategies (e.g., praise, rewards, one-on-one time) while decreasing negative parenting strategies (e.g., criticism, harsh or inconsistent discipline). Evidence-based parenting programs, such as Parent–Child Interaction Therapy (Eyberg, 1988) and Incredible Years (IY; Webster-Stratton, 2015), are grounded in this two-pronged approach. IY, for example, first focuses on increasing positive parenting behaviors (e.g., positive affect, child-led play, praise, encouragement) and then shifts to strategies for managing behavior problems (e.g., limit setting, consequences). Positive interactions between parents and children are framed as "money in the bank," from which parents can draw when setting limits and expectations (Henderson & Sargent, 2005). Therefore, in addition to receiving instruction in strategies for increasing positive parenting behaviors, parents enrolled in IY also develop strategies to replace negative parenting behaviors. On the other hand, other parenting interventions primarily focus on symptom reduction, and a holistic (i.e., targeting positive *and* negative parenting behaviors) approach can be implied but is not explicitly targeted. For example, although a parent of an oppositional child (who also exhibits ED) may receive training on rewards and consequences, consequences are a good replacement strategy for negative behaviors such as criticism, but rewards are not necessarily equivalent to increasing the parent's expression of positive affect more globally. Parenting training interventions would benefit from more explicit differentiation of positive and negative parenting and addressing these behaviors separately. This may be particularly important for children who are at heightened genetic "risk" for ED (e.g., children with the SS genotype), because they will be particularly responsive to shifts in positive and negative parenting.

Last, this study is one of the few studies that have examined G×E in the context of ED. Most studies look at complex clinical phenotypes, such as psychiatric disorders, many of which are hypothesized to have ED underpinnings. Given that our results are conceptually consistent with the psychopathology G×E literature, our findings suggest that ED may be a critical mediator to psychopathology *through* these G×E effects. As such, they provide support for the Research Domain Criteria (Insel et al., 2010) initiative and suggest that ED may be a reliable and measurable phenotype, with clear links to both biological and behavioral

components, that underlie psychopathological outcomes. Future studies should directly test this theory, using statistical approaches such as moderated mediation, which would allow investigation of ED mediating G×E pathways to later complex clinical outcomes.

Limitations

As in all studies, there are limitations that should be noted. First, this sample was overselected for DD and thus may not be representative of community samples of children and families. As child cognitive ability has been implicated in ED (Crnic et al., 2004), we controlled for child IQ and DD status in all analyses; however, it is nonetheless possible that the generalizability of our findings may be somewhat limited. Second, in this longitudinal study, we employed similar yet somewhat different ED tasks at different ages, and different teams coded the tasks at different ages. It is possible, therefore, that some change over time can be attributed to methodological shifts between laboratory visits and coding teams. It is also possible, and perhaps more likely, that these shifts added random variance and therefore reduced the consistency of the findings. Of note, considerable efforts were made to standardize the laboratory tasks and coding methods; all tasks were puzzles completed by parent–child dyads, and coding was completed with a thoroughly specified manual across all visits. In addition, our findings that ED decreased across this developmental period are consistent with theoretical assertions and previous empirical investigations (Blandon et al., 2008; Fabes et al., 2002; Murphy et al., 1999). Thus, as we achieved replication across measures and studies, we are confident that our findings regarding change in ED over time reflect a real developmental process. Third, the sample size is an additional limitation of this study, relative to the complexity of the analyses. We encourage other researchers with sample sizes of more substantial statistical power to examine the relationships presented in this study further. Last, our results are conceptually consistent with vantage sensitivity, as serotonin transporter genotype appeared to moderate the effects of parenting on ED in more adaptive conditions (i.e., higher positive parenting and lower negative parenting). However, we did not employ formal statistical testing of differences among estimated trajectories. As few studies have examined G×E in a way that accounts for the developmental nature of phenotypes, researchers have not yet established the quantitative methods needed to test for G×E within a longitudinal framework. Given the accumulating evidence for G×E effects on developmental patterns, it is important for researchers to work toward establishing methods for quantitative evaluation of such effects.

Conclusions

Study results indicated that child ED decreases across early childhood (age 3 to 6 years) and that individual differences in developmental trajectories of emotion regulation are, in part, attributed to G×E interactions. Serotonin transporter genotype interacted with both negative and positive parenting behaviors in predicting growth curves of ED. Children with the SS genotype appeared to decrease in ED at a faster rate, under conditions of low negative parenting or high positive parenting. Children with the SL/LL genotype had trajectories of ED that were minimally affected by parenting. These findings provide conceptual support for the vantage sensitivity framework and highlight the importance of examining outcomes in the context developmental growth, as opposed to a single time point. In addition, our results underscore the importance of programs for parents of young children with ED. Specifically, interventions should be targeted at increasing positive parenting *and* decreasing negative parenting, as the two were found to have unique effects on ED.

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