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Interplay of Genes and Early Mother-Child Relationship in the Development of Self-Regulation from Toddler to Preschool Age

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Abstract

Background—A broad capacity for deliberate self-regulation plays a key role in emotion regulation. This longitudinal investigation from infancy to preschool age examines genotype by environment (G × E) interaction in the development of self-regulation, using molecular measures of children's genotypes and observed measures of the quality of early mother-child relationship, as reflected in attachment organization in infancy.

Methods—In 89 children, we assessed the polymorphism in the serotonin transporter gene (5-HTTLPR, *ss/sl* vs. *ll* allele status), security of attachment to mothers at 15 months in the Strange Situation, and children's ability for self-regulation at 25, 38, and 52 months, using behavioral batteries of tasks that called for deliberately suppressing a dominant response and performing instead a sub-dominant response.

Results—There was a robust G × E interaction between genetic risk and the quality of early relationship. Among children who carried a short 5-HTTLPR allele (*ss/sl*), those who were insecurely attached developed poor regulatory capacities, but those who were securely attached developed as good regulatory capacities as children who were homozygotic for the long allele (*ll*). There was no effect of security for *ll* homozygotes.

Conclusions—Those findings, consistent with diathesis-stress model, bridge research on self-regulation in typically developing children with research on non-human primates and research on psychopathology. They also indicate that a secure attachment relationship can serve as a protective factor in the presence of risk conferred by a genotype.

Keywords

ATTACHMENT; EMOTION REGULATION; GENETICS; G × E interactions; EFFORTFUL CONTROL; 5-HTTLPR polymorphism; PARENT-child RELATIONSHIPS

Interplay of Genes and Early Mother-Child Relationship in the Development of Self-Regulation from Toddler to Preschool Age Children's capacity for deliberate self-control, often studied as self-regulation, inhibitory control, or effortful control, has been inherently linked to emotion regulation. Fox and Calkins (2003) listed effortful control as an intrinsic temperament factor that underpins self-control of emotion. Sometimes, emotion regulation is seen as a subset of broader self-regulatory processes, and sometimes, emotion regulation and self-regulation are seen as having consonant qualities (e.g., Kopp, 1989). Self-regulation, the capacity to control deliberately one's affect and behavior, accounts for children's growing capacity to voluntarily uncouple their behavioral response from the immediate emotional impulse (Eisenberg & Spinrad, 2004; Kopp, 1982). For example, children gradually become able to suppress or delay touching prohibited objects or engaging in a strongly desired but

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prohibited action when so instructed, to suppress an aggressive or angry act when such an act is unacceptable, and to sustain an aversive activity when requested to do so by a caregiver. Such deliberate effortful control and the control of emotion share a key requirement – a suppression of an impulsive response and instead, carrying out an opposite act. Consequently, effortful control and modulated expression of both positive and negative affect have been often conceptually and empirically linked (Carlson & Wang, 2007; Eisenberg, Fabes, Guthrie, & Reiser, 2000; Eisenberg & Spinrad, 2004; Kieras, Tobin, Graziano, & Rothbart, 2005; Kochanska, Murray, & Harlan, 2000; Kochanska, Aksan, Penney, & Doobay, 2007; Posner & Rothbart, 2000; Rothbart & Bates, 2006).

Because of its critical importance for socialization, the fundamental capacity for self-regulation has been extensively studied (Kopp, 1982; Kochanska et al., 2000). The ability to suppress a predominant response –often one immediately desired by the child --and to perform instead a subdominant response, often unappealing, but required by caregivers and other social agents begins to develop in the second year (Kochanska et al., 2001; Kochanska, et al., 2000; Posner & Rothbart, 2000; Rothbart & Bates, 2006). Individual differences in the capacity for self-regulation have been strongly implicated in many aspects of adaptive development and psychopathology (Maccoby, 2007).

A modern approach to development must involve the integration of constructs across multiple levels, “from neurons to neighborhoods” (Shonkoff & Phillips, 2000). Consequently, those constructs include both biological and environmental factors that interface in complex ways (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Fox & Calkins, 2003). “Genotype × Environment” (G × E) interactions are among most often studied forms of that interface (Rutter, Moffit, & Caspi, 2006). A G × E interaction occurs when environmental experience moderates the effect of a person’s genotype on physical or mental health outcomes, or when a genotype moderates an environmental effect (Moffit, Caspi, & Rutter, 2005).

In developmental psychology, studies of such interactions have often focused on children’s temperament as a moderator of socialization (Bates, Pettit, Dodge, & Ridge, 1998; Belsky, 1997; Belsky, Hsieh, & Crnic, 1998; Kochanska, Aksan, & Joy, 2007; Rothbart & Bates, 2006). In those studies, temperament has been considered a proxy for genotype, even though, with a few exceptions (Fowles & Kochanska, 2000), it has been measured behaviorally. Acknowledging genetic contributions to temperament, other scholars have adopted behavior genetic designs, such as twin and adoption studies (Goldsmith, 2003; Goldsmith, Lemery, Buss, & Campos, 1999).

Recently, a surge in inter-disciplinary collaborations has brought exciting advances in the study of G × E interactions. In a landmark article, Caspi and colleagues (Caspi et al., 2003) demonstrated that a history of stressors moderates the effect of genetic risk on depression. In developmental science, research on non-human primates that allows for systematic manipulation of early environment has led to groundbreaking insights into G × E interactions in early social-emotional development (Suomi, 2004, 2005, 2006; Champoux, Bennett, Shannon, Higley, Lesch, & Suomi, 2002).

Multiple studies of G × E interactions have focused on a polymorphism in the serotonin (5-HT) transporter gene regulatory region (5-HTTLPR). Serotonin is an inhibitory neurotransmitter in the central nervous system. Dysfunctions in the serotonergic system have been strongly implicated in broadly ranging regulation of mood, attention, and psychopathology (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001; Champoux et al., 2002; Lucki, 1998; Sourbrie, 1986; van Goozen, Fairchild, Snoek, & Harold, 2007). The 5-

HTT gene has also been linked to neural areas that are parts of the executive attention network involved in self-regulation (Posner, Rothbart, & Sheese, 2007).

The 5-HTTLPR polymorphism has two common alleles, the short (*s*) and the long (*l*). The short allele (*s*) has been linked to reduced 5-HTT transcription, lower 5-HTT protein levels, and diminished serotonin re-uptake compared to individuals with the long (*l*) allele. Individuals who are either homozygous for the short allele (*ss*) or heterozygous (*sl*) have been found to be at risk for a range of emotional and behavioral maladaptive outcomes. Those outcomes include under-regulated, impulsive, excessively and inappropriately aggressive, risk-taking behavior, alcohol use, as well as depressive or anxious psychopathology (Barr et al., 2004; Lesch et al., 1996; Propper & Moore, 2006; Suomi, 2005).

Even more importantly, however, both human and non-human studies have increasingly documented substantial G × E interactions between the genetic risk associated with 5-HTTLPR polymorphism (having a short allele) and environmental or experiential factors. Generally, those studies have shown that environmental factors moderate the link between the genetic risk and maladaptive outcomes: Individuals who carry a short allele develop a host of problems when they also experience sub-optimal or stressful environmental conditions. Suomi and colleagues have repeatedly demonstrated that monkeys with ss or *sl* genotype show a range of significant self-regulatory problems (impulsivity, inappropriate aggression, orienting problems, risk taking), but *only* if they had been separated from their mothers and raised in a peer nursery. For monkeys raised in natural, supportive mother-infant relationships, there was no effect of genotype (Champoux et al., 2002; Suomi, 2004, 2005, 2006).

Emerging non-experimental evidence with human children has consistently dovetailed with those findings. The “high-risk genotype” (having a short allele, *ss* or *sl*) has been found to confer a risk for depression, but only in children who have been exposed to maltreatment, abuse, or neglect (Kaufman et al. 2004,2006), and for fearfulness, but only in children who have grown up in a family with poor social support (Fox et al., 2005).

To our knowledge, however, no study with human children has addressed G × E interactions in the development of self-regulation, using a multi-trait multi-method longitudinal design, molecular measures of the 5-HTTLPR genotype, repeated assessments of self-regulation from toddler to preschool age, and observational measures of the early rearing environment, specifically, attachment security in infancy. We present such data in the current article.

A secure early mother-child relationship has been repeatedly implicated as important for the development of emotional regulatory abilities, ranging from regulating one’s own emotional arousal to complex executive capacities (Cole, Martin, & Dennis, 2004; Hofer, 1994; Schore, 2001; Sroufe, 1996, 2005). Biological factors, however, including genes, also play an important role, as demonstrated by molecular (Diamond, Briand, Fossella, & Gehlbach, 2004; Posner, 2005; Suomi, 2005) and behavioral genetic studies (Goldsmith, 2003; Goldsmith et al., 1999). Most pertinent to the current study is the following hypothesis, based on research with primates (Suomi, 2006, p. 52): “Secure attachment relationships somehow confer resiliency to individuals who carry alleles that may otherwise increase their risk for adverse developmental outcomes (“maternal buffering”)”.

To date, however, few studies of G × E interactions operationalized early environmental risk specifically as insecure attachment. Often, beneficial or harmful early parent-child relationships have been approximated using other measures. In his work with primates, Suomi (2004) compared peer and maternal rearing. Kaufman et al. (2004) identified the

history of child maltreatment using legal and social agencies' records. Fox et al. (2005) used mothers' reports of perceived social support.

To examine the hypothesis that early security may be an important environmental factor that moderates the effect of genotype on children's self-regulation, we assessed children's attachment security with their mothers at the end of the first year, using the established Strange Situation paradigm. Children's genotype, 5-HTTLPR polymorphism, was assessed as *ss/sl* or *ll* allele status.

We targeted a major aspect of self-regulation: children's effortful control, or the capacity to deliberately suppress a dominant behavior and to perform instead a sub-dominant behavior (Rothbart & Bates, 2006). We assessed it behaviorally at 25, 38, and 52 months, using a multi-task battery at each age.

Based on the extant non-human and human research, we expected security to moderate the impact of the genotype on children's capacity for self-regulation. In children with high-risk genotypes, who have a short allele, *ss* or *sl*, we expected to find the effect of their early attachment security, such that those who had been insecure would develop poorer self-regulatory capacities than those who had been secure. We did not expect to find such effect in children homozygous for the long allele, *ll*.

In this context, we also examined whether the G × E effect would conform to *the differential susceptibility hypothesis* or to *the genetic vulnerability hypothesis*, a subject of a recent debate (Belsky, 2005; Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007). The former hypothesis predicts that children with certain vulnerable traits --highly reactive, highly negative, carriers of the short 5-HTT allele --respond more strongly to *both* negative and positive variations in the environment, and thus, may end up with *worse or better* outcomes than children without those dispositions. In contrast, the latter hypothesis, while also predicting that children with those vulnerabilities do *worse* than others when exposed to harmful environments, it does not predict that they would do *better* than their low-risk peers when exposed to beneficial environments. In other words, beneficial environments can merely buffer children from risks conferred by genotypes.

Method

Participants and Design

Participants responded to ads in local community venues. The two-parent families represented a broad range of education, from high school (24% of mothers, 30% of fathers) to post-college (21% of mothers, 20% of fathers), and of income, from under $20,000 (8%) to over $70,000 (34%). Among mothers, 91% were White, 3% Hispanic, 1% each African American, Asian, Pacific Islander, and 3% "other" non-White. Among fathers, 83% were White, 8% Hispanic, 3% African American, 3% Asian, and 3% "other" or missing. In 20% of families, one or both parents were non-White. This research complied with all ethical principles, including informed consent, and it has been annually reviewed by the authors' IRB.

The longitudinal study included multiple assessments during lengthy home and laboratory sessions: at Time 1, when children were 7 months (N = 102, 51 girls); at Time 2, 15 months (N = 101, 51 girls), at Time 3, 25 months (N = 100, 50 girls), at Time 4, 38 months (N = 100, 50 girls); at Time 5, 52 months (N = 99, 49 girls). Only mother-child data are presented here.

The sessions were videotaped for later behavioral coding. Independent teams coded different measures. Typically, 15%-20% of cases were used for reliability, with more used for rare codes. The coders realigned periodically to prevent drift. The measures were aggregated at multiple levels to assure their robustness (Rushton, Brainerd, & Pressley, 1983).

Assessment of Children's Attachment Security, 15 Months

Paradigm—The standard *Strange Situation* was conducted at the beginning of the session. It was coded by professional attachment coders at another university, blind to all information regarding the children.

Coding—Reliability, kappa, based on 20 randomly selected cases, was .78 for the four attachment categories (secure, avoidant, resistant, disorganized/unclassifiable) and .85 for the coding of secure versus insecure attachment. All cases coded with low confidence by one coder and all disorganized/unclassifiable cases were double-coded and adjudicated.

Genotyping: Assessment of 5-HTTLPR Status, 52 Months

Mothers of 89 children agreed to participate in this assessment. DNA was obtained using buccal swabs and genotype at the 5HTTLPR was determined for each sample (Barry, Kochanska, & Philibert, in press; Bradley, Dodelzon, Sandhu, & Philibert, 2005; Philibert et al., 2002). Eighty eight of 89 samples were successfully genotyped. There were 13 *ss* homozygotes (3 girls, 10 boys), 47 *sl* heterozygotes (23 girls, 24 boys), and 28 *ll* homozygotes (18 girls, 10 boys). Hardy Weinberg equilibrium testing was non-significant ($p < 0.66$). The difference in gender distribution across different genotypes, *ss/sl* vs. *ll* was not significant, $\chi^2 = 3.35$, $df = 1$, $p < .10$.

There were 48 secure and 40 insecure children (12 avoidant, 16 resistant, and 12 disorganized/unclassifiable). There were no significant differences in the distribution of security vs. insecurity across gender, $\chi^2 = 2.22$, $df = 1$, *ns*. The difference in the distribution of security vs. insecurity across *ss/sl* and *ll* genotypes, however, was significant, $\chi^2 = 6.93$, $df = 1$, $p < .01$; among insecure children, 33 had *ss/sl* genotype, and 7 were *ll* homozygotes, whereas among secure children, 27 had *ss/sl* genotype, and 21 were *ll* homozygotes.

Assessment of Children's Self-Regulation, 25, 38, and 52 Months

Batteries of tasks, most of which were multi-trial to yield robust scores, were administered. They were interspersed with other contexts during the laboratory sessions. These batteries, developed in our laboratory, have been described in other articles (e.g., Kochanska, Coy, & Murray, 2001; Kochanska et al., 2000, 2007), and are widely used in the field. Thus, the description here will be brief (information is available upon request).

The tasks capture the following five inter-related core aspects of the child's capacity to deliberately suppress a dominant response and to perform instead a sub-dominant response: delaying, slowing down gross and fine motor activity, suppressing/initiating activity to signal, lowering voice, and effortful attention. Not all functions were assessed at all ages; some tasks were repeated, and new tasks were added as permitted by the child's increasing maturity. Every task was presented as a game rather than a prohibition or request, and the child was praised regardless of performance.

Battery at 25 months—There were five tasks. Four captured *delaying* (two tasks that required waiting to reach for an M&M placed under a cup, and in two tasks that required waiting to unwrap a gift) and one captured *suppressing/initiating activity to signal* (taking turns while building a block tower).

Battery at 38 months—There were nine tasks. Three *delaying* tasks involved waiting to reaching for M&M, deliberately choosing a prize from a box filled with small toys, and waiting to unwrap a gift). Two *slowing-down* tasks called for slowing motor activity (walking a 6-ft line; and guiding a toy turtle slowly along a curved path to the barn). One task called for *suppressing or inhibiting a response* to one type of signal and *producing or initiating* a response to another (a turn-taking game). *Lowering* voice was tapped in a whispering task. Two *effortful attention* (Stroop-like) tasks required ignoring a dominant perceptual feature of a stimulus for the sake of a subdominant feature, Day-Night and Snow-Grass (Carlson and Moses, 2001).

Battery at 52 months—There were 14 tasks. Five *delaying* tasks again involved waiting to reach for an M&M, waiting to chew an M&M placed on the child's tongue, deliberately choosing a prize from a box filled with toys, and waiting to unwrap two gifts. Three *slowing-down* tasks called for slowing fine and gross motor activity (drawing lines, walking a line, and moving a toy toward a play barn). Three *suppressing/initiating activity to signal* tasks involved a go-no go response (to red and green signs, and to commands given by a toy bird and toy dragon), and turn-taking while building a block tower. *Lowering voice* was assessed in a whispering task. Two *effortful attention* (Stroop-like) tasks were Day-Night and Snow-Grass.

Coding and reliability—The codes were strongly behaviorally based and required little inference. For each trial, higher score reflected better capacity for self-regulation (coding manuals are available from the first author). The scores were then averaged across trials, where applicable. Reliability of coding was extremely high across all three ages and across many teams. Reliabilities, kappas, ranged from .71 to 1.00, and alphas ranged from .81 to 1.00.

Data aggregation—The scores were averaged across trials, where applicable. The individual task scores were then standardized and aggregated into *self-regulation composites* at 25, 38, and 52 months (Cronbach's alphas .71, .67, and .72; means and standard deviations, −.01 and .66; −.01 and .52; −.01 and .53, respectively). Those composites were longitudinally stable, *r's* ranging from .37 to .57, all p's < .001, average $r = .49$, alpha = .73, and thus they were aggregated across 25, 38, and 52 months into an *overall composite of self-regulation*, $M = -.01$, $SD = .46$.

Results

Multiple regression analysis predicting self-regulation

First, we conducted a hierarchical multiple regression, where the overall composite of self-regulation across 25, 38, and 52 months was the dependent variable, attachment security (insecure vs. secure), genotype (5-HTTLPR status, homo-or heterozygotic for the short allele, *ss/sl*, versus homozygotic for the long allele, *ll*), and their interaction, attachment security × genotype, were the predictors, and child gender was the covariate. Table 1 presents the results.

In the final equation, girls had higher scores on the self-regulation composite; girls, $M = .13$, $SD = .38$, boys, $M = -.15$, $SD = .50$, consistent with many published studies (Bjorklund & Kipp, 1996). The effects of security and genotype were significant, but they were qualified by the significant security × genotype interaction, and thus should not be interpreted.

Follow-up tests of the interaction

To probe the interaction effect, we performed tests of the simple slopes (Aiken & West, 1991). The results indicated that the effect of security on children's self-regulation was significant for children with high-risk genotypes, *ss/sl*, b=1.18,SE= .50, $p < .02$, but not for children with low-risk genotypes, *ll*, b = .41, SE = 1.56, ns. As depicted in Figure 1, among children who carried a short 5HTTLPR allele (*ss/sl*), those who were insecurely attached developed poor regulatory capacities, but those who were securely attached developed as good regulatory capacities as children who were homozygotic for the long allele (*ll*). There was no effect of security for *ll* homozygotes.

Discussion

This study bridges a molecular genetic approach with the science of relationships within a longitudinal design using extensive observational measures. The straightforward findings dovetail with the extant human and non-human research, inform the debate on G × E interactions in human development, and have implications for research on risk and resilience. The expected significant G × E interaction was supported. Children's attachment security moderated the effect of their genotypes: 5-HTTLPR polymorphism (specifically, having a short allele, *ss* or *sl*) was associated with a diminished self-regulatory capacity from age 2 to 4 ½. That risk, however, was significant *only* for children who had been insecurely attached to their mothers at the end of the first year, and it was absent for children who had been securely attached. As Suomi (2006) proposed, secure attachment indeed served as a buffer against risk otherwise conferred by the child's genotype.

In addition to the analyses for the overall composite of self-regulation across 25, 38, and 52 months, we also examined parallel equations predicting self-regulation scores at each of those assessments. The standardized regression coefficients for the G × E interaction terms were, respectively, −.29, −.28, and −.40. Although, as often happens when composite measures are disaggregated (Rushton et al., 1983), only the last of the three reached significance, $p < .05$, nevertheless those separate data suggest that a similar process operated across the studied age range. Consequently, given also the considerable developmental stability of the self-regulation construct, we presented the most robust analyses for the trait-like self-regulation composite from 25 to 52 months. Future research on G × E interactions regarding other developmental constructs should not, however, preclude a possibility that those effects may appear in only some, and not all, developmental periods.

Our findings appear more consistent with *the genetic vulnerability hypothesis* than with *the differential susceptibility hypothesis* (Belsky, 2005; Belsky et al., 2007). Children with high-risk genotypes who had formed secure attachment with their mothers had as good --but not better --selfregulatory capacity as children with low-risk genotypes. Security seemed to function as a buffer against the risk conferred by having the short 5-HTT allele.

Future studies need to address the causal mechanisms that account for those effects. One such potential mechanism involves a possibility that security is particularly consequential for children with high-risk genotypes, because it can enhance (or hinder) effective emotional arousal modulation in individuals who are genetically less well equipped to handle this task (Herrmann et al., 2007; Hofer, 1987). In contrast, children with low-risk genotypes possess more effective physiological regulation capacities, and for them, relationship-based supports are less important. This may be a promising future direction of research.

Limitations of this study include a relatively small sample, and the findings should be replicated in a larger group of families. Furthermore, the contemporary developmental psychopathology approach calls for the study of risk and protective factors in both typically

developing and atypical populations, whereas in this study, the children were normally developing and the families were generally well functioning. Most measures likely fell mostly within the adaptive range and their variability was relatively constrained, although the sample did include sufficient numbers of secure and insecure children to permit the analyses, consistent with the distribution of security reported in the review by van IJzendoorn, Schuengel, and Bakermans–Kranenburg (1999) for "normal US samples, age < 24 months" (p. 230). Yet even so, the findings were significant. The studied effects are likely to be amplified in children and families at a higher risk, for example, families where early parent-child relationships are grossly sub-optimal (Kaufman et al., 2004).

Conclusion

The findings inform both basic and translational research on emotional development. They support diathesis-stress or dual-risk models by demonstrating an interaction between genotype and environment in children's development of effective self-regulation, a critical skill implicated in modulating emotion expression and exercising restraint over immediate desires. The results support attachment scholars' views that early security provides a foundation and scaffolding for the child's self-regulation (Hofer, 1994; Schore, 2001; Sroufe, 1996, 2005). They additionally reveal that the role of security is particularly significant for children whose genotypes may put them at risk for self-control deficiencies. Early security can be seen as a critical protective factor that can offset or buffer developmental risks conferred by genetics, consistent with several bodies of animal and adult human research.

Research that bridges molecular genetics and social relationships to explain adaptive and maladaptive developmental pathways is only beginning to blossom. That research undoubtedly holds promise for a fuller understanding of the complex nature of adaptive and maladaptive pathways in emotional and social development.

- Growing evidence documents genotype by environment (G × E) interactions for adaptive and maladaptive developmental outcomes.
- Polymorphism in the serotonin transporter gene, 5-HTTLPR (*ss/sl* vs. *ll* allele status), specifically, having a short allele (*ss/sl*), has been linked to deficits of self-regulation and other forms of psychopathology. Environment has been shown to moderate that link, such that individuals who carry a short allele develop poor outcomes when they also experience sub-optimal or stressful environmental conditions.
- This longitudinal multi-method study found that among *ss/sl* children, those who as infants had been insecurely attached to their mothers developed poor regulatory capacities in toddler and preschool years, but those who had been securely attached developed as good regulatory capacities as children homozygotic for the long allele (*ll*). There was no effect of security for *ll* homozygotes.
- The findings, consistent with diathesis-stress model, indicate that secure attachment can serve as a protective factor in the presence of risk conferred by a genotype.

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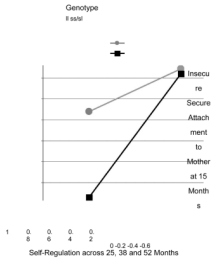

Figure 1.

Security of infants' attachment to mother at 15 months moderates the effect of infants' 5-HTTLPR genotypes on the overall self-regulation composite across 25, 38 and 52 months (simple slopes of attachment security on *ss/sl* and *ll* children's self-regulation).

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Table 1

Children's attachment security to mothers at 15 months, 5-HTTLPR status, and their interaction as predictors of children's self-regulation from 25 to 52 months

Predictor(s)	Outcome: children's overall self-regulation composite[a]					
	Step 1		Step 2		Step 3	
	F	Beta	F	Beta	F	Beta
Child gender[b]	13.97***	−.37	9.83**	−.32	9.86**	−.31
R^2 = .14, $F(1,86)$ = 13.97***						
Attachment security[c]			1.29	.12	4.13*	.24
5-HTTLPR status[d]			3.48+	.19	7.60**	.48
R^2 = .20, $F(3,84)$ = 6.97***						
Attachment security ×5-HTTLPR status					4.14*	−.40
R^2 = .24, $F(4,83)$ = 6.46***						

[a] Composite of self-regulation at 25, 38, and 52 months.

[b] 0 = Girls, 1 = Boys.

[c] 0 = Insecure, 1 = Secure.

[d] 0 = *ss/sl*, 1 = *ll*

+ p < .10.

* p < .05.

** p <.01.

*** p < .001.