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Multivariate Behavior Genetic Studies of Parenting and Early Child Development

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Multivariate Behavior Genetic Studies of Parenting and Early Child Development

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Dedication

This dissertation is dedicated to my loving brother, Edmund K. Cheung, who always encourages me to pursue my passion and supports me in every possible way. His love and guidance shape the essence of who I am and give me confidence to overcome any challenges in life. I would also like to dedicate this dissertation to the memory of my dearest mother, Winnie S. Pun, who showed me my potential early on and gave me the best life coach one could have ever asked for – my brother.

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Without anyone of you, I would not be where I am today. From the deepest of my heart, THANK YOU.

Multivariate Behavior Genetic Studies of Parenting and Early Child Development

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Transactional perspectives posit that caregivers, in addition to instilling their norms and values in their children through socialization, modify their parenting practices in response to children's characteristics. Although both phenotypic and behavioral genetic literature have consistently documented the mutual influences of parenting and early child development on one another, multivariate examinations of these parent- and child-driven processes are scarce. This dissertation investigates gene-environment interplay at both general and specific dimensions of parenting and early child development. All three studies capitalize on primary data collection from a population-based sample of families with twins or multiples aged 0-6 years. Study 1 presents evidence for substantial genetically mediated child influences on both general and specific dimensions of parenting. Study 2 supports the importance of child-environment dynamics in early child development by presenting evidence for age-related growth in generalist genetic influences shared across multiple ability domains. To further elucidate the association between parenting and child development, Study 3 probes the extent to which genetic and environmental pathways mediate the associations between an array of parenting practices and child outcomes at both general and specific dimensions. These

three studies together highlight the complexity and dynamic nature of associations between parenting and early child development.

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MULTIVARIATE BEHAVIOR GENETIC STUDIES OF PARENTING AND EARLY CHILD DEVELOPMENT

A transactional perspective posits that caregivers, in addition to instilling their norms and values in their children through socialization, modify their parenting practices in response to children's characteristics. For example, higher levels of maternal sensitivity may lessen the development of child externalizing behaviors and, at the same time, lower levels of child externalizing behaviors may evoke higher levels of maternal sensitivity (Belsky, Pasco Fearon, & Bell, 2007). Previous work on this topic has commonly examined parenting behaviors and child outcomes in isolation. However, for both parenting and child functioning, it is well-known that distinct domains fall along a series of correlated dimensions (e.g., Bornstein, Tamis-LeMonda, Hahn, & Haynes, 2008; Carroll, 2003; Gottfredson, 2002; Gray & Steinberg, 1999) and function interdependently rather in isolation of each other. Moreover, among existing literature on the transactions between parenting and early child development, relatively few studies capitalized on genetically informative data to test the role of child genetic influences in such transactional processes. In a series of three multivariate behavioral genetic studies, the current dissertation capitalizes on primary data collection from a population-based sample of families with twins and multiples aged 0-6 years to investigate gene-environment interplay in parenting and early child development.

Previous behavioral genetic studies have consistently shown that parents modify their behaviors in response to child genetically driven characteristics (Avinun & Knafo, 2013; Klahr & Burt, 2014); yet, nearly all of these studies have examined parenting practices in isolation of one another. Using a multivariate approach, Study 1 examines child genetic and environmental influences on both general and specific dimensions of parenting. Results indicate that broad contextual and parental characteristics contribute

substantially to both general parenting style and specific parenting practices; at the same time, parents adjust their childrearing behaviors, particularly specific parenting practices, in response to child genetically driven characteristics.

Parents matching their childrearing behaviors to children's genetically driven characteristics serves as a specific example of a more general class of processes by which children select and evoke environmental experiences on the basis of their genetically influenced characteristics. Transactional models predict that these experiences, in turn, have causal effects on child development. For instance, relatively small genetic advantages in a specific cognitive ability or exploratory behavior may evoke greater cognitive stimulation from caregivers, which may in turn promote the development of many different cognitive abilities. Based on this rationale, a number of scholars have hypothesized that transactional processes should lead to the emergence and strengthening of common genetic effects shared across multiple ability domains (e.g., Dickens, 2007; van der Maas et al., 2006). Using a multivariate approach, Study 2 examines developmental transformations in the genetic structure of early child abilities to ascertain the role of child-environment dynamics in early child development. Consistent with transactional models, results indicate that genetic influences on early child abilities increase across ages and much of this developmental increase is localized to genetic influences shared across multiple ability domains.

To further elucidate the interplay between parenting and child development, Study 3 probes the extent to which genetic and environmental pathways mediate the associations between an array of parenting practices and child outcomes at both general and specific dimensions. Results indicate that shared environment is the predominant contributor to associations between multiple dimensions of parenting and child outcomes. This suggests that the broad contextual and parental characteristics (i.e., environmental

factors shared by children reared together) contributing to the parenting behavior of interest also contribute to the corresponding child outcome of interest (i.e., parent-to-child influences). Results also indicate nontrivial genetic mediations at the broad dimension, suggesting that parents adjust their general childrearing behaviors to genetically driven variation in general child outcomes. Nonshared environmental mediation, which indicates differential childrearing behavior possibly for reasons idiosyncratic to parents (i.e., parent-to-child influences) or related to non-genetically driven variation in the child outcome of interest (i.e., environmental factors unique to a child; child-to-parent influences), appears to play a rather trivial role in the associations between parenting practices and child outcomes at the broad dimension.

All three studies are motivated by a model in which caregivers, in addition to exerting their influences on early child development via socialization, modify their parenting practices in response to children's genetically driven characteristics. These studies together highlight the complexity and dynamic nature of associations between parenting and early child development.

Chapter 1: Multivariate Behavioral Genetic Analysis of Parenting in Early Childhood

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Parents play an important socializing role in the development of myriad cognitive, emotional, and behavioral skills during early childhood (Carlson & Corcoran, 2001; Chang, Schwartz, Dodge, & McBride-Chang, 2003; Dubow & Ippolito, 1994; Patterson, Debaryshe, & Ramsey, 1989; Simpkins et al., 2009; Stormshak, Bierman, McHahon, & Lengua, 2000). Parenting behaviors that are overtly didactic, cognitively stimulating, emotionally supportive, and warm are typically considered higher in quality and are empirically associated with positive developmental outcomes (Hutchings et al., 2007; McKee et al., 2007; Pettit, Bates, & Dodge, 1997; Tamis-LeMonda, Shannon, Cabrera, & Lamb, 2004; Yeung, Linver, & Brooks-Gunn, 2002). However, as convincingly argued by Bell (1968), correlations between parenting behaviors and developmental outcomes are not by themselves prima facie evidence for socialization effects. While research on parenting skills training provide support for parent-to-child causation (e.g., Eyberg et al., 2001; Taylor & Biglan, 1998; Webster-Stratton, 1994), child social skills training alone has also been found to improve parenting quality (e.g., Webster-Stratton, Reid, & Hammond, 2004) and provide support for child-to-parent causation.

As any given parenting behavior has the potential to arise from a mixture of parent-driven and child-driven processes (e.g., Avinun & Knafo, 2013; Kendler & Baker, 2007; Klahr & Burt, 2014), these processes may have distinct implications for the

[1] Drs. Elliot Tucker-Drob and Paige Harden are the principle investigators of the Texas "Tiny" Twin Project, which provides data used in Study 1. Drs. Tucker-Drob and Harden also provided feedback to the first author throughout the development and publication of Study 1.

covariation among parenting behaviors. In this paper, we apply a multivariate, behavioral genetic methodology to address the question: To what extent do broad and narrow dimensions of parenting reflect young children's genetically variable characteristics? We measure multiple correlated parenting behaviors among a twin sample of ages 0-5 years. Specifically, we examine the generality and specificity at which child genetically-driven characteristics influence parenting.

Parental Socialization

According to parental socialization models, parents act as the main agent in acculturating young children (Maccoby, 1992; Patterson et al., 1989). Socialization models hold that parents instill their values, beliefs, and expectations in their children through various actions. One possible way is to directly communicate their judgment and experiences to their children. Another possible way is through reasoning with their children when their children misbehave. At the same time, children potentially internalize these standards by observing their parents in various situations. Social learning theory holds that children identify with their parents through imitating their parents' actions (Bandura 1969; Bandura & Huston, 1961).

Parent-Child Transactions

A parental socialization model overlooks child-driven processes as a source of parent-child correlations (Bell, 1968). According to the transactional perspectives, not only do parents transmit their values and culture to their child through socialization, but the child also affects his or her parents' behaviors (Bell, 1968; Bell & Chapman, 1986; Belsky, 1984; Sameroff & MacKenzie, 2003). Longitudinal cross-lagged analyses indicate both parent-to-child and child-to-parent influences. Among 5- to 10-year-old children, higher levels of maternal sensitivity predicted lower levels of externalizing

behaviors approximately 4 years later, and lower levels of externalizing behaviors predicted higher levels of maternal sensitivity 4 years later (Belsky, Pasco Fearon, & Bell, 2007). Likewise, Lansford and colleagues (2011) followed children from 6 to 9 years old and found that, even after accounting for the deleterious effect of physical discipline on later behavioral development, higher levels of antisocial behaviors provoked higher levels of physical discipline a year later. Tucker-Drob and Harden (2012a) also found support for a transactional perspective; after accounting for a child's cognitive ability at 2 years old, parental cognitive stimulation when the child was 2 years old predicted the child's reading ability 2 years later, and cognitive ability at 2 years old predicted the level of parental cognitive stimulation the child received 2 years later. These studies demonstrate that it is important to examine not only parental socialization processes, but also child-driven processes that are likely to affect parents' behaviors.

Multidimensional Structure of Parenting

The role of parenting in child development has been extensively studied (e.g., Baumrind, 1971; Burt, Krueger, McGue, & Iacono, 2003; Burt, McGue, Krueger, & Iacono, 2005; Dallaire et al., 2006; Fearon, Bakermans-Kranenburg, van IJzendoorn, Lapsley, & Roisman, 2010; Gershoff, Lansford, Sexton, Davis-Kean, & Sameroff, 2012; Groh, Roisman, van IJzendoorn, Bakermans-Kranenburg, & Fearon, 2012; McLeod, Wood, & Weisz, 2007; Repetti, Taylor, & Seeman, 2002). Parenting practices can generally be classified by their hypothesized impact on child development – positive parenting for practices that promote adaptive functioning or negative parenting for those that contribute to maladjustment. Bradley and Caldwell (1995) proposed a two-tiered, multidimensional approach to further classify parenting practices by their function, source, modality, intensity, reactivity, and complexity. For the purpose of this study, we

focus on the functional aspect of parenting: 1) sustenance, which emphasizes children's nutritional needs and physical development; 2) stimulation, which facilitates children's cognitive development; 3) support, which promotes children's regulatory skills and social-emotional adjustment; 4) structure, which emphasizes organization and predictability in relation to children's adaptive functioning; and 5) surveillance, which emphasizes children's safety and welfare.

Conceptual and empirical studies of parenting primarily describe parent-to-child influences and strongly indicate that distinct parenting practices are correlated at multiple levels. However, to our knowledge, the etiology of such multivariate structures has not yet been examined. Thus, an outstanding question is whether infants and preschoolers shape parenting uniquely for each specific dimension, or evoke general parenting approaches spanning multiple dimensions and therefore contribute to the co-occurrence of distinct parenting practices. In this study, we first construct a hierarchical structure of parenting using the framework described above. Then, to elucidate child-to-parent influences and their role in the co-occurrence of distinct parenting practices, we examine these child-driven processes within the hierarchical structure of parenting using a behavioral genetic approach.

"Heritable" Environments

Based on a transactional perspective, how parents raise their children depends both on their own characteristics, including personality, values, beliefs and experience, and on their children's characteristics, such as temperament and behavioral tendencies. As described previously, one powerful source of evidence for transactional processes is a longitudinal cross-lagged analysis; another useful approach is a behavioral genetic design. The classic twin model uses information from different types of biological

relatives (e.g., monozygotic [MZ] twins versus dizygotic [DZ] twins, adopted siblings versus biological siblings) to decompose variance in a phenotype into three components: variance due to genetic differences (*A*), variance due to environmental differences across kinship pairs (shared environment; *C*), and variance due to environmental differences within kinship pairs (nonshared environment; *E*). In most cases, this quantitative behavioral genetic approach is used to study child phenotypes, such as cognitive performance or externalizing behaviors. Genetic influences are inferred from the extent to which more genetically related individuals (e.g. MZ twins) are more similar on the phenotype (e.g. aggression) than are less genetically related individuals (e.g. DZ twins). Shared environmental influences are inferred from the extent to which children reared in the same family resemble one another on the phenotype after accounting for their genetic relatedness. Nonshared environmental influences are inferred from the extent to which genetically identical children reared in the same home (i.e. MZ twins reared together) do not perfectly resemble one another on the phenotype. Although less commonly done, this same approach can be applied to environmental measures, such as parenting practices. As Plomin (2004) quipped, "environments have no DNA" (p. 346). Rather, the "heritable" variation in an environmental measure reflects the extent to which environments have become matched to children's genotypes. For example, when parents alter their parenting practice in response to children's temperament, which is strongly linked to genetic dispositions, this child effect shows up as genetic influences on parenting in a child-based behavioral genetic model.

The influence of children's genes on their environment (e.g., quality of parenting received) is called gene-environment correlation (*r*GE; Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983); children select, construct, and evoke environmental experiences on the basis of their genetically influenced dispositions and behaviors. Three

forms of *r*GE are frequently discussed (see Figure 1). First, genes that children received from their parents also contribute to the environments they are reared in; this is called passive *r*GE. For example, hostile or overreacting parents are more likely to pass on genes that predispose children with difficulties regulating their emotions. Second, persons in a child's environments (e.g., parents or teachers) notice and respond to some genetically driven characteristics unique to a child; this is called evocative *r*GE. For example, for children who are genetically disposed to enjoy educational activities such as reading, parents notice their children's interest in learning and provide higher levels of cognitive stimulation to them. Third, genetic propensities may determine which environment or situation children choose to engage in; this is called active *r*GE. For example, children who have genetically driven proclivities toward engaging with and solving difficult problems may be more likely to pursue educational activities related to natural sciences, thus increasing their likelihood of pursuing careers in science later in life. Evocative and active *r*GE differ in a subtle but important way. In evocative *r*GE, environments change to match the genetically driven characteristics unique to a child without the child's active choice to seek out such accommodations; whereas in active *r*GE, a child proactively selects environments that are congruent with his or her unique, genetically driven characteristics. In a child-based twin design (as opposed to a design looking at parents who are twins), genetic influences on environmental measures capture evocative and active *r*GEs that lead to differentiation of environments by the genotypes of children.

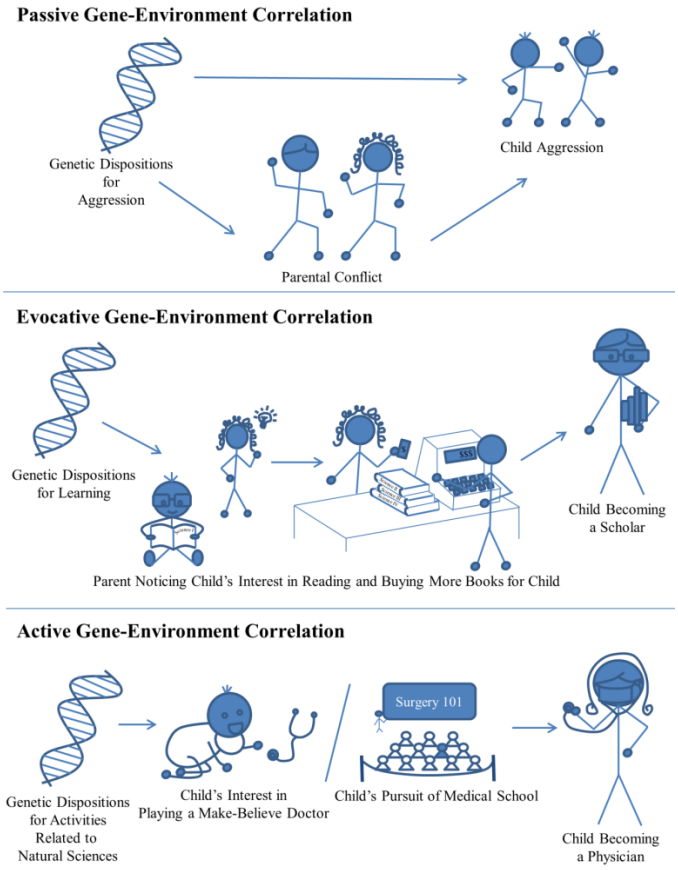

Figure 1. Scenarios delineating passive, evocative, and active gene-environment correlations (*r*GE). In the top panel (delineating passive *r*GE), genetic dispositions for aggressive behaviors are expressed both in parents and, after offspring inherited the associated genes, in the children; at the same time, these genetic dispositions for aggressive behaviors contribute to a family environment in which children learn to act aggressively by, for example, observing their parents' aggressive behaviors. In the middle panel (delineating evocative *r*GE), a child's genetic dispositions for learning manifest through his early interest in reading; his mother notices his interest in reading and reinforces it by purchasing more books for him (i.e., his mother responds to his genetically driven behaviors without his active role in asking for such response from her), which facilitates his pursuit of a scholarly career. In the bottom panel (delineating active *r*GE), a child's genetic propensities for engaging with activities related to natural sciences manifest through her early interest in activities such as playing a make-believe doctor as an infant and in pursuing medical school after college, which facilitate her pursuit of a career in medicine (i.e., she proactively seeks experiences that reinforce her genetic dispositions).

In addition to sharing genetic materials and therefore eliciting similar experiences, children can be exposed to the same environments simply because they are reared together. In a child-based twin design, shared environmental variation in parenting indicates that both children in a family receive similar parenting, regardless of the children's genetic relatedness. This family-level environmental variation is consistent with a parental socialization model, in which parenting is driven by the parents' own characteristics (e.g., personality or psychopathology) and by family-level broad contextual factors (i.e., environmental factors in which parents are embedded; e.g., neighborhood characteristics, marital relationships, etc.) rather than by the characteristics of their children. Applying child-based behavioral genetic methods to parenting measures helps disentangle parent-driven processes (reflected in shared environmental variance) from child-driven processes (reflected in genetic variance).

Previous Univariate Behavioral Genetic Studies of Parenting

Avinun and Knafo (2013) meta-analyzed 32 child-based twin studies and found that children's genetically driven characteristics explained 23% of variance in parenting, whereas environmental differences at the family level (i.e., shared environments) and those at the child level (e.g., nonshared environments) explained 43% and 34% of variance in parenting, respectively. Similarly, meta-analyzing 47 genetically informative studies conducted at the child level, Klahr and Burt (2014) observed that 23-40%, 27-39%, and 32-44% of variation in parenting was attributed to children's genetically driven characteristics, shared environments, and nonshared environments, respectively. Although results across studies converge to suggest that children exert a moderate influence on parenting, certain parenting practices are more susceptible to child

influences than others. For example, meta-analyzing three studies that used parent-reports, Avinun and Knafo found that affect-based parenting practices (e.g., warmth) were more influenced by genetically driven child characteristics than those related to discipline (e.g., control; 25% versus 11%). Meta-analyzing 18 studies that used parent-reports, Klahr and Burt observed that genetically driven child characteristics explained a greater portion of individual differences in maternal negativity (51%) than in maternal warmth (35%) and maternal control (36%). These findings suggest that child genetic effects on parenting vary by types of parenting behaviors.

Multivariate Behavioral Genetic Approach

Although univariate behavioral genetic approaches have been applied to a host of different parenting behaviors, and the heritabilities of different parenting behaviors have been compared to one another, we are not aware of previous research that has used a multivariate behavioral genetic approach to examine the extent to which child genetically driven characteristics influence general and specific dimensions of parenting. A multivariate behavioral genetic model examines the extent to which the same genetic and environmental factors contribute to different phenotypes. That is, do child genetically and environmentally driven characteristics affect broad dimensions underlying multiple parenting practices, or unique dimensions that are idiosyncratic to individual parenting practices? If general dimensions of parenting vary by children's genetically influenced characteristics, such child-to-parent effects would be evident as genetic influences on the higher-order parenting factor(s). Alternatively, if a specific dimension of parenting varies by children's genetically influenced characteristics, such child-to-parent effects would be evident as genetic influences unique to that particular parenting domain or measure.

Current Study

Our study uses a twin sample to investigate whether child genetically and environmentally driven characteristics influence broad parenting approaches, specific parenting practices, or both. We first ascertain the phenotypic structure of parenting measures in the current study and then examine the loci of child genetic and environmental influences on parenting within this structure.

METHODS

Participants

Data for the current article come from a downward extension of the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013) to the first five years of life. Families with twins or multiples of ages 0 to 5 years who lived in the state of Texas were identified using birth records provided by the Texas Department of State Health Services and then sent a recruitment letter. Recruitment also includes attending annual conventions of Texas Mothers of Multiples, sending electronic recruitment letters to associated email list serves, and accepting families for participation who registered on the Texas Twin Project website after hearing about the study from friends or from web searches. Data were collected and managed using a secure, web-based application designed for research data collection and management (Harris et al., 2009). Once a family enrolled in the study, the primary caregiver either received an online survey link that was unique to that particular family or, if he or she preferred, a paper survey. Participating families were sent longitudinal follow-up surveys until the twins or multiples turned 6 years old. Surveys were sent every 2 months after last survey completion for children from birth until 2 years, every 3 months for children between ages 2 and 3 years, every 5 months for children between ages 3 and 5 years, and one last survey after the twins or multiples

turned 5 years old. Parenting items were administered only when the twins or multiples were equal to or older than 6 months old.

Recruitment and longitudinal follow-ups for the project is ongoing. For the current article, data were available from 236 individual twins. The average age of twins was 2.50 years (*SD* = 1.23) at the first survey wave. This twin sample is 73.73% Caucasian, 4.24% Latinos/Hispanic, 3.39% African American, and 13.56% racially mixed. Among these 236 individual twins, 1.69% of their primary caregivers reported the highest completed level of parental education as high school graduate, 8.47% as some college, 34.75% as college graduate, and 55.08% as beyond college. Most of these primary caregivers were the birth mothers of their twins (94.92%).

Zygosity was determined using ratings of physical similarity of twins in a pair; each primary caregiver rated on a 3-point Likert scale ranging from *Not Alike* to *Exactly Alike* on 4 items and on a dichotomous scale on 8 other items. Ratings on the 4 items with a 3-point Likert scale were re-coded to have the same range of possible scores as the 8 items with dichotomous scales. One item was reverse-coded so higher scores indicate greater physical similarity. We averaged the ratings on all 12 items for each twin pair to compute an overall score on physical similarity, resulting in a bimodal distribution with a range of 0 to 1. Pairs with a score of 0 to .74 were assigned to be DZ twins and those with a score equal to .75 or higher were assigned to be MZ twins. Zygosity assignment using physical similarity ratings has been found to be highly reliable and comparable to results from DNA sampling (Forget-Dubois et al., 2003; Price, Freeman, et al., 2000; Rietveld et al., 2000). This resulted in our sample of 48 individual MZ twins (22 males and 26 females), 106 same-sex individual DZ twins (44 males and 62 females), 80 opposite-sex individual DZ twins (40 males and 40 females), and 2 individual DZ twins with incomplete sex information (1 male and 1 unreported).

Each family provided data for at least one wave (if the family completed the survey only at the baseline wave) and up to nine different waves (if the family completed the survey at the baseline wave as well as multiple follow-up waves). When available, we included both data collected at the baseline wave and those collected at follow-up waves in our analyses. To account for nonindependence of data obtained from the same families across different survey waves, we used the Complex Survey option in *Mplus* statistical software (Muthén & Muthén, 2010) in all of our structural equation modeling. In other words, we considered observations on the same individual from different survey waves as independent datapoints and preserved the precision of our estimates by accounting for potential biases from nonindependence of data on the same individual across waves. With 86 individual twins providing observations only at the baseline wave and 150 individual twins providing 150 observations at the baseline wave and 306 observations at follow-up waves, our final sample contains a total of 542 observations. Among these 542 observations, the average age at measurement was 2.44 years (*SD* = 1.21).

Measures

Parental Cognitive Stimulation

Each primary caregiver rated the amount of *Daily Stimulating Interactions* and *Learning Activities* each of their twins received from them. Twenty-one items on parental cognitive stimulation were created in-house to include activities that are commonly theorized as facilitating a child's cognitive development. The 2-factor solution from our Exploratory Factor Analysis (EFA) fit our data reasonably well (χ^2 [274] = 523.03, $p < .05$, RMSEA = .03, CFI = .85, TLI = .82), with the two factors correlating at .33 ($p < .05$). Sample items for *Daily Stimulating Interactions* include "How often do you play peek-a-boo/hide-and-seek or hide a toy for your child to find?" and those for *Learning*

Activities include "How often do you bring your child to outdoor educational activities or field trips (e.g. visiting the zoo, petting farm, science museum, nature center, etc.)?" Primary caregivers provided responses on a 7-point Likert scale ranging from *All the Time* to *Not at all/Not applicable*. All items were reverse-coded so higher scores indicate higher levels of parental cognitive stimulation. Cronbach's alpha was .86 for the 12 items measuring *Daily Stimulating Interactions* and .68 for the 8 items measuring *Learning Activities*.

Parenting Young Children (PARYC)

Primary caregivers rated their use of 3 different parenting practices on each twin separately: *Supporting Positive Behavior*, *Setting Limits*, and *Proactive Parenting*. PARYC contained 7 items for each of these 3 domains and was designed to measure caregivers' self-perceived use of various parenting practices on young children (McEachern et al., 2012). Primary caregivers reported their use of each strategy by marking on a continuum ranging from 0 – *Not at all* to 100 – *Most of the Time*. Using a sample of 579 infants and preschoolers, McEachern and colleagues found the factor loadings to be moderately high for all PARYC items and a modest to moderate association between PARYC and standardized measures of parenting perceptions, child behaviors, and utilization of community services. Sample items for *Supporting Positive Behavior* include "Reward your child when s/he did something well or showed a new skill," those for *Setting Limits* include "Stick to your rules and not change your mind," and those for *Proactive Parenting* include "Give reasons for your requests (such as We must leave in five minutes, so it's time to clean up)." We averaged the item ratings to obtain a factor score for each of the 3 domains and higher scores indicate greater use of that particular type of parenting strategies. Cronbach's alpha was .70 for the 7 items

measuring *Supporting Positive Behavior*, .86 for those measuring *Setting Limits*, and .91 for those measuring *Proactive Parenting*.

Emotion Socialization Questionnaire (ESQ)

Primary caregivers rated their levels of *Emotional Support*, *Emotional Magnification*, and *Emotional Neglect* towards each twin on 9 items per domain from ESQ. ESQ is a self-report questionnaire adapted from the Emotions as a Child scale (EAC) designed to measure caregivers' reactions to children's expression of sadness, anger, and fear (Klimes-Dougan et al., 2007). Primary caregivers rated how typical they would react in a particular way to children's expression of negative emotions by marking on a continuum ranging from 0 – *Not at All Typical* to 100 – *Very Typical*. We averaged the item ratings for each domain so higher scores indicate higher levels on that factor. Previous work using EAC has found a moderately high correlation between administrations at different times and a moderate Cronbach's alpha for each EAC domain (Garside & Klimes-Dougan, 2002; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001). Other studies have found modest to moderate associations between EAC and standardized measures of child behavioral maladjustment (i.e., Child Behavior Checklist, Youth Self-Report, and Teacher's Report Form; Brand & Klimes-Dougan, 2010; O'Neal & Magai, 2005). Sample items for *Emotional Support* include "Asked my child about it" and "Helped my child deal with the problem;" those for *Emotional Magnification* include "Got sad myself" when the child was sad and "Got angry with my child" when the child was angry; and those for *Emotional Neglect* include "Gave my child space to deal with it" and "I didn't respond." Cronbach's alpha was .75 for the 9 items measuring *Emotional Support*, .79 for those measuring *Emotional Magnification*, and .70 for those measuring *Emotional Neglect*.

Parenting Scale (PS)

Primary caregivers rated their use of 3 different types of discipline strategies on each twin separately: 5 items on *Laxness*, 5 items on *Overreactivity*, and 3 items on *Hostility*. PS was designed to measure caregivers' self-perceived use of maladaptive discipline in response to children's different misbehaviors (Arnold, O'Leary, Wolff, and Acker, 1993; Rhoades & O'Leary, 2007). Primary caregivers reported their use of each strategy by marking on a continuum ranging from 0 to 100, of which each end represented an opposite approach. Reitman and colleagues (2001) observed that PS was strongly correlated with standardized measures of other parenting practices and moderately correlated with parental characteristics such as stress and parenting attitude. Sample items for *Laxness* include "When I want my child to stop doing something: [ranging from 0] *I firmly tell my child to stop* [to 100] *I coax or beg my child to stop*;" those for *Overreactivity* include "When my child misbehaves: [ranging from 0] *I usually get into a long argument with my child* [to 100] *I don't get into an argument*;" and those for *Hostility* include "When my child misbehaves: [ranging from 0] *I rarely use bad language or curse* [to 100] *I almost always use bad language*." Seven of the 15 items were reverse-coded so higher scores indicate greater use of the discipline strategy listed. We averaged the item ratings to obtain a factor score for each of the 3 domains and higher scores indicate greater use of that particular type of parenting strategies. Cronbach's alpha was .80 for the 5 items measuring *Laxness*, .82 for the 5 items measuring *Overreactivity*, and .41 for the 3 items measuring *Hostility*. Cronbach's alpha for *Hostility* was also lower in Rhoades and O'Leary (2007; i.e., .52 for maternal ratings and .49 for paternal ratings), who applied Spearman-Brown correction and obtained a corrected alpha of approximately .80 if the scale was based on 10 items. Similarly, we applied Spearman-Brown correction and obtained a corrected alpha of .70 for *Hostility* if

it was measured by 10 items. Moreover, *Hostility* measures some severe forms of hostile behaviors toward a child that likely have a low base rate in the population; both parents in Rhoades and O'Leary's study and those in our current study endorsed rare use of such parenting practices, which may have contributed to the relatively low Cronbach's alpha for *Hostility*. Despite the low ratings on *Hostility*, it is important to include this variable in our analyses because of its unique theoretical relevance for development (Rhoades & O'Leary, 2007). Additionally, because being lax in discipline is indicative of a lack of consistent enforcement of discipline, we renamed *Laxness* as *Laxness-Consistent Parenting* in all our structural equation modeling.

Hierarchical Structure of Parenting Variables

Based on the theoretical classification of parenting outlined earlier, we grouped the parenting measures in this study by their impact on child development – *Positive Parenting* for parenting qualities that are thought to positively influence child development, and *Negative Parenting* for those that are thought to negatively influence child development. We then categorized the *Positive Parenting* measures into three domains: 1) *Cognitive Stimulation* – defined by *Daily Stimulating Interactions* and *Learning Activities*, both of which facilitate children's cognitive development through engaging their attention and promoting information-processing skills; 2) *Warmth*, defined by *Supporting Positive Behavior* and *Emotional Support* – both of which facilitate children's social and emotional adjustment through promoting effective regulatory and coping skills; and 3) *Structured Parenting* – defined by *Setting Limits*, *Proactive Parenting*, and *Laxness-Consistent Parenting*, all of which facilitate children's adaptive functioning through creating a predictable environment and promoting organizational skills. Similarly, we categorized the *Negative Parenting* measures into two domains: 1)

Maladaptive Emotional Socialization – defined by *Emotional Magnification* (i.e., matching children's negative emotional display) and *Emotional Neglect* (i.e., ignorance and indifference to children's negative emotional display), both of which impede children's social and emotional adjustment through modeling ineffective approaches to stress and dismissing their emotional needs; and 2) *Escalation* – defined by *Overreactivity* and *Hostility*, both of which impede children's adaptive functioning through creating an unstable environment and modeling ineffective problem-solving skills.

RESULTS

Data Preparation and Descriptive Statistics

Data on several parenting variables were transformed to remove skewness (see Table 1). Depending on the degree of skewness, each variable was either square-root or log transformed to better conform to a normal distribution. Additionally, because

Table 1. Skewness Statistics of Variables before and after Transformations

	Before		Transformation	After	
	Kurtosis/*SE*	Skewness/*SE*		Kurtosis/*SE*	Skewness/*SE*
Emotional Support	.33 / .22	-.85 / .11	Square-root	-.81 / .22	.09 / .11
Laxness-Consistent Parenting	3.49 / .22	1.27 / .11	Square-root	-.35 / .22	.04 / .11
Emotional Magnification	2.05 / .22	1.27 / .11	Square-root	-.31 / .22	.36 / .11
Emotional Neglect	3.25 / .22	.55 / .11	Square-root	.41 / .22	-.48 / .11
Overreactivity	.19 / .22	.77 / .11	Square-root	-.12 / .22	-.12 / .11
Hostility	5.76 / .22	2.17 / .11	Log	-.84 / .22	.23 / .11

estimates from twin designs can be biased by influences of age and sex on phenotypes examined (McGue & Bouchard, 1984), we partialled out the linear and quadratic effects of age, sex, and the interactions of age and sex on all variables using multiple regression analyses; unstandardized residuals were used in all of our subsequent analyses. Table 2 provides the descriptive statistics and the correlation matrix for all 11 parenting variables. All positive parenting measures were moderately and positively correlated with each other, as were all the negative parenting measures. Correlations between positive and negative parenting measures were generally negative and moderate in magnitude. Table 2 also lists the number of observations available for each parenting variable.

Table 2. Descriptive Statistics (before Transformation and Standardization) and Correlations between Parenting Measures

		N	*M* (*SD*)	1	2	3	4	5	6	7	8	9	10
1	Daily Stimulating Interactions	540	4.08 (1.39)	-									
2	Learning Activities	540	3.78 (.92)	**.49**	-								
3	Supporting Positive Behavior	510	81.69 (11.72)	**.45**	**.40**	-							
4	Emotional Support	508	89.36 (8.90)	**.20**	**.19**	**.39**	-						
5	Setting Limits	506	77.47 (16.02)	**.33**	**.36**	**.66**	**.35**	-					
6	Proactive Parenting	504	76.18 (20.62)	**.54**	**.30**	**.59**	**.37**	**.74**	-				
7	Laxness-Consistent Parenting	494	19.61 (15.22)	**-.19**	**-.18**	**-.42**	**-.29**	**-.52**	**-.35**	-			
8	Emotional Magnification	506	11.68 (11.02)	.06	-.03	**-.25**	**-.28**	**-.32**	-.08	**.22**	-		
9	Emotional Neglect	508	21.03 (10.92)	**.22**	.06	.03	**-.14**	.05	**.11**	.04	**.28**	-	
10	Overreactivity	496	22.08 (16.58)	**.16**	**-.15**	**-.27**	**-.23**	**-.35**	-.07	**.15**	**.55**	**.25**	
11	Hostility	498	6.45 (8.78)	.04	-.05	**-.16**	**-.26**	**-.19**	-.06	**.09**	**.37**	**.22**	**.50**

Note. *N* for each variable represents the number of observations available on that particular variable in our univariate analyses. Bolded = $p < .05$.

Univariate ACE Analyses

To examine child genetic and environmental effects on primary caregivers' parenting behaviors, we analyzed our data with structural equation modeling using full-information maximum likelihood estimation in *Mplus* statistical software (Muthén & Muthén, 2010). Rather than list-wise deletion of data from participants with only partial data, full-information maximum likelihood estimation in *Mplus* capitalizes on all available data under the assumption that any systematic patterns of missingness related to unobserved scores on the outcome variables are statistically accounted for by the available data included in the model. We first fit a univariate ACE model to each parenting measure to examine child genetic and environmental contributions to parenting. Variance in each parenting measure was decomposed into a linear combination of three biometric components: *A*, *C*, and *E*. Cross-twin correlations between corresponding *A*s were fixed to 1 for MZ twins, who share nearly all of their segregating genetic materials, and to .5 for DZ twins, who, on average, share approximately half of their segregating genetic materials. Cross-twin correlations between corresponding *C*s were fixed to 1 for all twin pairs. *E*s were uncorrelated.

In the context of this child-based design, in which the *A*, *C*, and *E* factors at the child level were fit to child experiences instead of child phenotypes, the interpretations of these factors are somewhat novel. *A* represents variation in parenting that associates with genetically driven child characteristics. *C* represents variation in parenting that associates with child environmental factors that contribute to similarities in parenting across children reared in the same family. As Klahr and Burt (2014) described, "shared environmental influences on parenting at the level of the child include such potentially important factors as the family's socioeconomic status, neighborhood characteristics, and

culture. However, they also include the effects of parental characteristics (e.g., parent personality and other genetically influenced characteristics), at least to the extent that these characteristics create similarities in parenting across children regardless of the siblings' genetic relatedness" (p.573). *E* represents variation in parenting that associates with non-genetic differences between siblings in a pair as well as any measurement error. Measurement error, as its name implies, occurs only to variables at the measurement level. Therefore, *E* influences operating at the latent-factor levels suggest that primary caregivers consistently treat each twin differently due to environmental factors that are unique to each twin.

Results from the univariate ACE model are presented in Figure 2, which indicate that parenting practices are largely, although not exclusively, influenced by shared environmental factors, i.e., factors at the child level that contribute to similarities in parenting across twins reared in the same family after accounting for the twins' genetic relatedness. Although the estimates for child genetic effects on *Laxness-Consistent Parenting* and *Emotional Neglect* did not reach statistical significance, these univariate findings indicate a general presence of child genetic influences on parenting. Univariate findings also indicate the presence of nonshared environmental variation in parenting behaviors. However, it is important to note that, in these univariate models, nonshared environmental influences include measurement error. To the extent that nonshared environmental variation exists beyond measurement error, this means that caregivers, to some extent, treat their twins differently for reasons unrelated to the twins' genetically influenced characteristics but possibly in response to environmentally driven child characteristics. In sum, the univariate results indicate that parenting largely reflects broad contextual and parental characteristics and, at the same time, varies within families for both genetic and non-genetic reasons.

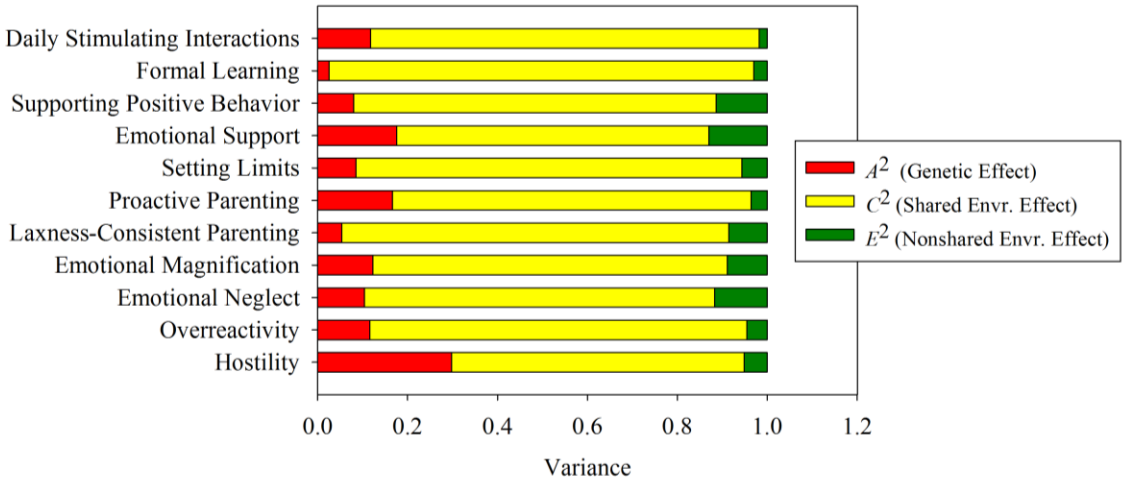

Figure 2. Proportion of total variance in each parenting measure explained by child genetic and environmental factors. Shared Envr. Effect = Shared Environmental Effect. Nonshared Envr. Effect = Nonshared Environmental Effect. All parameter estimates are standardized. All parameter estimates, except for child genetic effects on *Laxness-Consistent Parenting* and *Emotional Neglect*, are statistically significant at $p < .05$.

Multivariate Phenotypic Analyses

To ascertain the multivariate structure of the parenting measures employed, we conducted two confirmatory factor analyses (CFA) at the phenotypic level. We first fit a model with only one level of broad factors representing the five hypothesized domains of parenting (CFA 1), and estimated the loadings of our parenting measures on these factors and the correlations among the five factors. Chi-square goodness-of-fit test indicated that CFA 1 fit our data reasonably well (χ^2 [34, N = 540] = 90.94, $p < .01$, MLR scaling = 2.15, AIC = 14092.52, BIC = 14277.05, RMSEA = .06, CFI = .92, TLI = .87). Correlations observed among the five domains of parenting are consistent with the existence of two higher order dimensions – *Positive Parenting* and *Negative Parenting* (see Table 3). We then fit a hierarchical model with 5 broad factors representing our

parenting domains and 2 additional higher-order factors representing the clustering of these domains (CFA 2), which also fit our data well (χ^2 [38, N = 540] = 89.55, $p < .01$, MLR scaling = 2.33, AIC = 14097.70, BIC = 14265.07, RMSEA = .05, CFI = .93, TLI = .89). Results from CFA 2 are consistent with our proposed structure of parenting. Chi-square goodness-of-fit comparison indicates that CFA 1 and CFA 2 fit our data equivalently well ($\Delta\chi^2$ = 3.41, Δdf = 4, $p > .05$; see Table 3 for parameter estimates from both CFAs). CFA 2, being more parsimonious, is therefore the preferred phenotypic model in representing the multivariate structure of the parenting variables. This general pattern was also supported by results from our post-hoc Exploratory Factor Analysis, which indicated that a 2-factor solution (χ^2 [34, N = 540] = 144.67, $p < .01$, MLR scaling = 2.01, AIC = 14187.74, BIC = 14372.27, RMSEA = .08, CFI = .84, TLI = .75) fit our data better than a 1-factor solution (χ^2 [44, N = 540] = 245.03, $p < .01$, MLR scaling = 2.37, AIC = 14458.53, BIC = 14600.15, RMSEA = .09, CFI = .72, TLI = .64; $\Delta\chi^2$ = 80.56, Δdf = 10, $p < .01$; see Table 4 for estimates from these EFA solutions), with the correlation between the two factors estimated at -.46 ($p < .05$).

Table 3. Parameter Estimates (with Confidence Intervals in Brackets) from Phenotypic Confirmatory Factor Analyses

Subordinate Measures/Factors		Higher Order Factors	CFA 1	**CFA 2**
Daily Stim. Int.	On	Cognitive Stimulation	.80 [.65, .94]	**.80 [.67, .93]**
Learning Activities			.71 [.57, .85]	**.71 [.57, .84]**
Sup. Pos. Beh.		Warmth	.78 [.67, .88]	**.78 [.68, .88]**
Emotional Support			.50 [36, .64]	**.50 [.36, .64]**
Setting Limits		Structured Parenting	.94 [.89, .99]	**.94 [.88, 99]**
Proactive Parenting			.80 [.74, .86]	**.80 [.74, .86]**
Lax.-Cons. Parenting			-.53 [-.65, -.40]	**-.53 [-.65, -.40]**
Emo. Mag.		Mal. Emo. Soc.	.87 [.48, 1.25]	**.89 [.49, 1.29]**
Emotional Neglect			.31 [.15, .48]	**.30 [.13, .47]**
Overreactivity		Escalation	.86 [.74, .97]	**.85 [.73, .97]**
Hostility			.56 [.43 .69]	**.56 [.44, .69]**
Cognitive Stimulation		Positive Parenting	-	**.62 [.44, .80]**
Warmth				**1.09 [.94, 1.23]**
Structured Parenting				**.82 [.70, .93]**
Mal. Emo. Soc.		Negative Parenting	-	**.70 [.40, .99]**
Escalation				**.96 [.79, 1.13]**
Cognitive Stimulation	With	Warmth	.69 [.48, .89]	-
		Structured Parenting	.50 [.31, .69]	
		Mal. Emo. Soc.	(-.17 [-.36, .02])	
		Escalation	-.43 [-.60, -.27]	
Warmth		Structured Parenting	.89 [.72, 1.05]	
		Mal. Emo. Soc.	-.55 [-.85, -.25]	
		Escalation	-.70 [-.86, -.24]	
Structured Parenting		Mal. Emo. Soc.	-.41 [-.59, -.24]	
		Escalation	-.54 [-.71, -.37]	
Mal. Emo. Soc.		Escalation	.68 [.36, 1.01]	
Positive Parenting		Negative Parenting	-	**-.68 [-.86, -.51]**

Note. Bolded = best-fitting model. Estimate in parentheses = $p > .05$. CFA 1 = Phenotypic confirmatory factor analysis with 1 level of higher-order parenting factors. CFA 2 = Phenotypic confirmatory factor analysis with 2 levels of higher-order parenting factors. Daily Stim. Int. = Daily Stimulating Activities. Sup. Pos. Beh. = Supporting Positive Behavior. Lax.-Cons. Parenting = Laxness-Consistent Parenting. Emo. Mag. = Emotional Magnification. Mal. Emo. Soc. = Maladaptive Emotional Socialization.

Table 4. Loading Estimates (with Confidence Intervals in Brackets) from Exploratory Factor Analysis

Parenting Measures		EFA 1		EFA 2
		Factor 1	**Factor 2**	Single Factor
Daily Stimulating Interactions		**.45 [.15, .75]**	**(-.06 [-.42, .29])**	.50 [.36, .65]
Learning Activities		**.45 [.16, .74]**	**(<.01 [-.31, .31])**	.46 [.29, .62]
Supporting Positive Behavior		**.67 [.40, .95]**	**(-.14 [-.39, .11])**	.77 [.71, .83]
Emotional Support		**(.25 [-.36, .86])**	**-.41 [-.78, -.04]**	.50 [.36, .64]
Setting Limits		**.90 [.82, .98]**	**(<.01 [-.08, .08])**	.86 [.80, .92]
Proactive Parenting	On	**.87 [.71, 1.03]**	**.13 [.01, .24]**	.76 [.68, .85]
Laxness-Consistent Parenting		**-.46 [-.75, -.16]**	**(.14 [-.13, .41])**	-.55 [-.68, -.42]
Emotional Magnification		**(-.07 [-.85, .71])**	**.64 [.24, 1.04]**	-.45 [-.59, -.30]
Emotional Neglect		**(.20 [-.25, .66])**	**.45 [.21, .67]**	(-.07 [-.24, .11])
Overreactivity		**(-.24 [-.99, .51])**	**.61 [.20, 1.02]**	-.59 [-.74, -.45]
Hostility		**(.08 [-.69, .85])**	**.67 [.32, 1.01]**	-.32 [-.48, -.16]

Note. Bolded label = best-fitting solution. Estimates in parentheses = $p > .05$. EFA 1 = Two-factor solution of our phenotypic exploratory factor analysis. EFA 2 = One-factor solution of our phenotypic exploratory factor analysis. In EFA 2, Factor 1 and Factor 2 are correlated at -.46, $p < .05$.

Multivariate ACE Analyses

Having identified the structure of parenting that best fit the phenotypic data, we fit two multivariate common and specific ACE factors models to examine the distribution of child genetic and environmental influences within this structure of parenting. We first tested the model with independent pathways representing domain-general genetic and environmental influences across the 5 domains of parenting (Model 1; see Figure 3). While the measure-specific (residual) variance of each specific parenting measure was decomposed into the 3 biometric components *A*, *C*, and *E* as described earlier, variance in each broad factor was constrained to be fully explained by 9 components by way of a higher-order independent pathways structure: a general set of *A*, *C*, and *E* factors on which all 5 broad factors loaded (i.e., A_c, C_c, and E_c); separate *A*, *C*, and *E* factors representing either *Positive* or *Negative Parenting* on which its subordinate broad factors loaded (i.e., A_p, C_p, E_p, A_n, C_n, and E_n); and *A*, *C*, and *E* factors specific to each broad parenting factor. Chi-square goodness-of-fit test indicated that Model 1 fit our data well (χ^2 [444, N = 271] = 824.16, $p < .01$, MLR scaling = 1.07, AIC = 10432.13, BIC = 10813.95, RMSEA = .08, CFI = .93, TLI = .92).

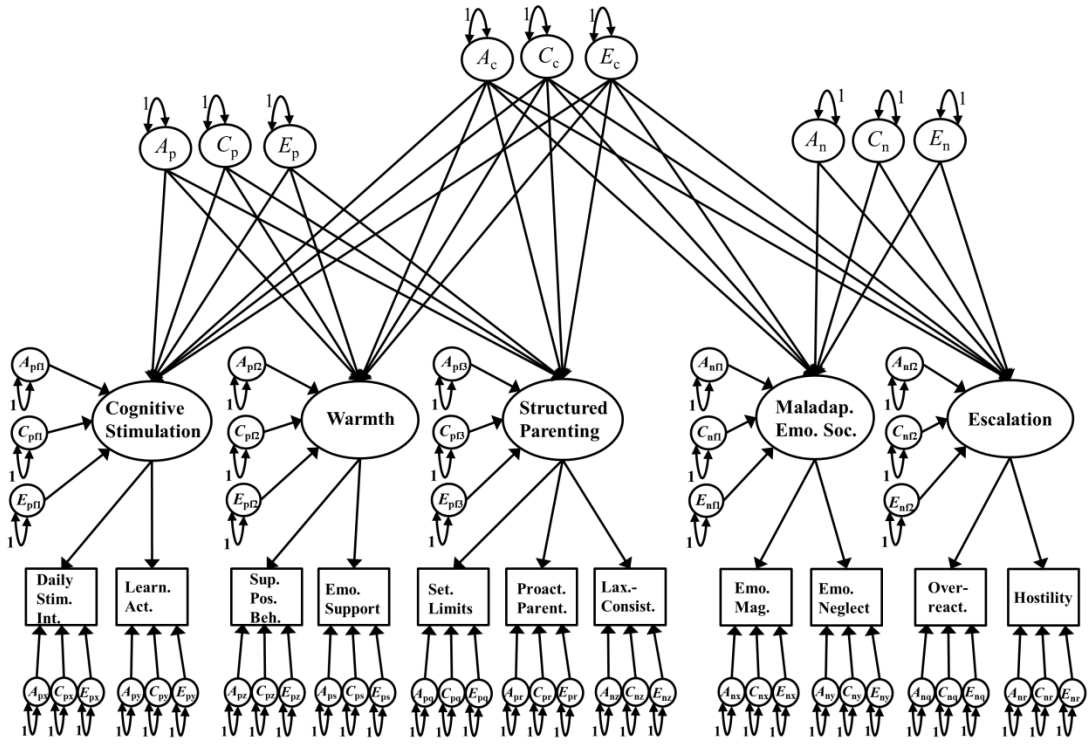

Figure 3. Multivariate independent pathways model (i.e., Model 1). Maladap. Emo. Soc. = Maladaptive Emotional Socialization. Daily Stim. Int. = Daily Stimulating Interactions. Learn. Act. = Learning Activities. Sup. Pos. Beh. = Supporting Positive Behavior. Emo. Support = Emotional Support. Set. Limits = Setting Limits. Proact. Parent. = Proactive Parenting. Lax.-Consist. = Laxness-Consistent Parenting. Emo. Mag. = Emotional Magnification. Emo. Neglect = Emotional Neglect. Overreact. = Overreactivity.

We then fit a common pathways model in which we constrained the influences of A_p, C_p, and E_p to manifest through the higher-order factor *Positive Parenting* and those of A_n, C_n, and E_n to manifest through *Negative Parenting* (Model 2; see Figure 4; χ^2 [462, N = 271] = 822.91, $p < .01$, MLR scaling = 1.08, AIC = 10406.60, BIC = 10723.59, RMSEA = .08, CFI = .93, TLI = .93). Correlations between corresponding *A*s, *C*s, and *E*s

on *Positive Parenting* and *Negative Parenting* were calculated to examine the child genetic and environmental influences common to both factors. Models 1 and 2 fit our data equivalently well ($\Delta\chi^2$ = 7.27, Δdf = 18, p > .05). Model 2, being more parsimonious, is therefore the preferred behavioral genetic model in representing the multivariate structure of the parenting variables. Our results suggest that influences of *A*, *C*, and *E* common to multiple parenting domains are best represented by common pathways. Parameter estimates from the two multivariate ACE models are listed in Tables 5 to 8.

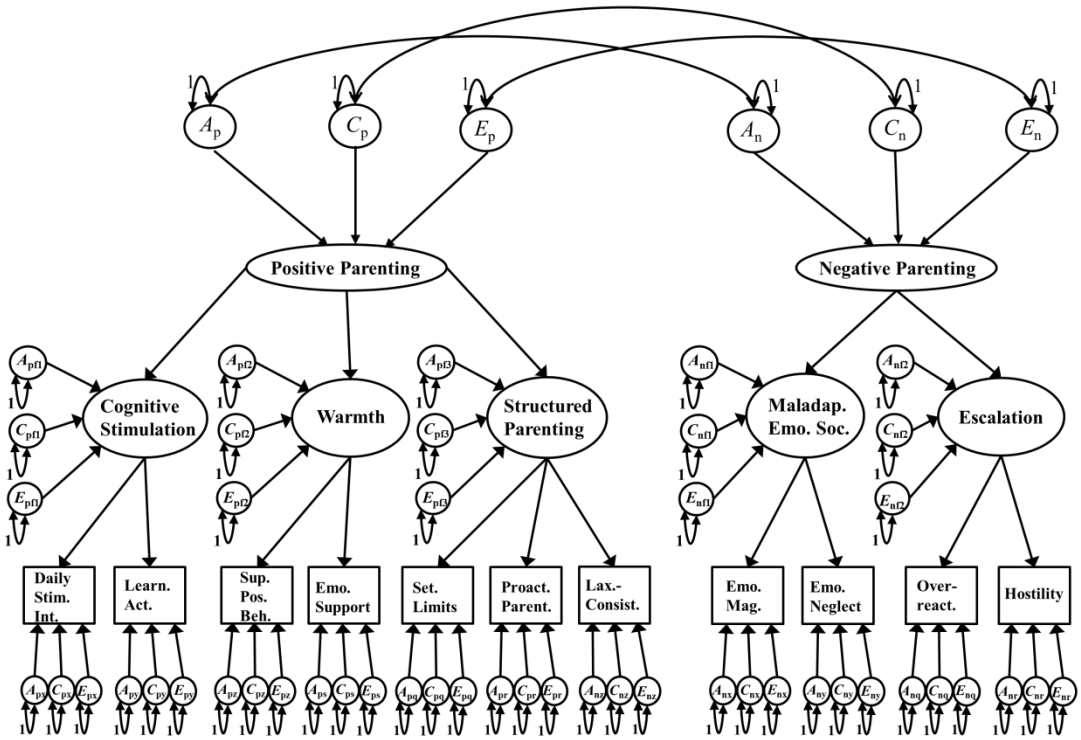

Figure 4. Multivariate common pathways model (i.e., Model 2). Maladap. Emo. Soc. = Maladaptive Emotional Socialization. Daily Stim. Int. = Daily Stimulating Interactions. Learn. Act. = Learning Activities. Sup. Pos. Beh. = Supporting Positive Behavior. Emo. Support = Emotional Support. Set. Limits = Setting Limits. Proact. Parent. = Proactive Parenting. Lax.-Consist. = Laxness-Consistent Parenting. Emo. Mag. = Emotional Magnification. Emo. Neglect = Emotional Neglect. Overreact. = Overreactivity.

Table 5. Standardized Loading Estimates (with Confidence Intervals in Brackets) from Behavioral Genetic Models

Subordinate Measures/Factors		Higher Order Factors	Model 1	**Model 2**
Daily Stimulating Interactions		Cognitive Stimulation	.86 [.59, 1.13]	**.84 [.68, .99]**
Learning Activities			.65 [.32, .97]	**.67 [.49, .85]**
Supporting Positive Behavior		Warmth	.81 [.73, .89]	**.83 [.75, .90]**
Emotional Support			.50 [.37, .64]	**.50 [.36, .64]**
Setting Limits		Structured Parenting	.94 [.89, .1.00]	**.94 [.88, 1.00]**
Proactive Parenting			.80 [.73, .86]	**.80 [.74, .86]**
Laxness-Consistent Parenting			-.51 [-.65, -.36]	**-.51 [-.66, -.37]**
Emotional Magnification	On	Maladaptive Emotional Socialization	.98 [.29, 1.67]	**.91 [.48, 1.33]**
Emotional Neglect			.27 [.04, .49]	**.30 [.13, .48]**
Overreactivity		Escalation	.87 [.74, .99]	**.87 [.75, .99]**
Hostility			.55 [.43, .68]	**.54 [.42, .67]**
Cognitive Stimulation		Positive Parenting	-	**.62 [.43, .80]**
Warmth				**.99 [.92, 1.06]**
Structured Parenting				**.84 [.72, .95]**
Maladaptive Emotional Socialization		Negative Parenting	-	**.67 [.38, .97]**
Escalation				**.94 [.75, 1.13]**

Note. Bolded = best-fitting model. All estimates at $p < .05$. Model 1 = Multivariate independent pathways model. Model 2 = Multivariate common pathways model.

Table 6. Standardized Parameter Estimates of A_c, C_c, E_c, A_p, C_p, E_p, A_n, C_n, and E_n influences (with Confidence Intervals in Brackets)

	Effects of	Model 1	Effects of	Model 1	**Model 2**
Cognitive Stimulation	A_c	(.07 [-.31, .46])	A_p	.21 [.07, .35]	-
Warmth		(-.15 [-1.13, .83])		(.16 [-.06, .38])	
Structured Parenting		(-.07 [-.44, .30])		(.15 [-.17, .46])	
Positive Parenting		-		-	**.20 [.06, .34]**
Cognitive Stimulation	C_c	.59 [.33, .85]	C_p	.78 [.58, .98]	-
Warmth		.96 [.85, 1.07]		(.08 [-.39, .55])	
Structured Parenting		.84 [.70, .97]		(-.06 [-.49, .37])	
Positive Parenting		-		-	**.98 [.95, 1.00]**
Cognitive Stimulation	E_c	(-.03 [-.23, .16])	E_p	(.01 [-.32, .35])	-
Warmth		(.02 [-1.65, 1.68])		(.15 [-.16, .46])	
Structured Parenting		(-.06 [-.81, .70])		(.07 [-.49, .64])	
Positive Parenting		-		-	**.09 [.03, .15]**
Mal. Emo. Soc.	A_c	(.28 [-.19, .75])	A_n	(.22 [-.65, 1.09])	-
Escalation		(-.04 [-.99, .91])		.24 [.10, .38]	
Negative Parenting		-		-	**.29 [.18, .41]**
Mal. Emo. Soc.	C_c	-.43 [-.74, -.12]	C_n	(.46 [-.05, .97])	-
Escalation		-.67 [-.82, -.51]		.55 [.41, .69]	
Negative Parenting		-		-	**.95 [.92, .99]**
Mal. Emo. Soc.	E_c	(-.06 [-1.00, .87])	E_n	.22 [.07, .38]	-
Escalation		(.03 [-.17, .23])		(.05 [-.02, .12])	
Negative Parenting		-		-	**.09 [.02, .15]**

Note. Bolded = best-fitting model. Estimates in parentheses = $p > .05$. Model 1 = Multivariate independent pathways model. Model 2 = Multivariate common pathways model. In Model 2, A_p and A_n were correlated at .66 [-.01, 1.32], C_p and C_n at -.76 [.57, .96], and E_p and E_n at -1.00 [>-1.01, <-.99]. Mal. Emo. Soc. = Maladaptive Emotional Socialization.

Table 7. Standardized Parameter Estimates of Domain-Specific *A*, *C*, and *E* Influences (with Confidence Intervals in Brackets)

Effect of			Model 1	**Model 2**
A	Uniquely On	Cognitive Stimulation	(<.01 [>-.01, <.01])	**.16 [.08, .25]**
		Warmth	(<.01 [>-.01, .01])	**(.10 [-.82, 1.02])**
		Structured Parenting	(.19 [-.10, .48])	**(.19 [-.07, .46])**
		Maladaptive Emotional Socialization	(<.01 [-.01, .01])	**(.14 [-.76, 1.04])**
		Escalation	(<.01 [-.01, .01])	**(<.01 [>-.01, <.01])**
C		Cognitive Stimulation	(-.01 [-.05, .04])	**.77 [.63, .91]**
		Warmth	(<.01 [>-.01, <.01])	**(<.01 [>-.01, <.01])**
		Structured Parenting	.48 [.22, .73]	**.51 [.31, .72]**
		Maladaptive Emotional Socialization	.65 [.04, 1.26]	**.69 [.35, 1.03]**
		Escalation	.43 [.24, .62]	**(.34 [-.18, .86])**
E		Cognitive Stimulation	(<.01 [>-.01, <.01])	**(<.01 [>-.01, <.01])**
		Warmth	(<.01 [>-.01, <.01])	**(.08 [-.35, .51])**
		Structured Parenting	(<.01 [>-.01, <.01])	**(<.01 [>-.01, <.01])**
		Maladaptive Emotional Socialization	(<.01 [>-.01, <.01])	**.23 [.04, .42]**
		Escalation	(<.01 [>-.01, <.01])	**(<.01 [>-.01, <.01])**

Note. Bolded = best-fitting model. Estimates in parentheses = $p > .05$. Model 1 = Multivariate independent pathways model. Model 2 = Multivariate common pathways model.

Table 8. Standardized Parameter Estimates of Measure-Specific *A*, *C*, and *E* influences (with Confidence Intervals in Brackets)

Effect of			Model 1	**Model 2**
A	Uniquely On	Daily Stimulating Interactions	.26 [.11, .41]	**.27 [.16, .39]**
		Learning Activities	(.13 [-.09, .34])	**(.13 [-.07, .32])**
		Supporting Positive Behavior	(.22 [-.24, .68])	**(.21 [-.25, .66])**
		Emotional Support	.39 [.11, .67]	**.40 [.15, .65]**
		Setting Limits	(.18 [-.06, .42])	**(.16 [-.11, .43])**
		Proactive Parenting	.33 [.14, .52]	**.33 [.15, .51]**
		Laxness-Consistent Parenting	(.22 [-.08, .52])	**(.23 [-.06, .51])**
		Emotional Magnification	(.06 [-3.44, 3.33])	**(.26 [-.17, .70])**
		Emotional Neglect	(.32 [-.02, .66])	**(.31 [-.03, .65])**
		Overreactivity	.27 [.07, .46]	**.24 [.05, .44]**
		Hostility	.52 [.34, .70]	**.53 [.35, .71]**
C		Daily Stimulating Interactions	(.42 [-.10, .93])	**.46 [.16, .75]**
		Learning Activities	.73 [.46, 1.00]	**.71 [.55, .88]**
		Supporting Positive Behavior	.44 [.29, .59]	**.41 [.26, .56]**
		Emotional Support	.69 [.54, .83]	**.68 [.54, .83]**
		Setting Limits	(.16 [-.17, .50])	**(.19 [-.09, .47])**
		Proactive Parenting	.47[.34, .61]	**.47 [.34, .60]**
		Laxness-Consistent Parenting	.78 [.67, .88]	**.77 [.67, .88]**
		Emotional Magnification	(.09 [-7.40, 7.57])	**(.25 [-1.27, 1.78])**
		Emotional Neglect	.85 [.75, .94]	**.84 [.74, .93]**
		Overreactivity	.37 [.10, .65]	**.38 [.12, .64]**
		Hostility	.61 [.44, .78]	**.61 [.44, .79]**
E		Daily Stimulating Interactions	.13 [.08, .18]	**.13 [.07, .19]**
		Learning Activities	.17 [.11, .24]	**.17 [.11, .23]**
		Supporting Positive Behavior	.32 [.19, .45]	**.33 [.21, .44]**
		Emotional Support	.35 [.22, .48]	**.36 [.24, .47]**
		Setting Limits	.22 [.15, .30]	**.23 [.16, .30]**
		Proactive Parenting	.17 [.08, .26]	**.17 [.10, .25]**
		Laxness-Consistent Parenting	.30 [.21, .39]	**.29 [.20, .38]**
		Emotional Magnification	(.19 [-.23, .62])	**(.21 [-.02, .44])**
		Emotional Neglect	.34 [.19, .49]	**.33 [.18, .48]**
		Overreactivity	.21 [.09, .32]	**.20 [.12, .28]**
		Hostility	.23 [.14, .31]	**.23 [.14, .31]**

Note. Bolded = best-fitting model. Estimates in parentheses = $p > .05$. Model 1 = Multivariate independent pathways model. Model 2 = Multivariate common pathways model.

Figure 5 illustrates the results from the preferred multivariate ACE model (Model 2). Similar to the CFA results at the phenotypic level, this model indicated that the parenting measures could be categorized into 5 different domains, which formed 2 clusters (see Table 5), namely *Positive Parenting* and *Negative Parenting*. Shared environmental variance was .98 for *Positive Parenting* and .95 for *Negative Parenting*, and the correlation between C_p and C_n was -.76. Orthogonal to the shared environmental factors observed at the broadest level, shared environmental variance unique to *Cognitive Stimulation*, *Structured Parenting*, and *Maladaptive Emotional Socialization* was .77, .51, and .69, respectively. At the measurement level, shared environmental variance unique to a given parenting measure was .38 to .84, except that of *Setting Limits* and *Emotional Magnification* did not reach statistical significance.

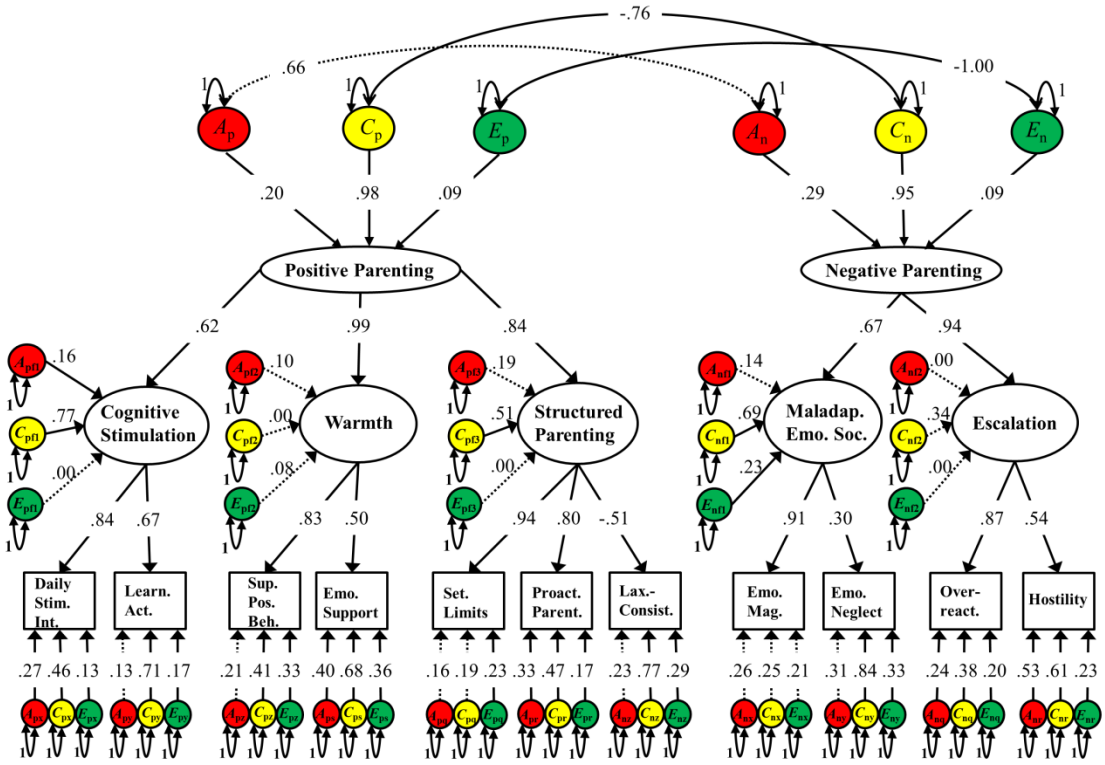

Figure 5. Results from the preferred behavioral genetic model (i.e., Model 2). Solid lines indicate statistically significant paths (i.e., $p < .05$) and dotted lines indicate statistically nonsignificant paths. Maladap. Emo. Soc. = Maladaptive Emotional Socialization. Daily Stim. Int. = Daily Stimulating Interactions. Learn. Act. = Learning Activities. Sup. Pos. Beh. = Supporting Positive Behavior. Emo. Support = Emotional Support. Set. Limits = Setting Limits. Proact. Parent. = Proactive Parenting. Lax.-Consist. = Laxness-Consistent Parenting. Emo. Mag. = Emotional Magnification. Emo. Neglect = Emotional Neglect. Overreact. = Overreactivity.

We also observed some significant, albeit smaller, genetic influences at multiple levels in our multivariate structure of parenting. Genetic variance was .20 for *Positive Parenting* and .29 for *Negative Parenting*. Although marginally significant ($p = .05$), child genetic effects on *Positive Parenting* were correlated with those on *Negative*

Parenting at .66. Additional to the genetic variance observed at the broadest level, we observed a genetic variance of .16 unique to *Cognitive Stimulation*. Above and beyond these child genetic influences observed at the broad factor level, genetic variance unique to a given parenting measure was .27 for *Daily Stimulating Activities*, .40 for *Emotional Support*, .33 for *Proactive Parenting*, .24 for *Overreactivity*, and .53 for *Hostility*.

Similar to child genetic influences, child nonshared environmental influences were also observed at multiple levels in our multivariate structure of parenting. At the broadest level, child nonshared environmental variance was .09 for both *Positive Parenting* and *Negative Parenting*, and these nonshared environmental factors were correlated at -1.00. Orthogonal to the child nonshared environmental influences observed at the broadest level, we observed a nonshared environmental variance of .23 unique to *Maladaptive Emotional Socialization*. Child nonshared environmental variance unique to a given parenting measure was .13 to .36, except that of *Emotional Magnification* did not reach statistical significance. It is important to note again that, as discussed earlier, nonshared environmental factors at the measurement level include measurement error.

DISCUSSION

Behavioral genetic methods have been extensively developed for estimating common and specific genetic effects on multiple phenotypes. Yet, to our knowledge, such methods have not previously been applied to examine the generality or specificity of children's influences on multiple measures of parenting. Although the socialization literature has pointed to a low dimensional structure along which parenting quality affects early child development, child genetic and environmental influences on parenting have not been decomposed along similar dimensions. Our study used a multivariate behavioral

genetic method to examine the loci of child genetic and environmental influences on parenting.

We observed shared environmental influences on both broad and specific dimensions of parenting. In this child-based study, shared environments represent environmental factors that contribute to consistent parenting across children reared in the same family. Therefore, our results suggest that much variation in parenting is attributed to broad contextual factors (i.e., environments in which parents are embedded; e.g., socioeconomic status, cultural background, and family dynamics) and/or parental characteristics (e.g. parent's experience as a child, values, expectations, and personality). Such shared environmental influences were detected at all levels of generality and specificity: ranging from domain-general dimension to the measure-specific dimension. At the broadest level, the strong and negative correlation between shared environmental effects on *Positive Parenting* and those on *Negative Parenting* suggests that a substantial portion of these broad contextual and parental influences exerts contrary effects on these two parenting styles (e.g., promoting parenting behaviors that are thought to positively influence child development while discouraging those that are thought to negatively influence child development). We also uncovered domain-specific shared environmental influences on *Cognitive Stimulation*, *Structured Parenting*, and *Maladaptive Emotional Socialization*, and myriad shared environmental factors at the measurement level. Simply put, some broad contextual and parental characteristics contribute broadly to overall parenting style, whereas others contribute uniquely to particular domains of parenting or specific parenting practices.

In contrast to the large and ubiquitous influences of child shared environmental factors across levels of analysis, child genetic influences were more modest. At the broadest level, our results suggest that caregivers somewhat alter their general parenting

approaches in response to child characteristics driven by genes. Although marginally significant, the moderate and positive correlation between child genetic effects on *Positive Parenting* and those on *Negative Parenting* indicates a trend that gene-based child characteristics evoking parenting approaches that are thought to positively influence child development also evoke parenting approaches that are thought to negatively influence child development. One possibility is that child genetic dispositions such as extroversion may lead to more parent-child interactions, which means more opportunities for both positive and negative interactions to occur. For example, children who always seek attention from their parents may receive positive and warm feedback when the parents are available but negative or harsh feedback when the parents are overwhelmed by other tasks on hand. More research is required to replicate and clarify these child genetic influences common to both positive and negative parenting styles.

We also observed child genetic influences unique to *Cognitive Stimulation*, a factor common to *Daily Stimulating Interactions* and *Learning Activities*. This suggests that a nontrivial portion of variation in the amount of cognitive stimulation parents provide to their young children is attributed to child genetically linked characteristics that are independent from those broadly influencing overall parenting style. It is plausible that parents adjust their supply of cognitive stimulation to children's genetically disposed aptitude and interest (Tucker-Drob & Harden, 2012a; Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011). For example, children with genetic propensities for fast learning may find a diverse range of cognitive stimulation intriguing while those with genetic propensities for slow learning may find it overwhelming. Other genetically driven child characteristics may be involved as well and more research is required to clarify these child genetic influences on amount of cognitive stimulation received.

Measure-specific child genetic influences were detected on *Daily Stimulating Activities*, *Emotional Support*, *Proactive Parenting*, *Overreactivity*, and *Hostility*. The observation that much child genetic influence occurs at the measurement level suggests that child genetic influences on parenting occur via characteristics that cause caregivers to individually modify many different specific parenting practices, above and beyond caregivers' overall parenting style. One noteworthy observation is that, except for those child genetic influences that broadly manifested through *Negative Parenting*, no domain-specific or measure-specific child genetic influences were detected for *Maladaptive Emotional Socialization* or its two indicators. This suggests that the genetically driven child characteristics evoking parenting behaviors that are thought to impede children's development of effective emotional regulatory skills are also the ones evoking parenting behaviors that are thought to intensify adverse parent-child interactions. Our overall findings on child genetic contribution to parenting are consistent with those from previous studies (e.g., Avinun & Knafo, 2013; Boivin et al., 2005; Button et al., 2008; Klahr & Burt, 2014; Knafo & Plomin, 2006; Neiderhiser et al., 2004; Pike et al., 1996; Plomin, Reiss, Hetherington, & Howe, 1994), all of which indicate that parents alter their parenting practices in response to genetically driven child characteristics. Furthermore, results from our multivariate analysis show that these child genetic influences operate both on very broad dimensions of parenting (*Positive Parenting* and *Negative Parenting*) and on specific parenting behaviors. This indicates that the role of child genetic characteristics in evoking differential parenting includes the refinement of more specific aspects of parenting, above and beyond their effects on general parenting styles.

Our results also indicate some non-genetic sources of child-specific variance in parents' behaviors, i.e., nonshared environmental effects. Nonshared environmental influences observed at the measurement level may not be surprising, as measure-specific

nonshared environmental effects may simply reflect measurement error. However, we also detected a nontrivial amount of nonshared environmental effects specifically on *Maladaptive Emotional Socialization* and broadly on both *Positive Parenting* and *Negative Parenting*. Nonshared environmental influences observed at the latent factor level are independent of those operating at the measurement level and are thus free of measurement error. These nonshared environmental effects on parenting may reflect child-specific parenting behaviors conceived by parents for unsystematic or idiosyncratic reasons (Caspi et al., 2004), or they may also reflect parental responses to non-genetic sources of variation in child characteristics such as different interactions with other caregivers. In particular, at the broadest level, the strong and negative correlation between nonshared environmental effects on *Positive Parenting* and those on *Negative Parenting* suggests that nearly all of these non-genetic child characteristics have contrary effects on these two parenting styles (e.g., evoking parenting behaviors that are thought to positively influence child development while suppressing parenting behaviors that are thought to negatively influence child development).

Considering our findings as a whole, parenting in early childhood largely varies at the family level (i.e., across children reared in different families) and thus likely reflects broad contextual and parental characteristics; yet parenting practices also vary, to some extent, across children reared together as a function of child genetic variability. It is important for future research to examine the developmental changes in such patterns. Theories of *r*GE (e.g. Scarr & McCartney, 1983) point to increasing influences of active and evocative processes with age as children are afforded greater freedom in selecting environments that are congruent with their genetic dispositions. One might therefore expect child genetic influences on parenting to increase with child age. However, increasing active and evocative *r*GE could additionally, or even alternatively, result from

children having greater opportunities to select and evoke extra-familial experiences over time (Tucker-Drob, Briley, & Harden, 2013; Tucker-Drob & Harden, 2012b). For instance, as children enter adolescence, they grow more autonomous in relation to their parents but less so in relation to their peers (Steinberg & Silverberg, 1986). From meta-analyzing studies that used samples of different ages, Klahr and Burt (2014) found that child genetic influences on parental warmth were about the same across ages but those on parental negativity decreased with child age, whereas Avinun and Knafo (2013) found more or less consistent child genetic influences on both maternal positivity and negativity across child ages. Additionally, the relation between age and child genetic influences on parenting may not be monotonic, first increasing as children grow more active in evoking specific behaviors from their caregivers, then decreasing as children spend more time in extra-familial settings. Future research should use longitudinal data to examine developmental changes in child genetic and environmental influences on parenting, especially around developmental transitions such as school entry, school transition, and the onset of puberty.

Our study is important in that, in addition to providing support for a transactional association between parenting and early child development, it is among the first to localize child genetic influences within a hierarchical structure of parenting. As suggested in the behavioral genetic literature on parenting (e.g., Avinun & Knafo, 2013; Kendler & Baker, 2007; Klahr & Burt, 2014), certain parenting practices are more susceptible to genetically driven child characteristics than the others. By using a multivariate design, our findings shed lights on the mechanisms underlying the co-occurrence of distinct but correlated parenting practices. Furthermore, our study of children in their first 5 years of life indicates that these transactional influences between parenting and child characteristics potentially begin soon after birth.

Our findings should also be interpreted in light of some limitations. First, the number of twin pairs providing data for this study is relatively small when compared to the typical sample size in modern quantitative genetic research. Nonetheless, the use of latent variable models, in which factor loadings are moderate-to-high, has been shown to mitigate parameter imprecision that is typically associated with smaller sample sizes (MacCallum, Widaman, Zhang, & Hong, 1999; Preacher & MacCallum, 2002). We further boosted parameter precision and statistical power by incorporating longitudinal waves of measurement, while employing estimation methods to correct standard errors for the resulting nonindependence of observations on the same individual across waves. Additionally, instead of mining a large set of pairwise hypothesis tests for those surpassing thresholds for statistical significance, we employed multivariate approaches to model the overall patterns of variation and covariation in the entirety of the data, and we focused on effect sizes rather than significance levels in interpreting these overall patterns.

Second, all our parenting measures are self-reports of parenting practices in the most recent one or two months. Self-report measures are subject to biases such as self-enhancement and social desirability. Nevertheless, participants completed their online surveys in the privacy of their home and were assured that their information would be kept confidential. These arrangements have been shown to reduce social desirability, especially when measuring personal behaviors (Richman, Kiesler, Weisband, & Drasgow, 1999). In the context of twin studies, parents rating both twins in the same pair can also be biased by contrast effects and introduce errors in the genetic and environmental estimates. If contrast effects operate similarly for MZ and DZ twins, this may downwardly bias shared environmental parameter estimates. If, however, contrast effects operate more substantially for DZ than for MZ twins, then genetic influences may

be upwardly biased. Similarly, if MZ twins are more similarly treated by their parents than are DZ twins, simply as a result of their parent's knowledge of their zygosity, genetic influences may be overestimated (this is referred to as a violation of the equal environments assumption). If, on the other hand, similar treatment results from parental responses to their children's genetically influenced characteristics, this should not bias parameter estimates (and not violate the equal environments assumption) – but simply reflect gene-environment correlations. Previous research has found the equal environment assumption to hold true under various situations (Borkenau, Riemann, Angleitner, & Spinath, 2002; Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013; Evans & Martin, 2000; Kendler, Neale, Kessler, Heath, & Eaves, 1993; Morris-Yates, Andrews, Howie, & Henderson, 1990; Scarr & Carter-Saltzman, 1979). Furthermore, parenting measures in our study provided concrete descriptions of scenarios and practices being assessed, which has likely reduced the contrast effects in our study (see Simonoff et al., 1998).

Third, parameter estimates for child genetic and environmental influences on parenting may vary by informants. Meta-analyzing more than 30 child-based studies (Avinun & Knafo, 2013; Klahr & Burt, 2014), parenting reported by parents themselves indicates greater child genetic influences (except for parental control) and smaller nonshared child environmental influences than those based on examiners' observation but similar or smaller child genetic influences and greater shared child environmental influences than parenting reported by children. Different estimates across informants likely stem from the fundamental differences between survey- and observation-based assessments (Avinun & Knafo, 2013; Klahr & Burt, 2014). Because parent- and child-reports focus on general parenting behaviors across times and settings, they likely reflect greater influences of genetically driven child characteristics on parenting; whereas observational data are based on time-limited behaviors specific to the interaction

observed, observational data likely reflect a greater influence of unique experiences (i.e., nonshared environmental influences at the child level). Despite the differences in magnitude, estimates are generally significant and moderate in size across informants (Avinun & Knafo, 2013; Klahr & Burt, 2014). Most importantly, our study focuses on the general distribution of child genetic and environmental influences across broad and specific dimensions of parenting rather than on any parameter estimate for a given factor or measure.

Fourth, it is unclear whether nonshared environmental influences observed are attributed to environmental factors that are unique to a child or to idiosyncratic or arbitrary factors that lead to differential treatment across children by the same parent. Fifth, although we found strong shared environmental effects on parenting, our study design did not allow us to decompose shared child environmental variation in parenting into genetic and environmental variance components associated with the parents themselves. For instance, heritable parental characteristics such as personality, educational attainment, and cognitive ability likely influence the type of behaviors that parents engage in. These parental genetic influences on parenting are included as genetic factors in parent-based designs but shared environmental factors in child-based designs. Comparing results from the two designs can be one way to clarify the extent to which parenting behaviors are attributable to parental versus child genetic factors (Neiderhiser et al., 2004). In particular, a children-of-twins design (D'Onofrio et al., 2003; Narusyte et al., 2008) would allow for simultaneous estimations of parent-driven and child-driven genetic and environmental effects on parenting. Meta-analyzing nine parent-based studies, Klahr and Burt (2014) suggested that parenting is attributed moderately to parents' genetic dispositions and substantially to parents' unique experiences that include

their upbringing, marital relationships, and, as observed in our study, characteristics of their children.

CONCLUSION

To our knowledge, this is the first study to apply multivariate behavioral genetic methods to parenting as an environmental measure at the child level. Our findings suggest that both general and specific parenting practices largely reflect broad contextual and parental characteristics; yet, caregivers also adjust their broad and specific practices, to some extent, in response to genetically influenced characteristics of their children. Our results are consistent with a transactional perspective in which children and parents mutually affect one another.

Chapter 2: From Specialist to Generalist: Developmental Transformations in the Genetic Structure of Early Child Abilities

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In the statistical sense, a strong general factor, *g*, underlies many disparate domains of cognitive functioning at all stages of human development from infancy through old age (Carroll, 2003; Gignac, 2014; Gottfredson, 2002; Jensen, 1998; Spearman, 1914; Tucker-Drob, 2009). The generalist genes perspective holds that this general factor occurs primarily because most genes contributing to one domain of cognitive functioning also contribute to other domains of cognitive functioning (Kovas & Plomin, 2006; Plomin, Kovas, & Haworth, 2007). Consistent with this perspective, genetic correlations between many diverse abilities are moderate to strong in magnitude (Alarcon, Plomin, Fulker, Corley, & DeFries, 1999; Butcher, Kennedy, & Plomin, 2006; Chow, Ho, Wong, Waye, & Bishop, 2013; Luo, Petrill, & Thompson, 1994; Petrill et al., 1998; Petrill, 2002; Petrill, 2005; Plomin & Spinath, 2002; Rice, Carey, Fulker, & DeFries, 1989), a phenomenon referred to as statistical pleiotropy. Moreover, one recent study reported moderate genetic correlations among the brain structures underlying different abilities (Schmitt et al., 2007).

An outstanding question is whether statistical pleiotropy is a developmentally invariant property of the human biological system, or the product of dynamic processes that emerge and strengthen over development. In contrast to the well-documented age-related increase in heritability of cognitive abilities (Briley & Tucker-Drob, 2013;

[2] Drs. Elliot Tucker-Drob and Paige Harden are the principle investigators of the Texas "Tiny" Twin Project, which provides data used in Study 2. Drs. Tucker-Drob and Harden also provided feedback to the first author throughout the development and publication of Study 2.

Haworth et al., 2010), age-related changes in statistical pleiotropy are not well-studied. Two different perspectives have been proposed in the literature to explain the existence of statistical pleiotropy, one of which would predict developmental increases in statistical pleiotropy (i.e., a *transactional* perspective; disproportionately more growth in generalist compared to specialist genetic influences), and the other of which would predict relatively stable associations among genetic influences on different abilities over development (i.e., an *endogenous* perspective; proportional increases in generalist and specialist genetic influences). Using data from a sample of young twins, the current study distinguishes between these competing predictions by investigating age moderation of the multivariate genetic structure of early child abilities.

Mechanisms of Increasing Heritability and Genetic Commonality

The heritability of cognitive abilities increases across development (Bartels, Rietveld, van Baal, & Boomsma, 2002; Boomsma et al., 2002; Briley & Tucker-Drob, 2013; Davis, Haworth, & Plomin, 2009; Haworth et al., 2010; Tucker-Drob, Briley, & Harden, 2013). Analyzing cross-sectional data on 11,000 pairs of twins from four different countries, Haworth and colleagues observed that heritability of general cognitive ability increased from 41% in childhood to 66% in young adulthood. Briley and Tucker-Drob meta-analyzed 16 genetically informative longitudinal studies, totaling 11,500 sibling pairs of ages 6 months to 18 years, and confirmed that the heritability of cognitive abilities increases over development. Importantly, the heritability of a particular ability represents the combined effects of both general genetic factors, which also contribute to variation in other abilities, and specific genetic factors, which contribute uniquely to variation in that specific ability. Changes in the heritability of a given ability over development may result from changes in general genetic factors, specific genetic

factors, or some combinations of the two. Here, we further describe two general classes of mechanisms that lead to different predictions about the pattern in which generalist and specialist genetic influences on abilities change with age.

Transactional Perspective

One way that statistical pleiotropy may emerge is through the multiplier effects of different abilities on one another. In their mutualism model, van der Maas et al. (2006) proposed that causation between biologically independent abilities may contribute to their intercorrelations. Under the mutualism model, reciprocal causation between different abilities leads to the emergence and strengthening of shared genetic variance over time. van der Maas and colleagues suggested that genetic correlations across different abilities may be weak or negligibly small very early in development; as development progresses, reciprocal processes result in increasing statistical pleiotropy. Similarly, Dickens (2007) proposed that the dynamic association between abilities and environments can result in the emergence and strengthening of statistical pleiotropy. If an individual has a particular advantage (or disadvantage) in a specific ability, this might prompt exposures to environments that broadly facilitate (or impede) the development of other abilities. For example, a child who has high verbal ability may be identified by parents and teachers as "smart" and consequently tracked into more challenging coursework both in reading and in math. The Dickens model predicts that the early genetically influenced individual differences in a specific ability result in evocation and active selection of environmental experiences relevant for the development of multiple abilities. As ability-environment dynamics accumulate, genetic correlations among different abilities are expected to strengthen.

Endogenous Perspective

What might be termed an endogenous perspective holds that pleiotropic genetic variation results from individual genes that play multiple roles in biological and psychological functions. This can occur, for instance, when a gene codes for multiple proteins each of which serves as a physiological basis for a different ability, or when a gene codes for a single protein that is important for multiple physiological functions, each of which supports a different ability (see Kovas & Plomin, 2006 and Plomin & Spinath, 2002). Importantly, this endogenous perspective holds that statistical pleiotropy is an inherent property of the human biological systems that subserve cognition and behavior. The associations between different domains of functioning are therefore predicted to remain more or less the same across ages. In other words, all else being equal, no developmental changes in genetic correlations among different abilities are expected. Although the magnitude of overall genetic influence may grow with age, the extent to which genetic factors are generalist compared to specialist is not expected to change. This perspective resembles that of Juan-Espinosa et al. (2002), who wrote "basic structure does not change at all, although, like the human bones, the cognitive abilities grow up and decline at different periods of life" (p. 406). Gignac (2014) further speculated that perhaps "the reason the strength of the *g* factor is largely invariant across age is because it is mediated substantially by biological characteristics" (p. 96). Based on this endogenous perspective, genetic commonality is expected to remain relatively constant across development.

Developmental Increase in Generalist Genes and Total Heritability

Importantly, total heritability of a phenotype is independent of its genetic commonality with other phenotypes. Two abilities that are highly heritable could share no genetic variance with one another (or they could share all genetic variance with one

another). Moreover, two abilities that are only modestly heritable could share all of their genetic variance with one another (or they could share none of it). It is possible, however, that developmental changes in genetic commonality emerge simultaneously with developmental increase in heritability. In other words, increasing heritability could occur largely via increases in generalist genetic variance. The top panel of Figure 6 illustrates this scenario. Each circle represents the total heritability of a given phenotype at a given developmental period. Not only does the circle grow in size across development, indicating increasing total heritability, but the proportion representing common genetic influences also grows across development indicating increasing genetic commonality. Thus, in this scenario, both total heritability and genetic structure change across development.

Alternatively, increasing heritability could occur via proportional increases in both common and unique genetic variance. The bottom panel of Figure 6 illustrates this scenario. As in the top panel, the size of each circle represents the total heritability of a given phenotype at a given developmental period. While the size of circle increases across development, indicating increasing total heritability, the area representing common genetic influences takes up a similar proportion of the circle across development. Thus, in this scenario, total heritability increases but genetic overlap across phenotypes (or the genetic structure of each phenotype) remains comparable across development.

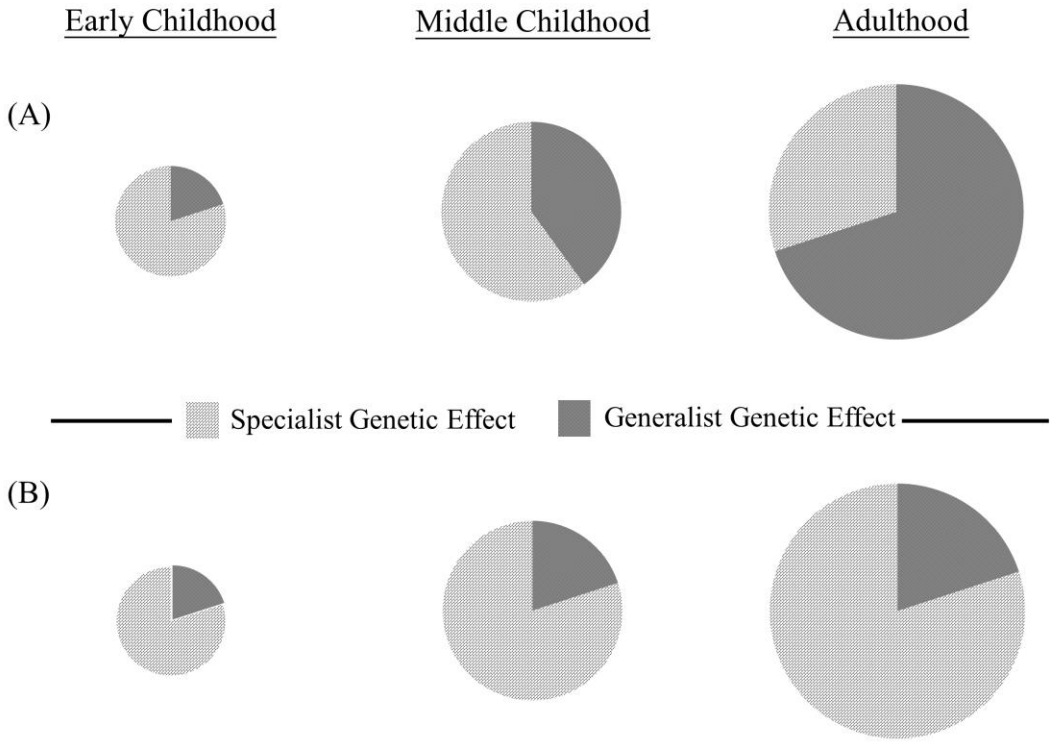

Figure 6. Two hypothetical scenarios for developmental changes in domain-general (generalist) and domain-specific (specialist) genetic effects on domains of functioning. The size of each circle represents total heritability. Top panel: The structure of genetic effects changes with age, with an increasing proportion of genetic effects occurring at the domain-general level. Bottom panel: The structure of genetic effects is age-invariant, with constant proportions of domain-general and domain-specific genetic effects across development.

Previous Evidence for Developmental Transformations in Genetic Commonality

Dynamic transactions between phenotypes, genes, and environments begin early in development (e.g., Tucker-Drob et al., 2013). During infancy and early childhood, average levels of abilities – and their longitudinal stability – dramatically increase (Tucker-Drob & Briley, 2014). Researchers, however, have rarely examined the structure

of genetic and environmental influences on early abilities among children of very young ages. One possible reason is that conventional measures of early infant abilities tend to be unidimensional (e.g., Cherny et al., 1994). Exceptions include Petrill, Saudino, Wilkerson, and Plomin (2001) who, in a sample of 2-year-olds, observed a heritability of .26 and a nonshared environmental influence of .06 for the *g* factor (molarity) and a heritability of .03-.32 and a nonshared environmental influence of .26-.56 unique to each of the subordinate tasks (modularity). The authors speculated that findings of both common and specific genetic effects "suggest a developmental trend from modularity to molarity when considered in relation to multivariate genetic results later in life that show that genetic effects on cognitive abilities contribute primarily to molarity rather than modularity" (p. 31). Based on an earlier analysis of the same data, which found that the correlation between genetic factors of verbal and nonverbal abilities was a modest .30 at age 2 years, Price, Eley et al. (2000) speculated that "genetic effects on cognitive abilities are modular early in development and then become increasingly molar" (p. 948). Indeed, in a more recent paper (Trzaskowski, Shakeshaft, & Plomin, 2013) that made use of ages 7 years and 12 years data from later longitudinal assessments of what appears to have been the same sample, biometric twin models revealed genetic correlations between approximately .60 and .70. Similarly, in a sample of children aged 4 years from the Colorado Adoption Project, Rice et al. (1989) observed moderately high positive genetic correlations between verbal, spatial, perceptual speed, and visual memory abilities that range from .56 to .89. Piecing together, these snapshots of different age groups, statistical pleiotropy might emerge and strengthen during the first few years of life, as would be predicted by a transactional perspective.

Current Study

The current study used multivariate data on early child abilities from an age-heterogeneous sample of young twins (ages 0–6 years) to test for transformations in the genetic and environmental structure of abilities with age. We applied models that capitalize on the known differences in genetic relationships between monozygotic twins (who share 100% of their genes) and dizygotic twins (who, on average, share 50% of their segregating genes), combined with the knowledge that both members of each twin pair (regardless of zygosity) have been reared together in the same home, to partition variation in both domain-general and domain-specific ability factors into additive genetic (*A*), shared environmental (*C*), and nonshared environmental (*E*) components. We then tested the extent to which each of these variance components differs with age.

METHODS

Participants

Data were collected as a downward extension of the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013) to families with twins or multiples aged 0–6 years who lived in the state of Texas. Qualifying families were identified both from birth records provided by the Texas Department of State Health Services and from community outreach. Community outreach efforts included attending annual conventions of Texas Mothers of Multiples, sending recruitment information to associated email list serves, and enrolling families who registered via the Texas Twin Project website. Most participating families completed surveys managed and stored on Research Electronic Data Capture (REDCap; Harris et al., 2009). Depending on a family's preference, an online or paper survey was sent immediately after the family enrolled in the study. Paper and online administrations of the measurement we employed (the Ages and Stages Questionnaire,

see below) have been found to have equivalent psychometric properties (Squires, Twombly, Bricker, & Potter, 2009). After a family completed the survey for the initial wave, follow-up surveys were sent every 2 months for children from birth until 2 years old, every 3 months for children between 2 and 3 years old, every 5 months for children between 3 and 5 years old, and one last survey for children between 5 and 6 years old. Data collection remains on-going.

For the current study, data were available from 296 individual twins and multiples. Among this sample, a pair of twins was diagnosed with Fragile X syndrome while another pair of twins showed substantially more advanced gross motor development but substantially delayed overall development when compared to the rest of the sample. Results were similar across analyses with and without data from these two twin pairs included. Here, we reported findings from analyses excluding these observations (i.e., based on a sample of 292 individual twins). The sample was 75.34% Caucasian, 4.11% Latino or Hispanic, 2.05% African-American, and 13.70% multi-racial. Less than 1% of these twin families reported having completed only high school, 7.53% reported having some college education, 36.99% reported having completed college, and 54.79% reported having completed education beyond college.

Zygosity for same-sex twins was determined from physical similarity ratings (e.g., hair structure, eye color, and shape of ear lobe, etc.). Primary caregiver of each twin pair rated four items on a 3-point Likert scale ranging from *Not Alike* to *Exactly Alike* and eight other items on a dichotomous scale. Zygosity assignment using physical similarity ratings is highly reliable and corresponds strongly with assignments based on DNA genotyping (Forget-Dubois et al., 2003; Heath et al., 2003; Price, Freeman et al., 2000; Rietveld et al., 2000). Following Harden, Kretsch, Tackett, and Tucker-Drob (2014), we conducted a two-class Latent Class Analysis (LCA) on all 12 items to determine each

same-sex twin pair's zygosity (opposite-sex twins are necessarily dizygotic). This resulted in the sample of 60 monozygotic twins (30 male and 30 female individual twins), 132 same-sex dizygotic twins (58 male and 74 female individual twins), and 100 opposite-sex dizygotic twins (50 male and 50 female individual twins). Sensitivity analyses indicated that models that excluded data from opposite-sex twins produced parameter estimates that were very similar to those in which data from opposite-sex twins were included. We, therefore, reported results from analyses of data from both same-sex and opposite-sex twins, in order to maximize our sample size, and, hence, the precision of our estimates.

In addition to data provided at the initial wave, most families in this sample provided data at one or more follow-up waves. Thus, data were available for up to nine different waves per family. To maximize the pool of observations available for our age-comparative analyses, we used all available data from both baseline and follow-up waves in conjunction with the Complex Survey option in *Mplus* statistical software (Muthén & Muthén, 2010) to account for the nonindependence of longitudinal repeated measurements from the same families across different survey waves. That is, we treated observations on the same twin from different waves as different lines of data and corrected the standard errors of model estimates for biases that could have otherwise potentially resulted from nonindependence of data obtained on the same individuals over time. This resulted in a total of 578 observations on 292 individual children—122 observations from monozygotic twins and 456 observations from dizygotic twins. The average age at measurement among these 578 observations was 2.45 years old (SD = 1.24 years).

Measures

Ages and Stages Questionnaire, Third Edition (ASQ)

The ASQ (Squires & Bricker, 2009) is a multidimensional measure of the occurrence of developmental milestones related to various domains of cognitive and psychomotor functioning. It was standardized on a sample of 12,695 individuals representative of the U.S. young children population on various dimensions, including sex, ethnicity, and various socioeconomic indices (Squires et al., 2009). The ASQ has been shown to accurately reflect young children's progress in attaining developmental milestones in different domains (i.e., high levels of sensitivity and specificity – 86% on average – across ages 2-60 months) and agree 86% on average with standardized developmental assessment based on observational tasks (Squires et al., 2009). A number of additional independent studies have also reported high levels of convergent validity of the ASQ with standardized researcher/clinician-administered measures, such as the Bayley Scales of Infant Development (Bayley; Gollenberg, Lynch, Jackson, McGuinness, & Msall, 2010; Schonhaut, Armijo, Schönstedt, Alvarez, & Cordero, 2013; Simard, Luu, & Gosselin, 2012; Yu et al., 2007). In comparison to many questionnaires that query the raters' intuitive judgments on the development of a child relative to other children of the same age, the ASQ minimizes rater bias by querying about the child's performance on concrete tasks. Primary caregivers rated each of their twins' performance on these concrete tasks on a 3-point Likert scale for five domains: *Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social*. Table 9 defines these domains and gives sample items. These five domains encompass the neurocognitive, psychosocial, and motor milestones used routinely in clinical settings as indicators of young children's physical, psychological, and neurological development (Council on Children With Disabilities, Section on Developmental Behavioral Pediatrics, Bright Futures Steering

Committee, & Medical Home Initiatives for Children with Special Needs Project Advisory Committee, 2006). Delays in reaching these developmental milestones may suggest early functional impairment, and may have cascading effects on later psychological development and real-world functioning across the lifespan (Murray, Jones, Kuh, & Richards, 2007; Sørensen et al., 2010; Taanila, Murray, Jokelainen, Isohanni, & Rantakallio, 2007; van Os, Jones, Lewis, Wadsworth, & Murray, 1997).

All items in ASQ are age-appropriate, meaning that twins at different ages are rated on different sets of items (Squires et al., 2009). Each domain contains 5–10 items depending on the age of the twins. Items from adjacent age-ranges (both above and below) at each age were administered in order to avoid floor and ceiling effects, and to allow the use of vertical scaling to capture children's age-related growth in each domain of development. For each domain of development, a minimum of three items were set to overlap in content for adjacent item sets. Domain scores were obtained from Rasch Item Response Theory (1PL IRT) analyses with higher scores indicating more advanced development. IRT-estimated item reliabilities (item commonalities) for *Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social* were .92, .89, .87, .85, and .82, respectively. Scaling these item-reliabilities using the Spearman-Brown prophecy formula for a 5-item composite measures (the minimum number of items administered in a given domain for a given age) yields scale reliabilities of .98, .98, .97, .97, and .96 for the five ASQ domains, respectively.

Table 9. Definition and Sample Items for Each ASQ Domain

Domain	Definition	Sample Items
Communication	Effective expression of thoughts and processing of information or instructions	*Does your child correctly use at least two words like 'me,' 'I,' 'mine,' and 'you?'* – for children aged 19 to 28.49 months
		Without giving your child help by pointing or using gestures, ask him/her to 'put the book on the table' and 'put the shoe under the chair.' Does your child carry out both of these directions correctly? – for children aged 25.5 to 44.99 months
Gross Motor	Motor development that involves large muscle groups and whole body movement	*Without holding onto anything for support, does your child kick a ball by swinging his/her leg forward?* – for children aged 21 to 38.99 months
		Does your child climb the rungs of a ladder of a playground slide or slide down without help? – for children aged 39 to 50.99 months
Fine Motor	Coordination of small muscle movements that occur in body parts such as fingers	*When you put a toy in his/her hand, does your baby hold it in his/her hand briefly?* – for children aged 1 to 2.99 months
		Does your child unbutton one or more buttons? – for children aged 39 to 56.99 months
Problem-Solving	Ability to use generic rules or logic and find solutions to problems	*Does your child finish the following sentences using a word that means the opposite of the word that is italicized? For example: 'A rock is hard, and a pillow is soft.'* – for children aged 57 to 71.99 months
		When [shown 3 circles of different sizes and] asked, 'which circle is the smallest?' does your child point to the smallest circle? – for children aged 39 to 71.99 months
Personal-Social	Self-care ability and basic skills that prepare them for successful social interactions	*Does your child wash his/her hands using soap and water and dry off with a towel without help?* – for children aged 39 to 56.99 months
		Does your baby smile at you? – for children aged 1 to 2.99 months

RESULTS

Descriptive statistics of domain scores obtained from the 1PL IRT analyses and the correlations between these domain scores and age are listed in Table 10. Regression analysis was conducted to account for the linear and quadratic influences of age on each domain of early child abilities (see Figure 7 for age trends of all five ASQ domains). Resulting residuals were z-standardized for all analyses reported below. All results presented below are based on these age-corrected standardized residuals. Correlations between domain scores, corrected for both the linear and quadratic influences of age, are also listed in Table 10. Consistent with past literature, we observed a positive manifold of correlations among the five ability domains. As reported below, structural equation models were fit using *Mplus* statistical software (Muthén & Muthén, 2010) to investigate age differences in the multivariate structure of early child abilities at both phenotypic and behavioral genetic levels.

Table 10. Descriptive Statistics and Correlations between Domains of Early Child Abilities

	M (*SD*)	Cor-relation with Age	Age- and Age^2-corrected Correlations					
			Cross-domain Correlations				Intra-class Correlations	
			Comm-unication	Gross Motor	Fine Motor	Problem-Solving	MZ	DZ
Comm-unication	17.31 (5.97)	**.91**	-				**.91**	**.69**
Gross Motor	15.07 (5.12)	**.89**	**.33**	-			**.83**	**.46**
Fine Motor	13.81 (4.66)	**.90**	**.43**	**.43**	-		**.77**	**.63**
Problem-Solving	12.96 (4.29)	**.89**	**.43**	**.36**	**.41**	-	**.86**	**.60**
Personal-Social	10.92 (3.88)	**.93**	**.42**	**.40**	**.46**	**.41**	**.90**	**.70**

Note. Bolded = $p < .01$.

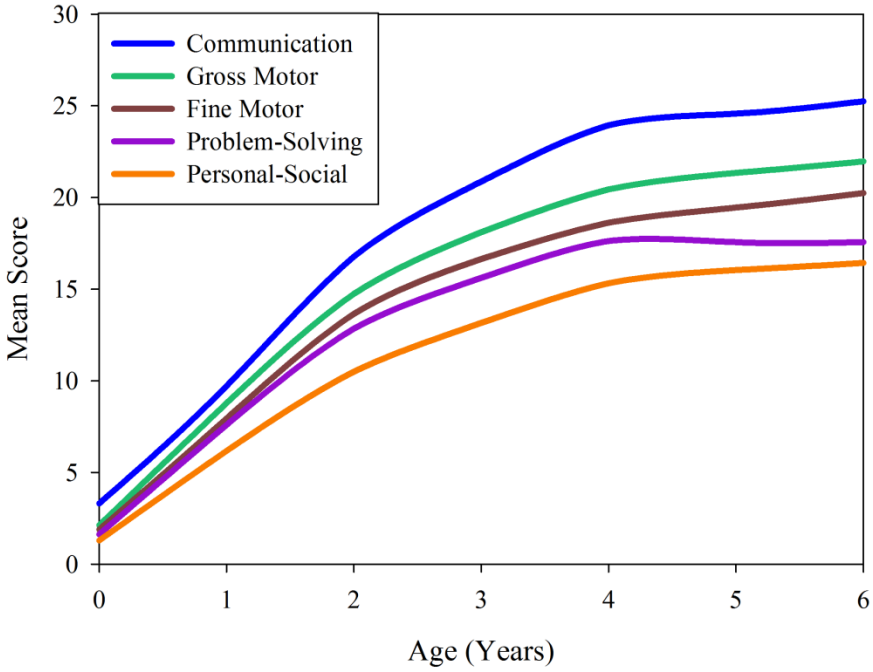

Figure 7. Age trends of the five domains of early child abilities. Mean score for a given domain at a given age is the average of raw domain scores for that age group.

Phenotypic Models

We began by examining whether the phenotypic structure of the five domains of early child abilities varies across ages. We specified a multivariate model in which the loadings of each ability on both the common factor and the ability-specific unique factor was moderated by age. Following Tucker-Drob (2009), this model is written as

$$G[x]_n = \upsilon[x] + \alpha[x] \times age_n + (\lambda_1[x] + \lambda_1'[x] \times age_n) \times g_n + (\lambda_2[x] + \lambda_2'[x] \times age_n) \times u[x]_n$$

In the above equation, [*x*] indicates the domain of early child abilities to which a term corresponds. For example, *G*[*x*] represents the score on a given domain of early child abilities (i.e., *G*[*Communication*], *G*[*Gross Motor*], *G*[*Fine Motor*], *G*[*Problem-Solving*], and *G*[*Personal-Social*]). Each score is determined by a combination of factors: υ[*x*] represents the regression intercept for a given domain of early child abilities, α[*x*] represents the main effect of age on a given domain of early child abilities (freely estimated, but expected to be 0, given that each ability was residualized for age prior to analyses), *g* represents the common latent factor *Broad Ability*, $\lambda_1[x]$ represents the main effect of *Broad Ability* on a given domain of early child abilities, $\lambda_1'[x]$ represents the interaction between age and *Broad Ability* on a given domain of early child abilities, $\lambda_2[x]$ represents the main effect of the ability-specific factor on a given domain of early child abilities, $\lambda_2'[x]$ represents the interaction between age and the ability-specific factor on a given domain of early child abilities, and u[*x*] represents the ability-specific (residual) factor in a given domain of early child abilities. The subscript *n* signifies terms that are allowed to vary by individuals. The latent variables *g* and u[*x*] were scaled to a z-metric ($M = 0$, $SD = 1$).

As delineated in the above equation, in this phenotypic model, the effects of *Broad Ability* and ability-specific (residual) factors were each modeled as the sum of (1) its main effect on the corresponding domain (i.e., $\lambda_1[x]$ and $\lambda_2[x]$), and (2) its interaction with age (i.e., $\lambda_1'[x]$ and $\lambda_2'[x]$). If *Broad Ability* factor loading of any domain varies as a function of age (i.e., if $\lambda_1'[x]$ is statistically significant), this implies age differences in the general concept of early child abilities; any age differences in factor loadings observed from this phenotypic model would inform the subsequently conducted behavioral genetic analyses. Alternatively, if *Broad Ability* factor loadings do not vary as a function of age in the phenotypic model (i.e., $\lambda_1'[x]$ is statistically nonsignificant), this suggests

measurement invariance and factor loading of each domain on the latent factor *Broad Ability* would then be specified to be invariant across ages in our behavioral genetic models. Increasing unique variance as a function of age (i.e., $\lambda_2'[x]$ is greater than zero at a statistically significant level) implies age-differentiation of early child abilities. That is, depending on the sign of the interaction coefficient $\lambda_2'[x]$, domains of early child abilities may become more (or less) distinct from each other as children age.

To elucidate age influences on the phenotypic structure of early child abilities, we fit four versions of the phenotypic multivariate model and compared their model fit statistics to identify the best-fitting model. We first fit a model in which all the interaction coefficients (i.e., $\lambda_1'[x]$ and $\lambda_2'[x]$) were freely estimated. Second, to increase the model's statistical power in detecting age differences, we constrained the interaction coefficient to be proportional to the corresponding main effect in each regression path (i.e., $\lambda_1'[x] = \lambda_1[x] \times \lambda_1'$ and $\lambda_2'[x] = \lambda_2[x] \times \lambda_2'$, where λ_1' and λ_2' are invariant across domains). Third, we constrained the interaction coefficients to be the same across domains of early child abilities at both the factor and residual levels (i.e., $\lambda_1'[x] = \lambda_1'$ and $\lambda_2'[x] = \lambda_2'$, where λ_1' and λ_2' are invariant across domains). Finally, we fixed all the interaction coefficients at zero (i.e., $\lambda_1'[x] = \lambda_2'[x] = 0$).

Table 11 lists the model fit statistics of all the phenotypic models. The model with no interactions fit the data no worse than the more complex models, and this model was therefore preferred. Table 12 lists the parameter estimates from all the phenotypic models. Results from the preferred model (i.e., the model in which all the interaction coefficients were fixed at zero) indicate that *Broad Ability* accounts for 34-48% (e.g., for *Gross Motor*, $.59^2 / [.59^2 + .83^2] \times 100\%$) of the variance in each ability at all ages, with the remaining variance being unique to that ability.

Table 11. Phenotypic Model Fit Statistics

Model		AIC	BIC	LL	*df*	MLR scaling	χ^2 for Model Com-parison	Δdf
1	Freely estimated	8773.99	8916.10	-4354.00	33	2.01	-	-
2	Proportional	8782.49	8890.14	-4266.24	25	2.23	-132.72	8
3	Same across domains	8782.27	8889.93	-4366.14	25	2.22	0.21[i]	0
4	**Fixed at zero**	**8779.92**	**8878.97**	**-4366.96**	**23**	2.23	0.78	2

Note. Bolded = preferred model. χ^2 for model comparison was calculated by comparing the nested model with the previously listed comparison model. 1 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient independently and freely estimated. 2 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be proportional to the main effect of the latent factor on the corresponding observed domain of development. 3 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be the same across domains of development. 4 = Phenotypic confirmatory factor analysis with all age-related interaction coefficients fixed at zero.

[i] difference in BIC is calculated to compare model fitness across Models 2 and 3 as the χ^2 values are equivalent and the degrees of freedom are the same across the two models.

Table 12. Parameter Estimates (with Confidence Intervals in Brackets) from Phenotypic Confirmatory Factor Analyses

Measures		Latent Factor	1		2		3		4[i]
			Main Effect	Inter-action	Main Effect	Interaction	Main Effect	Inter-action	Main Effect
Communication	On	Broad Ability	**.65**[.36, .93]	-.02[-.11, .07]	**.65**[.42, .88]	-.03 [-.11, .05] × corresponding main effect estimate for each domain	**.65**[.43, .87]	-.02 [-.08, .03]	**.60**[.46, .73]
Gross Motor			**.49**[.19, .76]	.05[-.05, .14]	**.64**[.44, .84]		**.65**[.43, .87]		**.59**[.46, .72]
Fine Motor			**.69**[.48, .91]	-.01[-.08, .07]	**.73**[.53, .94]		**.73**[.53, .94]		**.68**[.56, .79]
Problem-Solving			**.75**[.48, 1.02]	-.06[-.14, .03]	**.66**[.41, .91]		**.66**[.44, .88]		**.61**[.47, .74]
Personal-Social			**.78**[.51, 1.04]	-.06[-.15, .04]	**.70**[.47, .92]		**.70**[.48, .91]		**.64**[.52, .77]
Communication		Unique Variance for each domain	**.82**[.66, .98]	-.03[.36, .93]	**.74**[.64, .84]	<.01 [-.04, .04] × corresponding main effect estimate for each domain	**.74**[.64, .83]	<.01 [-.03, .03]	**.74**[.66, .81]
Gross Motor			**.88**[.71, 1.05]	-.03[-.09, .02]	**.83**[.73, .93]		**.83**[.73, .92]		**.83**[.76, .90]
Fine Motor			**.58**[.42, .73]	.05[-.01, .11]	**.71**[.61, .81]		**.71**[.60, .82]		**.71**[.64, .78]
Problem-Solving			**.86**[.68, 1.04]	-.04[-.10, .02]	**.76**[.65, .87]		**.76**[.66, .87]		**.77**[.69, .84]
Personal-Social			**.64**[.47, .81]	.04[-.03, .10]	**.72**[.62, .82]		**.72**[.62, .82]		**.72**[.65, .80]

Note. Bolded = $p < .01$. 1 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient independently and freely estimated. 2 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be proportional to the main effect of the latent factor on the corresponding observed domain of development. 3 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be the same across domains of development. 4 = Phenotypic confirmatory factor analysis with all age-related interaction coefficients fixed at zero.
[i] preferred model.

Behavioral Genetic Models

Next, we fit a multivariate common pathways model to examine age differences in domain-general and domain-specific genetic and environmental influences on early child abilities. This model is written as

$$
\begin{aligned}
G[x]_n = \; & \upsilon[x] + \alpha[x] \times age_n + (a_c + a_c' \times age_n) \times (\lambda[x] \times g_n) \times A_{cn} \\
& + (c_c + c_c' \times age_n) \times (\lambda[x] \times g_n) \times C_{cn} \\
& + (e_c + e_c' \times age_n) \times (\lambda[x] \times g_n) \times E_{cn} \\
& + (a_u[x] + a_u'[x] \times age_n) \times A_u[x]_n + (c_u[x] + c_u'[x] \times age_n) \times C_u[x]_n \\
& + (e_u[x] + e_u'[x] \times age_n) \times E_u[x]_n
\end{aligned}
$$

In this behavioral genetic model, the factor loading of each domain on the latent factor *Broad Ability* was constrained to be age-invariant (as measurement invariance was observed from the preferred phenotypic model). Variance of *Broad Ability* and unique variance of each domain were each divided into three biometric components: genes, with A_c representing common (domain-general) genetic factors and A_u representing unique (domain-specific) genetic factors; shared environmental factors that made the twins more similar to each other, with C_c representing domain-general shared environmental factors and C_u representing domain-specific shared environmental factors; and nonshared environmental factors that are unique to each child and made the twins less similar to each other, with E_c representing domain-general nonshared environmental factors and E_u representing domain-specific nonshared environmental factors. E_u, at the measurement level, also includes measurement error.

In the equation for this behavioral genetic model, each score is determined by a combination of factors: $\upsilon[x]$ represents the regression intercept for a given domain of early child abilities, $\alpha[x]$ represents the main effect of age on a given domain of early child abilities (freely estimated, but expected to be 0, given that each ability was residualized for age prior to analyses), $\lambda[x]$ represents the main effect of *Broad Ability* on a given domain of early child abilities, g represents the common latent factor *Broad Ability*, a_c represents the main effect of domain-general genetic factors (A_c), a_c' represents the interaction between age and A_c, c_c represents the main effect of domain-general shared environmental factors (C_c), c_c' represents the interaction between age and C_c, e_c represents the main effect of domain-general nonshared environmental factors (E_c), e_c' represents the interaction between age and E_c, $a_u[x]$ represents the main effect of genetic factors unique to a given domain of early child abilities ($A_u[x]$), $a_u'[x]$ represents the interaction between age and $A_u[x]$, $c_u[x]$ represents the main effect of shared environmental factors unique to a given domain of early child abilities ($C_u[x]$), $c_u'[x]$ represents the interaction between age and $C_u[x]$, $e_u[x]$ represents the main effect of nonshared environmental factors unique to a given domain of early child abilities ($E_u[x]$), and $e_u'[x]$ represents the interaction between age and $E_u[x]$. The subscript n signifies terms that are allowed to vary by individuals. The latent variables g, A_c, C_c, E_c, $A_u[x]$, $C_u[x]$, and $E_u[x]$ were scaled to a z-metric ($M = 0$, $SD = 1$).

As seen in the equation for the multivariate behavioral genetic model, each path representing genetic or environmental influences is a sum of (1) the main effect of that genetic or environmental factor (i.e., a_c, c_c, e_c, $a_u[x]$, $c_u[x]$, and $e_u[x]$), and (2) its interaction with age (i.e., a_c', c_c', e_c', $a_u'[x]$, $c_u'[x]$, and $e_u'[x]$). At the latent factor level, if genetic influences on *Broad Ability* increase as a function of age (i.e., a_c' is greater than zero at a statistically significant level), this suggests that genetic commonality in early

child development grows with age. If any of the domain-specific genetic influences increases as a function of age (i.e., $a_u'[x]$ is greater than zero at a statistically significant level), this suggests that the importance of specialist genes in early child development grows with age. Note that age differences can occur exclusively at the broad factor level, the measurement level, or co-occur at both levels.

Similar to analyses conducted at the phenotypic level, we fit five versions of multivariate common pathways model and compared their model fit statistics to identify the best-fitting model. First, we fit a model in which all the interaction coefficients (i.e., a_c', c_c', e_c', $a_u'[x]$, $c_u'[x]$, and $e_u'[x]$) were freely estimated. Second, we constrained the interaction coefficient to be proportional to the corresponding main effect in each regression path at the measurement level (i.e., $a_u'[x] = a_u[x] \times a_u'$, $c_u'[x] = c_u[x] \times c_u'$, and $e_u'[x] = e_u[x] \times e_u'$, where a_u', c_u', and e_u' are invariant across domains). Third, we constrained the interaction coefficients to be same across domains of early child development for both genetic and environmental influences at the measurement level (i.e., $a_u'[x] = a_u'$, $c_u'[x] = c_u'$, and $e_u'[x] = e_u'$, where a_u', c_u', and e_u' are invariant across domains). Fourth, we fixed all the interaction coefficients at the measurement level at zero (i.e., $a_u'[x] = c_u'[x] = e_u'[x] = 0$). Finally, we also fixed the interaction coefficients at the latent factor level at zero to test a model with no age interactions at all (i.e., $a_c' = c_c' = e_c' = a_u'[x] = c_u'[x] = e_u'[x] = 0$).

Table 13 lists the model fit statistics for all the behavioral genetic models. The behavioral genetic multivariate model with all interaction coefficients freely estimated fit the data best, and we, therefore, accept this model as the preferred behavioral genetic model. Combining information across the five abilities to conduct an omnibus test for age differences in genetic and environmental influences at the measurement level led to a

Table 13. Behavioral Genetic Model Fit Statistics

Model		AIC	BIC	LL	*df*	MLR scaling	Model to be Compared With	χ^2 for Model Comparison	Δdf
1	**Freely estimated**	**7153.69**	**7348.01**	**-3523.84**	**53**	**1.43**	2	24.73[i]	12
							3	29.94[i]	12
							4	45.81[i]	15
							5	70.40[i]	18
2	Proportional	7156.46	7306.78	-3537.23	41	1.54	3	4.57[ii]	0
							4	17.34[i]	3
							5	40.55[i]	6
3	Same across domains	7161.02	7311.35	-3539.51	41	1.55	4	13.24[i]	3
							5	35.49[i]	6
4	Interaction coefficients fixed at zero at measurement level	7177.81	7317.13	-3550.90	38	1.53	5	24.01[i]	3
5	All interaction coefficients fixed at zero	7201.51	7329.84	-3565.76	35	1.56	-	-	-

Note. Bolded = preferred model. 1 = Behavioral genetic model with each age-related interaction coefficient independently and freely estimated. 2 = Behavioral genetic model with each age-related interaction coefficient at the measurement level constrained to be proportional to the corresponding main effect. 3 = Behavioral genetic model with each age-related interaction coefficient constrained to be the same across domains of development for both genetic and environmental influences at the measurement level. 4 = Behavioral genetic model with all age-related interaction coefficients fixed at zero at the measurement level. 5 = Behavioral genetic model with all age-related interaction coefficients fixed at zero.

[i] $p < .05$.

[ii] difference in BIC is calculated to compare model fitness across Models 2 and 3 as the χ^2 values are equivalent and the degrees of freedom are the same across the two models.

significant loss of model fit. This suggests that age differences at the domain-specific level emerge independently for each domain.

Tables 14 and 15 list the parameter estimates from all the behavioral genetic models. At the domain-general level, we observed age differences in genetic and shared environmental influences but not in nonshared environmental influences (see Table 14 and Figures 8 and 9); genetic commonality increased while shared environmental commonality decreased with age. At the domain-specific level, there is little evidence for age differences in genetic and environmental influences, except for shared environmental influences on *Fine Motor* and nonshared environmental influences on *Fine Motor* and *Problem-Solving* (see Table 15 and Figures 8 and 9). We focus on results from the preferred behavioral genetic model in the following sections.

Table 14. Parameter Estimates (with Confidence Intervals in Brackets) at the Domain-General Level

Effects of			1[i]		2		3		4		5
			Main Effect	Inter-action	Main Effect	Inter-action	Main Effect	Inter-action	Main Effect	Inter-action	Main Effect
Broad Ability	On	Comm-unication	**1.00**[ii]		**1.00**[ii]		**1.00**[ii]		**1.00**[ii]		**1.00**[ii]
		Gross Motor	**.99**[.69, 1.30]		**.99**[.67, 1.29]		**.99**[.69, 1.30]		**1.03**[.70, 1.37]		**1.00**[.69, 1.31]
		Fine Motor	**1.21**[.91, 1.50]	-	**1.20**[.90, 1.50]	-	**1.21**[.90, 1.52]	-	**1.31**[.94, 1.68]	-	**1.25**[.93, 1.58]
		Problem-Solving	**1.01**[.73, 1.28]		**1.03**[.75, 1.31]		**1.04**[.76, 1.32]		**1.12**[.81, 1.43]		**1.05**[.76, 1.34]
		Personal-Social	**1.16**[.86, 1.45]		**1.15**[.87, 1.43]		**1.14**[.86, 1.42]		**1.15**[.86, 1.44]		**1.17**[.87, 1.46]
A_c			.02[-.23, .28]	**.11**[-.02, .19]	-.07[-.28, .14]	**.14**[.06, .21]	-.06[-.28, .15]	**.14**[.06, .21]	.07[-.15, .28]	**-.14**[-.21, -.06]	**.31**[.15, .46]
C_c		Broad Ability	**.71**[.44, .99]	**-.12**[-.22, -.02]	**.73**[.46, 1.01]	**-.13**[-.23, -.03]	**.73**[.46, 1.00]	**-.13**[-.23, -.03]	**.72**[.44, .99]	**-.13**[-.23, -.04]	**.44**[.28, .59]
E_c			.13[-.02, .28]	.02[-.05, .08]	**.20**[.07, .33]	-.01[-.07, .04]	**.20**[.05, .34]	-.01[-.08, .05]	**-.14**[-.28, -.01]	-.01[-.06, .05]	**.17**[.11, .24]

Note. Bolded = $p < .05$. 1 = Behavioral genetic model with each age-related interaction coefficient independently and freely estimated. 2 = Behavioral genetic model with each age-related interaction coefficient at the measurement level constrained to be proportional to the corresponding main effect. 3 = Behavioral genetic model with each age-related interaction coefficient constrained to be the same across domains of development for both genetic and environmental influences at the measurement level. 4 = Behavioral genetic model with all age-related interaction coefficients fixed at zero at the measurement level. 5 = Behavioral genetic model with all age-related interaction coefficients fixed at zero.

[i] preferred model. [ii] first loading factor was fixed at one to facilitate model convergence.

Table 15. Parameter Estimates (with Confidence Intervals in Brackets) at the Measurement Level

Effects of		1[i]		2		3		4	5
		Main Effect	Inter-action	Main Effect	Inter-action	Main Effect	Inter-action	Main Effect	Main Effect
A_u	Communication	.19[-.42, .81]	.07[-.12, .26]	**.43**[.21, .65]	-.05 [-.14, .04]	**.44**[.21, .67]	-.04 [-.10, .02]	**.36**[.13, .59]	**.34**[.11, .57]
	Gross Motor	**.81**[.59, 1.03]	-.04[-.15, .07]	**.81**[.64, .99]		**.82**[.66, .99]		**.74**[.61, .87]	**.74**[.61, .87]
	Fine Motor	**.39**[.01, .76]	-.09[-.23, .04]	0±.01		-.04[-.24, .16]		0±.01	0±.01
	Problem-Solving	**.56**[.21, .90]	-.05[-.15, .05]	**.51**[.13, .89]		**.55**[.23, .87]		**.44**[.16, .72]	**.48**[.20, .75]
	Personal-Social	.33[-.10, .75]	.03[-.11, .17]	**.48**[.28, .67]		**.48**[.28, .68]		**.42**[.24, .59]	**.39**[.23, .56]
C_u	Communication	**.78**[.54, 1.02]	-.11[-.21, <.01]	**.59**[.41, .76]	-.04[-.10, .03]	**.59**[.44, .73]	-.02 [-.05, .02]	**.55**[.42, .68]	**.55**[.43, .68]
	Gross Motor	0±.01	0±.01	0±.01		.04[-.07, .15]		0±.01	0±.01
	Fine Motor	**.48**[.27, .68]	.02[-.06, .10]	**.59**[.47, .71]		**.58**[.46, .70]		**.54**[.46, .63]	**.54**[.45, .62]
	Problem-Solving	**.74**[.55, .92]	**-.12**[-.17, -.07]	**.51**[.24, .77]		**.50**[.28, .72]		**.46**[.26, .65]	**.44**[.23, .65]
	Personal-Social	**.45**[.15, .76]	.04[-.07, .14]	**.58**[.42, .74]		**.60**[.44, .76]		**.54**[.40, .69]	**.55**[.42, .68]
E_u	Communication	**.36**[.15, .56]	<.01[-.06, .07]	**.22**[.13, .31]	**.25** [.03, .46]	**.23**[.13, .34]	**.06** [.02, .09]	**.37**[.30, .44]	**.37**[.30, .44]
	Gross Motor	.19[-.14, .53]	.09[-.05, .24]	**.27**[.16, .37]		**.28**[.15, .42]		**.41**[.28, .55]	**.41**[.28, .55]
	Fine Motor	.12[-.14, .38]	**.13**[.03, .22]	**.28**[.19, .37]		**.29**[.20, .39]		**.44**[.37, .52]	**.46**[.38, .53]
	Problem-Solving	.16[-.08, .40]	**.10**[<.01, .19]	**.25**[.14, .35]		**.26**[.14, .37]		**.41**[.29, .52]	**.41**[.29, .52]
	Personal-Social	**.35**[.12, .58]	-.04[-.14, .05]	**.15**[.07, .24]		**.14**[.01, .27]		**.26**[.16, .36]	**.25**[.15, .34]

Note. Bolded = $p < .05$. 1 = Behavioral genetic model with each age-related interaction coefficient independently and freely estimated. 2 = Behavioral genetic model with each age-related interaction coefficient at the measurement level constrained to be proportional to the corresponding main effect. 3 = Behavioral genetic model with each age-related interaction coefficient constrained to be the same across domains of development for both genetic and environmental influences at the measurement level. 4 = Behavioral genetic model with all age-related interaction coefficients fixed at zero at the measurement level. 5 = Behavioral genetic model with all age-related interaction coefficients fixed at zero.

[i] preferred model.

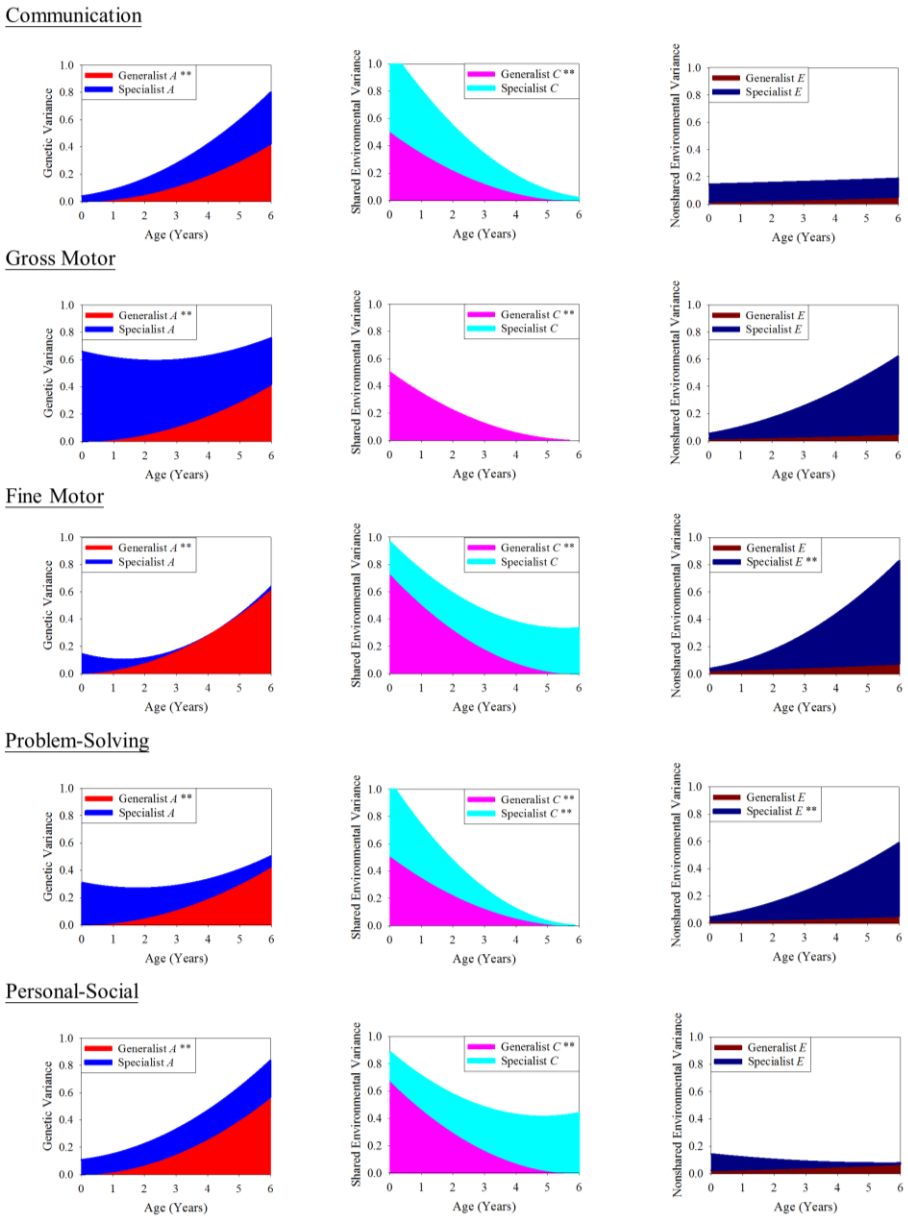

Figure 8. Age trends in unstandardized genetic and environmental contributions to the five domains of early child abilities, decomposed into domain-general (generalist) and domain-specific (specialist) components. Estimates are based on expectations from the preferred behavioral genetic model, in which all age-related interaction coefficients were independently and freely estimated. Rows correspond to ability domains (*Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social*). Columns correspond to genetic, shared environmental, and nonshared environmental variance components.

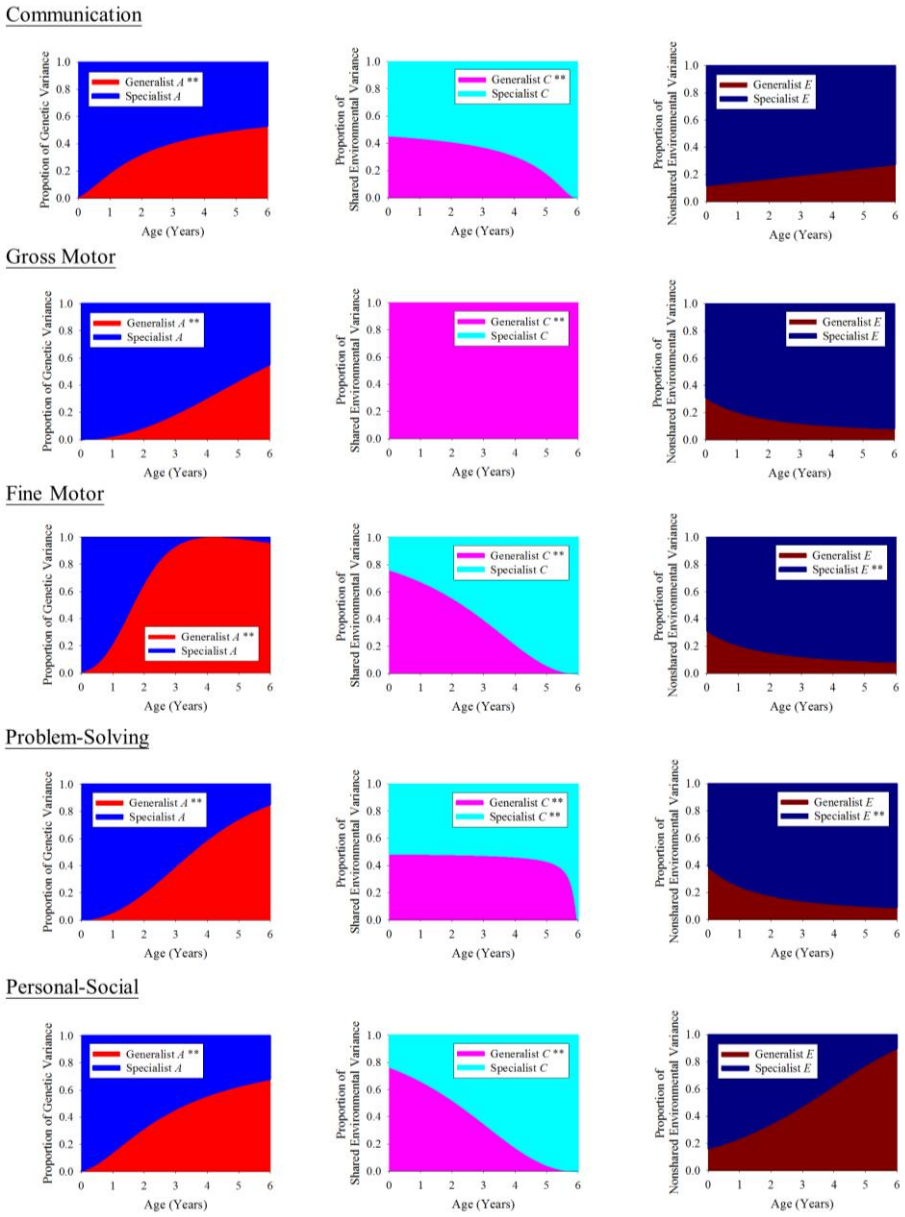

Figure 9. Age trends in structure of total genetic and environmental contributions to the five domains of early child abilities, decomposed into domain-general (generalist) and domain-specific (specialist) proportions. Estimates are based on expectations from the preferred behavioral genetic model, in which all age-related interaction coefficients were independently and freely estimated. Rows correspond to ability domains (*Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social*). Columns correspond to genetic, shared environmental, and nonshared environmental variance components.

Increasing Heritability

Unstandardized genetic variance increased from as low as .04 shortly after birth (e.g., for *Communication*, $[[.022 + 0 \times .105] \times 1]^2 + [.194 + 0 \times .070]^2$) to as high as .84 by age 6 (e.g., for *Personal-Social*, $[[.022 + 6 \times .105] \times 1.157]^2 + [.326 + 6 \times .032]^2$) for all domains of early child abilities except *Gross Motor*, in which unstandardized genetic variance ranged from .59 (i.e., $[[.022 + 2.32 \times .105] \times .993]^2 + [.811 + 2.32 \times [-.038]]^2$) to .76 (i.e., $[[.022 + 6 \times .105] \times .993]^2 + [.811 + 6 \times [-.038]]^2$) across ages (see the fourth and fifth columns of Tables 14 and 15, and the first column of Figure 8). Below, we describe the decomposition of genetic and environmental influences into generalist and specialist components.

Generalist Genetic and Environmental Effects

Figure 8 illustrates age differences in generalist and specialist genetic and environmental influences on each domain of early child abilities. Areas highlighted in red (see the first column of Figure 8) represent unstandardized variance in each domain explained by generalist genes. These panels demonstrate increasing unstandardized generalist genetic variance from almost 0 shortly after birth to approximately half a unit by age 6 for all domains of early child abilities. Areas highlighted in pink (see the second column of Figure 8) represent unstandardized variance in each domain explained by generalist shared environmental factors. These panels demonstrate decreasing unstandardized generalist shared environmental variance from approximately half a unit shortly after birth to almost 0 by age 6 for all domains of early child abilities. Areas highlighted in dark red (see the third column of Figure 8) represent unstandardized variance in each domain explained by generalist nonshared environmental factors. These panels demonstrate relatively trivial and constant influences of generalist nonshared environmental factors across ages on all domains of early child abilities.

Specialist Genetic and Environmental Effects

Results indicate substantial genetic effects at the domain-specific level (see estimates for A_u in the fourth and fifth columns of Table 15). However, none of the interactions between the domain-specific genetic factors and age reached statistical significance. In the first column of Figure 8, areas highlighted in blue represent unstandardized variance in each domain explained by specialist genes. These panels illustrate similar amounts of domain-specific genetic influences across ages for all domains of early child abilities.

We also observed substantial influences of specialist shared environmental factors (see estimates for C_u in the fourth and fifth columns of Table 15). At the domain-specific level, environmental factors that made children more similar to each other explained a sizable amount of variations in each domain of early child abilities except *Gross Motor*, on which shared environmental factors had no effects at all. In the second column of Figure 8, areas highlighted in light blue represent unstandardized variance in each domain explained by specialist shared environmental factors. These panels illustrate relatively trivial age differences in domain-specific shared environmental influences except for *Problem-Solving*. At the domain-specific level, unstandardized variance in *Problem-Solving* explained by shared environmental factors decreased from .54 shortly after birth (i.e., $[.737 + 0 \times [-.120]]^2$) to almost 0 by age 6 (i.e., $[.737 + 6 \times [-.120]]^2$; see the fourth row of the second column of Figure 8). For each of the other four domains of early child abilities, specialist shared environmental factors explained variation in young children's functioning to similar extents across ages.

Nonshared environmental influences at the domain-specific level are generally modest and only those on *Communication* and *Personal-Social* reached statistical significance (see estimates for E_u in the fourth and fifth columns of Table 15). In the third

column of Figure 8, areas highlighted in dark blue represent unstandardized variance in each domain explained by specialist nonshared environmental factors. These panels illustrate relatively trivial age differences in domain-specific nonshared environmental effects except for *Fine Motor* and *Problem-Solving*. Unstandardized nonshared environmental variance unique to *Fine Motor* increased from .01 shortly after birth (i.e., $[.120 + 0 \times .125]^2$) to .76 by age 6 (i.e., $[.120 + 6 \times .125]^2$; see the third row of the third column of Figure 8). Similarly, unstandardized nonshared environmental variance unique to *Problem-Solving* increased from .03 shortly after birth (i.e., $[.164 + 0 \times .095]^2$) to .54 by age 6 (i.e., $[.164 + 6 \times .095]^2$; see the fourth row of the third column of Figure 8). For the other three domains of early child abilities, influences of specialist environmental factors unique to a child remained modest across ages.

Developmental Trends in Proportional Generalist and Specialist Genetic Effects

Our results, as detailed above, indicate that the importance of specialist genes remains similar while that of generalist genes grows with age. This suggests that age-related increase in heritability is localized to generalist genes in early child functioning. As demonstrated in the first column of Figure 9, this also means that the proportion of total heritability attributed to specialist genes decreases but the proportion attributed to generalist genes increases with age. These developmental changes in genetic structure demonstrate a growing statistical pleiotropy (i.e., overlap of genetic influences) across domains of early child abilities with age. Our results are, thus, consistent with the predictions of a transactional perspective, which holds that statistical pleiotropy strengthens over time.

DISCUSSION

Domain-general genetic effects, i.e., statistical pleiotropy, on children's abilities have been well documented (e.g., Petrill, 2005; Plomin & Kovas, 2005; Plomin et al., 2007). Two major classes of underlying mechanisms have been postulated to account for domain-general genetic effects, and each provides different predictions for how statistical pleiotropy changes across development. The *endogenous* perspective predicts no age differences in the magnitude of statistical pleiotropy, whereas the *transactional* perspective predicts increasing statistical pleiotropy with age. With a twin sample ranging from ages 0 to 6 years, we tested for age differences in genetic and environmental influences on early child abilities at both the domain-general and domain-specific levels.

Consistent with the *transactional* perspective, our results indicate that age differences in genetic influences are localized to the domain-general level. Genetic influences on early child abilities gradually evolve from being predominantly modular shortly after birth to predominantly molar by school-entry age. Thus, as children develop, genes become more important in explaining variation in individual abilities as well as the association between different abilities. Transactional models (e.g., Dickens, 2007; van der Maas et al., 2006) predict that statistical pleiotropy emerges and strengthens over time via reciprocal effects of abilities on one another and between abilities and the environment. Such transactional processes may of course co-occur with other, possibly epigenetic, processes. For instance, as young children select and evoke experiences from their surroundings, those experiences may modulate gene expression (Tucker-Drob & Briley, 2014). At the same time, genes may become expressed or silenced over the course of development via biological maturation (see Briley & Tucker-Drob, 2013). Further research is needed to test and model such processes.

At the domain-specific level, we observed no age differences in genetic influences on early child abilities. At the same time, for four of the five domains of early child abilities, results indicate that total heritability increases with age. The combination of these findings suggests that, *relative to generalist genes*, specialist genes become less important in children's abilities with age. Our results also indicate that increasing heritability occurs mostly at the domain-general level. This is consistent with the hypothesis that, as children age and gain increasing autonomy, they mold and create experiences that both reinforce their initial genetic advantages (or disadvantages) and promote (or impede) their development in *multiple* domains of functioning. Because individual environmental experiences have the potential to stimulate multiple abilities simultaneously, an initial genetically influenced aptitude or proclivity in a particular domain might lead children to experiences that promote their development across many different ability domains.

In contrast to our findings for the other four domains of early child abilities, we did not observe an increasing heritability for *Gross Motor*. While it is possible that there may truly be no substantial developmental changes in heritability of this particular domain of functioning until later in development, this possibility would appear at odds with the dramatic mean-level increases in gross-motor development during early life. Interestingly, at the descriptive level, Figure 3 indicates that generalist genetic influences on *Gross Motor* ability increase with age, while specialist genetic influences on *Gross Motor* ability decrease with age. Thus, the relatively stable total genetic effects on *Gross Motor* ability with age appear to have masked pronounced, yet opposing, developmental trends in its generalist and specialist genetic components.

Here, we reported one of the first studies of developmental changes in genetic structure of early child abilities. As sample sizes grow and longitudinal measures

accumulate, we will be well-positioned to apply more specialized longitudinal models (e.g., growth curve models and cross-lagged models) to our data. Such models would allow us to more directly track developmental changes in total heritability and genetic commonality. Future work would also benefit from examining these topics at later developmental stages. As environmental exposures and life experiences in early and middle childhood tend to be very different, it is unclear how genetic commonality may unfold at later ages. For example, it is possible that genetic commonality may grow across lifespan as individuals gain more autonomy in creating experiences that reinforce their genetic predispositions and facilitate (or impede) their overall development. Alternatively, growth in genetic commonality may be slow later in development, as individuals become specialized in activities that they are particularly good at or enjoy doing.

Strengths and Limitations

Our study is among the first to test for age differences in genetic and environmental influences on multiple domains of early child abilities at both the domain-general and domain-specific levels in the first years of life. This contrasts with behavioral genetic studies of cognitive development that conventionally begin following children only after school entry or, in instances in which early years of life are studied, typically focus on global, unidimensional measures of ability. Nevertheless, it is important that we also highlight our study's limitations.

First, the number of individual twins providing data for our analyses was relatively low in comparison to many behavioral genetic studies. However, higher ratios of indicator number to factor number and consistently high factor loadings have been shown to mitigate the impact of relatively small sample sizes on model results (see

MacCallum, Widaman, Zhang, & Hong, 1999; also see Preacher & MacCallum, 2002). Moreover, we increased parameter precision by including longitudinal data from participants when available while employing estimation methods to prevent biases due to nesting of occasions within individuals. Additionally, rather than performing a large number of sequential hypothesis tests on a parameter-by-parameter basis, we used multivariate methods that compared different sets of parameter specification and constraint to one another. Our key findings derive from the joint pattern of results across all parameters in the model, and do not rely on a single key parameter or its *p*-value. Accordingly, we have taken an approach that emphasizes effect sizes, rather than significance levels.

Second, our findings are based on age-comparative analyses of data collected from individuals of different ages combined with those collected from the same individuals longitudinally. We used vertical scaling and created overlapping items across assessments for different age groups to ensure that scores produced are comparative across individuals of different ages for each domain. As mentioned earlier, we also used appropriate analytical procedures to account for the nonindependence of longitudinal data collected on the same individual. Nevertheless, future research would do well to capitalize on longitudinal data to fit explicit models of age-related change over time, rather than simply age-related difference.

Third, most parents of twins or multiples in this sample have completed college education or beyond. Developmental increases in heritability may not be as pronounced among more disadvantaged samples (Tucker-Drob et al., 2013; Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011). While the initial sample included a larger portion of participants recruited through community outreach, the ongoing recruitment focuses more heavily on identifying eligible families from birth records provided by the Texas

Department of State Health Services. Such effort should add further socioeconomic and ethnic diversity to the sample as it grows.

Fourth, data were collected using surveys completed by the twins' primary caregivers and are, thus, potentially subject to social desirability and the primary caregivers' biases about their children's abilities. However, instead of relying on parents' subjective impression of their children's development of various skillsets, this assessment of early child abilities is based on parents' report on children's performance on concrete tasks (e.g., *Does your child count up to 15 without making mistakes? If so, mark "yes." If your child counts to 12 without making mistakes, mark "sometimes."*). These tasks are designed to objectively reflect children's attainment of various developmental milestones in different domains of early child functioning. This reduces primary caregivers' biases and misjudgments as compared to sole reliance on their subjective impression of their children's abilities relative to other same-age children. All primary caregivers also completed the surveys in the privacy of their homes, which can effectively reduce social desirability when self-reporting personal behaviors (Richman, Kiesler, Weisband, & Drasgow, 1999).

Most importantly, parent ratings on the ASQ have been shown to correlate with independent observer ratings at .86, indicating excellent inter-rater reliability across informants (Squires et al., 2009). The ASQ has also consistently demonstrated high convergent validity with researcher/clinician-administered scales of early mental development across a number of independent studies. Squires and colleagues compared the parent-administered ASQ's sensitivity to young children's progress in attaining developmental milestones to that of the examiner-administered Battelle Development Inventory and found that the ASQ demonstrated high sensitivity and specificity (i.e., .86 on both indices). Compared to classification based on scores on the examiner-

administered Bayley, one gold standard in assessing early child development, the parent-administered ASQ demonstrated sensitivity as high as 1.00 and specificity as high as .97 (Gollenberg et al., 2010; Schonhaut et al., 2013; Simard et al., 2012). In particular, Schonhaut and colleagues found a moderately high correlation of .51-.75 between ASQ and Bayley scores among children of ages 8, 18, and 30 months. Using an international sample of 828 children of ages 12-60 months, Yu et al. (2007) found that ASQ demonstrated sensitivity of .63-.97 and specificity of .81-.84 when results were compared to those based on clinical examinations and neurodevelopmental assessments such as the Bayley, Griffiths Mental Development Scales, and Denver Developmental Screening Test.

Fifth, in our study, the same primary caregiver rated both twins in each pair. Using data from single informant may inflate the similarity in ratings across twins in a pair and, hence, the genetic and/or shared environmental variance estimates at a given time point. Yet, we do not expect such single-informant biases to systematically increase or decrease across age. Our key findings focus on the general pattern of developmental changes observed in heritability across major domains of early child abilities rather than the magnitude of a given estimate at a given time point.

Sixth, it is unclear whether the age-related increases we observed in nonshared environmental influences on *Fine Motor* and *Problem-Solving* at the domain-specific level indicate growing influences of environmental factors that are unique to a child or simply an increase in measurement error across item sets of increasing difficulty. Studies interested in testing for age differences in domain-specific nonshared environmental influences may, for example, include survey items as observed indicators in their multivariate models and set various domains of abilities as latent factors subordinate to the single latent factor representing overall ability. Because nonshared environments

include measurement error only at the observed variable level, separating domain-specific variance into latent and observed components is one potential way to capture any true specialist nonshared environmental influences and their changes across development.

CONCLUSION

Our study is among the first to test for age differences in the multivariate genetic structure of early child abilities. Results indicate that age-related increases in the heritabilities of early child abilities are mostly driven by the growing influence of generalist genes. These results are consistent with transactional models that predict strengthening of statistical pleiotropy over time via reinforcing transactions among different abilities and between these abilities and the environment.

Chapter 3: Genetic and Environmental Links between Parenting and Early Childhood Psychological Development

Authors[3]: Amanda K. Cheung, K. Paige Harden, & Elliot M. Tucker-Drob

Status: In preparation

Transactional perspectives posit that caregivers not only instill their values and norms in their children through socialization but also adjust their childrearing behaviors to children's characteristics and behaviors (Bell, 1968; Bell & Chapman, 1986; Belsky, 1984; Sameroff & MacKenzie, 2003). Behavioral genetic research indicates that parenting is "heritable" on the part of genetic variation in children, suggesting that children differentially evoke parenting on the basis of their genetically influenced characteristics (Avinun & Knafo, 2013; Kendler & Baker, 2007; Klahr & Burt, 2014). While much previous work in this area has estimated the magnitude of child genetic influences on parenting received, researchers have more recently begun to examine specific genetically-influenced child characteristics that might account for such differential parenting. Such work, however, is relatively recent and has been limited to pairwise examinations of how genetic and environmental influences on a specific child outcome relate to variation in a specific parenting practice. Bivariate approaches do not distinguish the extent to which associations are specific to the pairs of parenting practices and child outcomes under investigation, are reflective of associations at much more general dimensions of parenting and child development, or result from a mixture of domain-general and domain-specific associations. The current study, therefore, examines

[3] Drs. Elliot Tucker-Drob and Paige Harden are the principle investigators of the Texas "Tiny" Twin Project, which provides data used in Study 3. Drs. Tucker-Drob and Harden also provided feedback to the first author throughout the development and manuscript preparation of Study 3.

the relations between a broad assortment of parenting practices and child outcomes within a large multivariate, multidimensional, system.

Genetic and Environmental Pathways of Parent- and Child-Driven Processes

Quantitative behavioral genetic approaches elucidate the mechanisms underlying mutual influences between parenting and early child development by estimating the extent to which these associations are genetically and environmentally mediated. Parent-to-child influences refer to parents' socialization of their children into their norms, whereas child-to-parents influences refer to parents' adjustment of caregiving behaviors to characteristics of their children (see Scarr & McCartney, 1983; also see Figure 1 for a pictorial illustration on how child genetic factors influence parenting). For example, children with genetic dispositions for higher verbal skills may choose to read whenever they have a chance and, at the same time, their parents may bring them to the library every week in response to their interest in reading. In addition to genetic variation, parents may treat their children differently due to nongenetic factors – reasons that are idiosyncratic to the parents (e.g., differential parental expectations) or child differences driven by differential exposures (e.g., a child who suffered severe injuries from an accident likely receive higher levels of parental care than his or her siblings with no such incidents).

In a classic behavioral genetic model, variation in a child outcome is modeled as the sum of three biometric components: additive genes (*A*), shared environments (*C*) – environmental contributions to the similarity across children reared together, and nonshared environments (*E*) – environmental contributions to the dissimilarity across children reared together. Within-pair similarity on a given construct is compared across kinship pairs of different genetic relatedness (e.g., monozygotic [MZ] versus dizygotic

[DZ] twins, full versus half siblings, etc.) to ascertain genetic and environmental contributions to that particular construct. Greater similarity between children of greater genetic relatedness indicates genetic influences, whereas similarity between children reared together regardless of their genetic relatedness indicates shared environmental influences. Within-pair variation unaccounted for by genetic factors represents nonshared environmental influences – experiences that differentiate the outcomes of children (even genetically identical children, i.e., MZ twins) reared together.

When the classic behavioral genetic model is applied to measures of environments experienced by children, it can be used to make inferences about child genetic and environmental contributions to these environmental constructs. In child-based studies of parenting, shared child environments represent environmental factors shared within a kinship pair (or environmental differences across families), which may include broad contextual characteristics (i.e., environments in which a family is embedded; e.g., socioeconomic status, neighborhood quality, cultural background, etc.) and parental characteristics (e.g., parents' genetic dispositions, upbringing, life experiences, etc.). Child genetic influences on parenting refer to parents' modification of childrearing behaviors in response to child genetically driven characteristics, whereas nonshared child environmental influences on parenting indicate differential parenting within a family that is unrelated to child genetic variation (e.g., reasons idiosyncratic to the parent, experiences unique to a child, etc.). Such genetically informative designs have been applied in various studies of parenting (see Avinun & Knafo, 2013; Kendler & Baker, 2007; Klahr & Burt, 2014); results converge to indicate shared environmental effects (similarity in parenting within a kinship pair after accounting for their genetic relatedness, likely due to factors at the caregivers' level), child genetic effects (similarity in parenting within a kinship pair associated with the genetic relatedness of the pair,

which suggests that caregivers adjust their childrearing behaviors to children's genetically driven characteristics), and nonshared child environmental effects (dissimilarity in parenting within a kinship pair that is unrelated to genetic variation, which suggests differential childrearing behaviors for reasons idiosyncratic to caregivers themselves or in response to children's non-genetically driven characteristics).

Multidimensionality of Parent- and Child-Driven Processes

Although increasing effort has been put into clarifying the behavioral genetic mechanisms underlying the association between parenting and child outcomes in recent years, previous work has mostly examined these genetic and environmental processes between specific measures in isolation of other related constructs (e.g., Alemany, Rijsdijk, Haworth, Fañanás, & Plomin, 2013; Larsson, Viding, Rijsdijk, & Plomin, 2008; Tucker-Drob & Harden, 2012a). Each of these bivariate findings likely overlaps with one another and shed only partial light on the complex associations between parenting and child development. For example, caregivers may be more negative or harsh not only toward children genetically disposed with a bad temper but, possibly out of frustration, also toward those genetic disposed to be slower learners; at the same time, caregivers may provide more cognitive stimulation not only to children genetically disposed to be fast learners but also to those genetically disposed toward acting more prosocially (resulting in a more positive parent-child relationship that in turn encourages parental investment). In this scenario, children's aptitude and temperaments may be driven by overlapping sets of genetic dispositions (e.g., genetic risks for inattention and hyperactivity). Thus, child genetic influences on parental negativity may overlap – in full or in part – with those on parental cognitive stimulation by way of measurable, genetically-influenced, child characteristics.

Multivariate findings regarding child genetic effects on a wide assortment of parenting measures indicate that associations between parenting and child development are likely a combination of domain-general and domain-specific processes. Having accounted for the unified effects of child genetic factors on various distinct but related parenting behaviors, there remains a nontrivial amount of child genetic influences unique to each construct (see Study 1). This suggests that certain genetically driven child characteristics evoke differential parenting style at the broad dimensions whereas others prompt caregivers to refine their specific childrearing practices. Only through examining genetic and environmental links between parenting and child phenotypes from a multidimensional perspective can we better understand the complex associations between parenting and child development: To which genetically driven child characteristics and to what extent do caregivers adjust their behaviors at broad and specific dimensions of parenting? At each level of generality or specificity, do caregivers adjust their behaviors to children's genetic dispositions spanning across multiple domains or unique to a given phenotype?

Current Study

Evidence for genetically and environmentally mediated parent- and child- driven processes in child development was mostly limited to associations between specific parenting and child outcome measures. Multidimensional analysis of parenting data suggests that caregivers adjust their broad parenting style to genetically driven child characteristics that are independent from those to which they adjust their specific parenting practices. To better understand the complex associations between parenting and child development, it is important to identify the genetically driven child characteristics involved in these independent child-driven processes. That is, do caregivers adjust their

broad and specific caregiving behaviors in response to children's genetic potential in general development or specific functioning? Using multivariate parenting and child outcome data from a population-based sample of families with twins and multiples at ages 0-6 years, this study estimated the extent to which genetic and environmental pathways mediate the associations between parenting and child outcomes at both general and specific dimensions.

METHODS

Participants

Participants were drawn from a downward extension of the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013) to families with twins or multiples aged 0-6 years who were living in or born in the state of Texas. Potential families were identified from birth records provided by the Texas Department of State Health Services. Additional recruitment efforts included attending the annual convention of the Texas Mothers of Multiples and out-reach through list-serve of interest groups targeting families with twins or multiples. Once a family enrolled in the study, a paper or online survey was sent to the primary caregiver of twins or multiples according to his or her survey preference. After completing the first survey, participating families were eligible for follow-up surveys every 2 months for children under 2 years old, every 3 months for children between 2 and 3 years old, every 5 months for children between 3 and 5 years old, and one last survey for children between 5 and 6 years old.

Recruitment for the Texas Twin Project is on-going. For this study, data were available from 628 individual twins and triplets. Average age at baseline was 2.57 years old (*SD* = 1.29 years). This sample was 66.78% Caucasian, 5.22% Latino, 6.43% African American, 2.09% Asian, and 17.04% racially mixed. Among this young sample, 3.48%

of their primary caregivers did not complete high school, 4.35% completed high school, 9.22% received education beyond high school but did not complete college, 38.96% completed college, and 46.78% received education beyond college. Pre-tax household income median for families in this sample was USD109,000 and their average log-transformed income was USD84,027. Ninety-three percent of primary caregivers in this sample were the biological mother of their twins or multiples.

Zygosity was assigned based on physical similarity ratings. This method of zygosity assignment is highly reliable and matches well with that based on DNA genotyping (Forget-Dubois et al., 2003; Heath et al., 2003; Price et al., 2000; Rietveld et al., 2000). Primary caregivers rated the pair-wise physical similarity of their twins or multiples on 12 items (e.g., facial appearance, ear lobe shape, eye color, etc.). Zygosity of the 229 same-sex pairs was determined using a two-class Latent Class Analysis (see Harden, Kretsch, Tackett, & Tucker-Drob, 2014). The 115 opposite-sex pairs were classified as DZ twins. This resulted in a sample of 142 MZ twins (74 male and 68 female individual twins), 234 same-sex DZ twins (124 male and 110 female individual twins), 192 opposite-sex DZ twins (96 male and 96 female individual twins), and 60 triplets (8 male MZ triplets, 12 female MZ triplets, 18 male DZ triplets, and 22 female DZ triplets; each set of triplets made up to 3 pairs of "twin data;" we accounted for the nonindependence of data from pairs within the same triplet set using the Complex Survey option in *Mplus* statistical software [Muthén & Muthén, 2010]).

In addition to the baseline observations on each of the 628 individual twins or multiples, follow-up data were available on 270 of these individual twins for up to 11 follow-up waves. To maximize the use of data available while preserving the precision of resulting parameter estimates, we included both baseline and longitudinal follow-up data in all analyses using the Complex Survey option in *Mplus* statistical software (Muthén &

Muthén, 2010). Specifically, data on the same twin across different survey waves were entered on different rows of the dataset and the standard errors of parameter estimates were corrected for potential biases from the nonindependence of data on the same individual across time. This resulted in a final effective sample size of 1,400 observations from 628 individual twins – 310 observations from MZ twins, 1090 observations from DZ twins. Among these 1,400 observations, average age at measurement is 2.62 years old (*SD* = 1.30 years).

Measures

Parental Cognitive Stimulation

Twenty one items were created in-house to assess primary caregivers' self-report on their frequency of engaging their children in cognitive stimulating activities (see Study 1 for more information). Primary caregivers rated on a 7-point Likert scale (from *All the Time* to *Not at all/Not applicable*) on 12 items that measure day-to-day routines that promote children's cognitive development (i.e., *Daily Stimulating Activities*; e.g., "How often do you play peek-a-boo/hide-and-seek or hide a toy for your child to find?") and 8 items that measure the frequency of more organized cognitive stimulating activities (i.e., *Learning Activities*; "How often do you bring your child to outdoor educational activities or field trips [e.g. visiting the zoo, petting farm, science museum, nature center, etc.]?"). Scores were reversed and averaged across items so higher scores indicate higher levels of parental cognitive stimulation. Cronbach's alpha was .87 for *Daily Stimulating Interactions* and .68 for *Learning Activities*.

Parenting Young Children (PARYC)

PARYC measures primary caregivers' self-report on their use of parenting practices associated with positive child outcomes (McEachern et al., 2012). *Supporting*

Positive Behavior refers to practices that provide extrinsic or intrinsic positive reinforcement (e.g., "Reward your child when s/he did something well or showed a new skill"), *Setting Limits* refers to practices that enforce appropriate boundaries for child behaviors (e.g., "Stick to your rules and not change your mind"), and *Proactive Parenting* refers to practices that prepare children for upcoming activities (e.g., "Give reasons for your requests [such as *We must leave in five minutes, so it's time to clean up*]"). Primary caregivers rated on 7 items for each domain using a continuum that ranged from 0 – *Not at all* to 100 – *Most of the Time*, with higher average scores indicating more frequent use of adaptive parenting practices. McEachern and colleagues observed satisfactory factor loadings for all 3 domains and correlations with other standardized measures of parenting practices and child outcomes. Cronbach's alpha was .76 for *Supporting Positive Behavior*, .86 for *Setting Limits*, and .91 for *Proactive Parenting*.

Emotion Socialization Questionnaire (ESQ)

ESQ is a self-report measure adapted from the Emotions as a Child scale (EAC) and measures primary caregivers' self-report on their typical reactions to children's expression of sadness, anger, and fear (Klimes-Dougan et al., 2007). *Emotional Support* refers to parenting behaviors that encourage children's adaptive response to distress (e.g., "Asked my child about it" and "Helped my child deal with the problem"), *Emotional Magnification* refers to parenting behaviors that simply match children's expression of distress without modeling appropriate regulatory skills (e.g., "Got sad myself" when the child was sad and "Got angry with my child" when the child was angry), and *Emotional Neglect* refers to parents' disregard to children's emotional needs (e.g., "Gave my child space to deal with it" and "I didn't respond"). Primary caregivers rated on 9 items for each domain using a continuum that ranged from 0 – *Not at All Typical* to 100 – *Very*

Typical, with higher average scores indicating greater reliance on that emotional socialization practice. Previous studies using EAC observed high Cronbach's alpha for all domains of parental emotional socialization and high correlations between scores obtained across waves (Garside & Klimes-Dougan, 2002; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001), and high correlations with other standardized measures of child outcomes (Brand & Klimes-Dougan, 2010; O'Neal & Magai, 2005). Cronbach's alpha was .81 for *Emotional Support*, .79 for *Emotional Magnification*, and .79 for *Emotional Neglect*.

Parenting Scale (PS)

PS measures primary caregivers' self-report on their maladaptive response to child misbehaviors (Arnold, O'Leary, Wolff, and Acker, 1993; Rhoades & O'Leary, 2007). Primary caregivers rated on a continuum with each pole representing opposite approaches to child misbehaviors. *Laxness* refers to inconsistent enforcement of rules or instructions (e.g., "When my child does something I don't like: [ranging from 0] I do something about it [to 100] I often let it go"), *Overreactivity* refers to parental behaviors that exacerbate negative parent-child interactions (e.g., "When my child misbehaves: [ranging from 0] I usually get into a long argument with my child [to 100] I don't get into an argument"), *Hostility* refers to both verbal and physical aggression of parents directed at their child (e.g., "When my child misbehaves: [ranging from 0] I rarely use bad language or curse [to 100] I almost always use bad language"). Because disciplinary laxness refers to a lack of consistent enforcement of discipline, we named *Laxness* as *Laxness-Consistent Parenting* in all our analyses. Seven items were reverse-coded and higher average scores across all items indicate greater use of negative parenting practices. Previous work demonstrated strong correlations between PS and other standardized

measures of parenting (Reitman et al., 2001). Cronbach's alpha was .79 for the 5 items on *Laxness-Consistent Parenting*, .78 for the 5 items on *Overreactivity*, and .34 for the 3 items on *Hostility*. Following Rhoades and O'Leary, we applied Spearman-Brown correction and obtained a Cronbach's alpha of .63 for *Hostility* if it was measured by 10 items. Parental hostility toward children is more an exception rather than the norm; this low base rate of *Hostility* may have contributed to its low Cronbach's alpha in both Rhoades and O'Leary's work and the current study. Despite the low base rate of *Hostility*, the extent of its negative impact on child outcomes makes it an important construct to include in studies of parenting and child development.

Ages and Stages Questionnaire, Third Edition (ASQ)

ASQ measures children's attainment of developmental milestones across major domains of early cognitive and psychomotor functioning (Squires & Bricker, 2009). All ASQ items were age-appropriate; for each domain, primary caregivers rated on a set of 5-10 items that varied by child age. Item sets for adjacent age groups contained overlapping items to allow vertical scaling and hence valid score comparisons across children of different ages. For most items, primary caregivers rated their children's ability in completing certain specific, concrete tasks on a 3-point Likert scale (0 – *Not at all*, 1 – *Sometimes*, and 2 – *Yes*).

Communication refers to expressive and receptive language skills (e.g., "Does your child correctly use at least two words like 'me,' 'I,' 'mine,' and 'you?'" – for children aged 19 to 28.49 months, and "Without giving your child help by pointing or using gestures, ask him/her to 'put the book on the table' and 'put the shoe under the chair.' Does your child carry out both of these directions correctly?" – for children aged 25.5 to 44.99 months). *Gross Motor* refers to coordination of large muscle groups

involved in body movements (e.g., "Without holding onto anything for support, does your child kick a ball by swinging his/her leg forward?" – for children aged 21 to 38.99 months). *Fine Motor* refers to coordination of small muscle groups involved in movements of body parts such as fingers (e.g., "When you put a toy in his/her hand, does your baby hold it in his/her hand briefly?" – for children aged 1 to 2.99 months). *Problem-Solving* captures abilities to apply generic rules or logic to accomplish an objective (e.g., "Does your child finish the following sentences using a word that means the opposite of the word that is italicized? For example: 'A rock is hard, and a pillow is soft.'" – for children aged 57 to 71.99 months). *Personal-Social* refers to self-care abilities and social skills (e.g., "Does your child wash his/her hands using soap and water and dry off with a towel without help?" – for children aged 39 to 56.99 months, and "Does your baby smile at you?" – for children aged 1 to 2.99 months).

ASQ accurately and reliably measures children's progress in reaching developmental milestones across ages 2 to 60 months; among a nationally representative sample of 579 young children, ASQ demonstrated sensitivity and specificity of 86% on average (Squires et al., 2009). Other independent studies also observed high correlations between ASQ and standardized researcher/clinician-administered measures of child development (Gollenberg, Lynch, Jackson, McGuinness, & Msall, 2010; Schonhaut, Armijo, Schönstedt, Alvarez, & Cordero, 2013; Simard, Luu, & Gosselin, 2012; Yu et al., 2007). We obtained each domain score from Rasch Item Response Theory (1PL IRT) analyses, with higher scores indicating more advanced development. Item reliability estimates from our 1PL IRT analyses were .92, .90, .86, .86, and .84 for *Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social*, respectively. Application of Spearman-Brown prophecy formula resulted in corrected item reliability estimates of .96, .95, .92, .92, and .91, respectively, for the 5 ASQ domains.

Ages and Stages Questionnaire: Social-Emotional (ASQ:SE)

ASQ:SE measures children's development of social and self-regulatory skills that are important in psychosocial adjustments (Squires, Bricker, & Twombly, 2003). Similar to ASQ, all items in ASQ:SE are age-specific. Primary caregivers rated on a set of 18-32 items that varied by child age. Item sets for adjacent age groups contained overlapping items to allow vertical scaling and hence valid score comparisons across children of different ages. For most items, primary caregivers rated the frequency of their children's behaviors in certain specific, concrete situation on a 3-point Likert scale (0 – *Rarely or Never*, 1 – *Sometimes*, and 2 – *Most of the time*). *Social-Emotional* refers to social and self-regulatory abilities that are critical in adaptive functioning and development of meaningful interpersonal relationships (e.g., "Does your baby laugh or smile at you and other family members?'' – for children aged 9 to 15 months, and "Can your child stay with activities he or she enjoys for at least 15 minutes (not including watching television)?" – for children aged 54 to 66 months).

ASQ:SE accurately and reliably measures children's social-emotional competence across ages 6 to 72 months; among a nationally representative sample of 1,041 young children, ASQ:SE demonstrated a sensitivity of 78% and a specificity of 95%, accurately representing children's social-emotional development and differentiating children at risk for emotional and behavioral maladjustments across ages 6 to 72 months (Squires et al., 2003). Twenty-one of the 77 items were reverse-coded, with higher scores indicating greater competence. We obtained overall *Social-Emotional* scores from a 1PL IRT analysis of all 77 items, with an item reliability estimate of .22. Application of Spearman-Brown prophecy formula resulted in corrected item reliability estimates of .84. In contrast to other ASQ domains, *Social-Emotional* is a domain that reflects a wide range of skills from information processing to emotional regulation and to self-expression, each of

which manifests very differently for a 6-month-old infant versus a 72-month-old preschooler. When measuring broad constructs such as social-emotional competence across early childhood, diversifying the sampling of behaviors may result in lower inter-item correlation but the use of more items with lower item reliability is equivalently reliable or sometimes preferred to sampling with fewer items of greater item reliability (Little, Lindenberger, & Nesselroade, 1999).

ASEBA Child Behavior Checklist for Ages 1.5-5 (CBCL)

CBCL measures parents' impression of children's display of emotional and behavioral problems (Achenbach & Rescorla, 2000a). *Internalizing* refers to problem behaviors that are directed inwards such as emotional reactivity and social withdrawal (e.g., "whining," "unresponsive to affection," and "clings"), whereas *Externalizing* refers to problem behaviors that are directed "outwards" and infringe others' well-being or socially accepted norms (e.g., "clumsy," "defiant," and "fights"). Primary caregivers rate the extent of their children's problem behaviors on a 3-point Likert scale (0 – *Not true*, 1 – *Somewhat or sometimes true*, and 2 – *Very true or often true*). CBCL is commonly used in developmental research and demonstrates high cross-time and cross-informant reliability (Achenbach & Rescorla, 2000b). Independent studies also observed that CBCL reliably differentiated referral group from non-referral group and demonstrated moderate correlations with other standardized measures of early child development (Achenbach & Rescorla, 2000b). Raw sum scores are converted into standardized scores using the conversion rules provided by CBCL, with higher scores indicating greater degree of maladjustment. Standardization of raw sum scores is based on a normative sample of 700 young children drawn from a nationally representative sample; therefore, a standardized score represents the extent of problem behaviors exhibited by the rated child relative to

typical peers of ages 1.5 to 5 years (Achenbach & Rescorla, 2000b). Cronbach's alpha was .78 for the 36 items on *Internalizing* and .88 for the 24 items on *Externalizing*.

RESULTS

Parenting variables with skewed distributions were transformed to obtain a nearly normal distributions (see Table 16). To minimize potential biases in the genetic and environmental estimates due to age and sex differences (see McGue & Bouchard, 1984), we partialled out linear and quadratic influences of age, influences of sex differences, and the interaction among these variables on all parenting and child measures using multiple regression analyses. Age-, age^2-, and sex-partialled correlations among parenting and child measures were generally modest to moderate in size and in the expected directions (see Tables 17a-c, which also list the descriptive statistics of all 11 parenting and 8 child variables). All phenotypic and behavioral genetic multivariate models were tested using *Mplus* statistical software (Muthén & Muthén, 2010). To maximize the use of data available, we used full-information maximum likelihood (FIML) estimation in *Mplus*. FIML produces unbiased estimates under the assumption that any systematic missing data on the dependent variables can be statistically accounted for by data available on other variables for that same individual or row.

Table 16. Skewness Statistics of Variables before and after Transformation.

	Before Transformation		After Transformation	
	Kurtosis/*SE*	Skewness/*SE*	Kurtosis/*SE*	Skewness/*SE*
Emotional Support	2.89 / .15	-1.37 / .08	-.58 / .15	-.20 / .08
Setting Limits	1.67 / .15	-1.08 / .08	-.20 / .15	.14 / .08
Proactive Parenting	1.67 / .15	-1.38 / .08	-.43 / .15	.22 / .08
Laxness-Consistent Parenting	2.01 / .15	1.17 / .08	-.49 / .15	-.04 / .08
Emotional Magnification	1.59 / .15	1.27 / .08	.45 / .15	.35 / .08
Emotional Neglect	1.02 / .15	.27 / .08	.03 / .15	-.53 / .08
Overreactivity	.48 / .15	.86 / .08	-.21 / .15	-.01 / .08
Hostility	4.14 / .15	1.86 / .08	.20 / .15	.88 / .08

Table 17a. Descriptive Statistics (before Transformation and Standardization) of Parenting Measures and Age-, Age^2-, and Sex-Partialled Correlations between Transformed Parenting Measures.

		N	*M* (*SD*)	1	2	3	4	5	6	7	8	9	10
1	Daily Stimulating Interactions	1,108	4.03 (1.43)	-									
2	Learning Activities	1,109	3.70 (.92)	.62***	-								
3	Supporting Positive Behavior	1,035	81.50 (13.28)	.50***	.44***	-							
4	Emotional Support	1,028	89.58 (9.58)	.19***	.20***	.43***	-						
5	Setting Limits	1,013	77.49 (16.11)	.32***	.34***	.64***	.42***	-					
6	Proactive Parenting	1,018	75.84 (21.36)	.40***	.34***	.63***	.43***	.78***	-				
7	Laxness-Consistent Parenting	1,013	21.21 (16.60)	-.14***	-.15***	-.30***	-.26***	-.41***	-.32***	-			
8	Emotional Magnifi-cation	1,033	12.22 (11.55)	-.10**	-.06	-.26***	-.31***	-.37***	-.23***	.28***	-		
9	Emotional Neglect	1,030	20.55 (11.35)	.06*	.03	-.02	-.23***	-.01	-.01	.05	.25***	-	
10	Over-reactivity	1,026	21.59 (16.36)	-.19***	-.23***	-.34***	-.39***	-.50***	-.35***	.28***	.43***	.18***	-
11	Hostility	1,028	7.26 (9.11)	-.11**	-.08*	-.16***	-.28***	-.31***	-.22***	.18***	.41***	.15***	.45***

Note. *N* for each variable represents the number of observations available on that particular variable. *$p < .05$. **$p < .01$. ***$p < .001$.

Table 17b. Descriptive Statistics (before Transformation and Standardization) of Child Measures and Age-, Age^2-, and Sex-Partialled Correlations between Transformed Child Measures.

		N	*M* (*SD*)	1	2	3	4	5	6	7
1	Communication	1,227	18.36 (6.01)	-						
2	Gross Motor	1,217	16.04 (5.21)	.31***	-					
3	Fine Motor	1,210	12.67 (4.29)	.41***	.36***	-				
4	Problem-Solving	1,199	12.57 (4.32)	.49***	.31***	.45***	-			
5	Personal-Social	1,198	11.75 (4.00)	.50***	.33***	.49***	.48***	-		
6	Social-Emotional	1,172	.16 (.79)	.33***	.23***	.32***	.32***	.34***	-	
7	Internalizing	809	41.33 (8.63)	-.16***	-.19***	-.20***	-.15***	-.21***	-.47***	-
8	Externalizing	808	42.60 (9.02)	-.19***	-.15***	-.22***	-.20***	-.21***	-.52***	.60***

Note. *N* for each variable represents the number of observations available on that particular variable. ***$p < .001$.

Table 17c. Age-, Age^2-, and Sex-Partialled Correlations between Transformed Parenting and Child Measures.

	Communica-tion	Gross Motor	Fine Motor	Problem -Solving	Personal -Social	Social -Emotional	Internaliz -ing	Externaliz -ing
Daily Stimulating Interactions	.26***	.21***	.22***	.23***	.20***	.16***	-.09*	-.13***
Learning Activities	.15***	.13***	.16***	.16***	.13***	.15***	-.15***	-.19***
Supporting Positive Behavior	.22***	.22***	.21***	.22***	.29***	.33***	-.23***	-.27***
Emotional Support	.21***	.17***	.11***	.24***	.22***	.27***	-.11**	-.16***
Setting Limits	.17***	.18***	.20***	.20***	.20***	.34***	-.22***	-.32***
Proactive Parenting	.24***	.17***	.19***	.23***	.23***	.32***	-.20***	-.24***
Laxness-Consistent Parenting	-.12***	-.08*	-.14***	-.21***	-.17***	-.25***	.18***	.15***
Emotional Magnifi-cation	-.09**	-.08*	-.12***	-.15***	-.10**	-.23***	.19***	.22***
Emotional Neglect	-.06*	.03	.04	-.03	.04	-.04	-.05	.06
Over-reactivity	-.07*	-.13***	-.05	-.03	-.09**	-.18***	.18***	.28***
Hostility	-.10**	-.10**	-.08**	-.07*	-.09**	-.19***	.17***	.22***

Note. *$p < .05$. **$p < .01$. ***$p < .001$.

Hierarchical Structure of Child Measures

Both theoretical and empirical work supported at least a modest association between cognitive ability and behavioral problems (e.g., Brunnekreef et al., 2007; Bub, McCartney, & Willett, 2007; Campbell, 1995, 2006; Koenen, Caspi, Moffitt, Rijsdijk, & Taylor, 2006; Lynam & Henry, 2001; Masten et al., 2005; Nigg & Huang-Pollock, 2003), some of which examined this association among children as young as 24 months old. Given this link between child cognitive and behavioral development, in this study, eight child outcomes were broadly divided into 2 clusters (Model C1; see Figure 10): 1) *Broad Ability*, which was defined by *Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, *Personal-Social*, and *Social-Emotional*; and 2) *Problem Behavior*, which was defined by *Social-Emotional*, *Internalizing*, and *Externalizing*. *Broad Ability* represented children's general developmental status across major areas indicative of young children's physical, psychological, and neurological development, whereas *Problem Behavior* represented children's behavioral and emotional difficulties that might hinder adaptive functioning and development of meaningful relationships with others. *Social-Emotional* loaded on both *Broad Ability* and *Problem Behavior* as it assessed regulatory skills (a type of cognitive functioning) and emotional fluctuations as well as behavioral impulses. Model fit statistics suggested that this model fit our data well, χ^2 (18, N = 1,245) = 32.62, p = .02, MLR scaling = .97, AIC = 22,804.32, BIC = 22,937.62, RMSEA = .03, CFI = .99, TLI = .99.

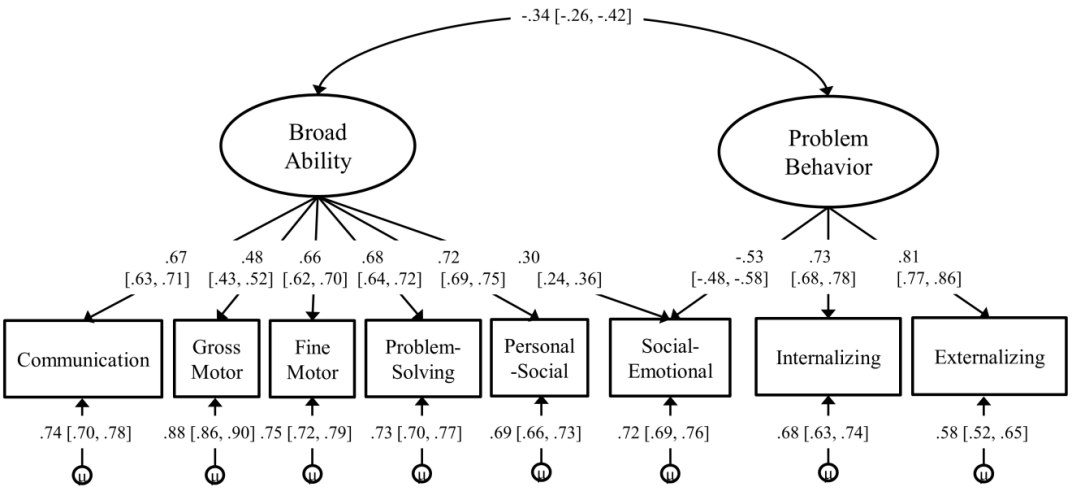

Figure 10. Phenotypic Model of Child Measures Grouped by Early Ability and Emotional-Behavioral Maladjustment (Model C1). All coefficients shown are standardized estimates. Confidence intervals are listed in brackets following each parameter estimate. All estimates are significant at $p < .001$.

When examining child outcomes during first years of life, another common approach is to categorize child measures by major domains of developmental milestones (e.g., Dyck, Piek, Kane, & Patrick, 2009; Hair, Halle, Terry-Humen, Lavelle, & Calkins, 2006; Howard, 2007; Sharma, O'Sullivan, & Baird, 2008). The eight child outcomes in this study were grouped into four different domains (Model C2; see Figure 11): 1) *Cognitive Development* – defined by *Problem-Solving* and *Personal-Social*, both of which assessed learning of patterns and application of learned rules in daily functioning; 2) *Social-Emotional Development* – defined by *Personal-Social*, *Internalizing*, *Externalizing*, and *Social-Emotional*, all of which assessed emotional and behavioral regulatory skills and risk for further maladjustment; 3) *Language Development* – defined by *Social-Emotional*, which included assessment of language skills in relating to others and expressing oneself appropriately, and *Communication*, which assessed expressive and receptive language skills; and 4) *Physical Development* – defined by *Gross Motor* and

Fine Motor, both of which assessed coordination of muscle groups or body parts. Model fit statistics suggested that this model also fit our data well, χ^2 (12, N = 1,245) = 11.44, p = .49, MLR scaling = .99, AIC = 22,796.06, BIC = 22,960.12, RMSEA < .01, CFI = 1.00, TLI = 1.00. Results from chi-square goodness-of-fit comparison test indicate a significant loss of model fit with less model complexity (i.e., Model C1), $\Delta\chi^2$ = 21.80, Δdf = 6, p < .01. However, as seen in Figure 11, correlations among *Cognitive Development*, *Language Development*, and *Physical Development* ranged from .80 to .98; this indicated that these three domains of child development did not meaningfully differ from one another and that there appeared to be two major clusters of child measures. Thus, we considered Model C1 the preferred phenotypic model representing the hierarchical structure of these eight child measures.

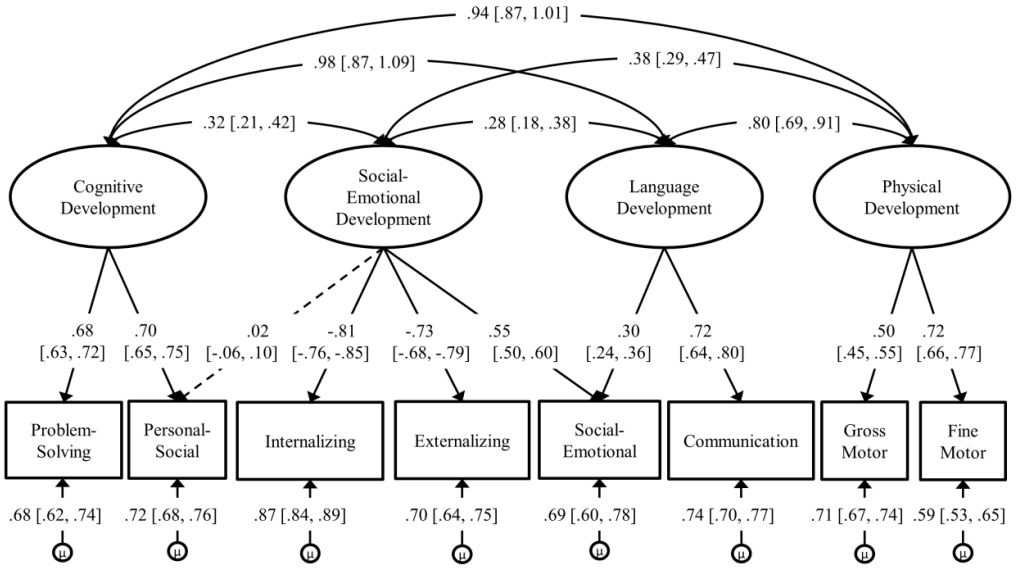

Figure 11. Phenotypic Model of Child Measures Grouped by Four Domains of Developmental Milestones (Model C2). All coefficients shown are standardized estimates. Confidence intervals are listed in brackets following each parameter estimate. All estimates are significant at p < .001, except for the factor loading of *Personal-Social* on *Social-Emotional Development*, which did not reach statistical significance (p = .62; path indicated by a dotted line).

Phenotypic links between parenting and early childhood phenotypes

We tested a series of multidimensional models to examine the association between parenting and child phenotypes at various dimensions. These models included the 11 parenting measures organized based on their theoretical impact on child development (see Study 1) and the 8 child measures categorized into overall functioning and emotional-behavioral difficulty (see Model C1). We first examined correlations between parenting and child phenotypes at the highest-order factor level only (Model P1). Model fit statistics suggested that this model fit our data reasonably well, χ^2 (140, N = 1,248) = 844.65, $p < .01$, MLR scaling = .84, AIC = 51,443.36, BIC = 51,797.28, RMSEA = .06, CFI = .91, TLI = .89. *Positive Parenting*, which included practices that were generally considered facilitative of adaptive child development, was associated with more advanced child development across major domains of functioning (i.e., *Broad Ability*; r = .44) and less emotional-behavioral maladjustment (i.e., *Problem Behavior*; r = -.44); at the same time, *Negative Parenting*, which included practices that were generally considered detrimental to child development, was associated with less advanced overall functional development (i.e., *Broad Ability*; r = -.17) and more emotional-behavioral difficulties (i.e., *Problem Behavior*; r = .41).

We next tested a model that included hypothesized correlations at the subordinate factor and measurement levels (Model P2). Child effect on parenting is commonly seen as parents modifying their childrearing approach to attain their desired outcome for their children's development (e.g., Bell, 1968; Belsky, 1984; Maccoby, 1992). Bell and Chapman (1986) summarized child characteristics that might have an impact on parenting into three main categories: 1) *dependent* children received more directive parenting, 2) *hyperactive* children received more controlling parenting, and 3) *person-oriented* children received more love-based parenting. The current study measured children's early skills

and emotional-behavioral problems, corresponding to the first two of the categories discussed in Bell and Chapman. Because children with less advanced skills might need more parental guidance on a given age-consistent task (i.e., more dependent), we hypothesized that variation in early skills, particularly those related to cognitive functioning, would be associated with the extent parents provided instruction, structure, and encouragement. Similarly, because children with more emotional and behavioral difficulties might elicit greater parental control or molding of child behavior, we hypothesized that variation in emotional or behavioral adjustment would be associated with the extent parents asserted rules, structures, and emotional responses. Table 18 lists the hypothesized correlations and the corresponding rationales. Model fit statistics suggested that this model fit our data reasonably well, χ^2 (127, N = 1,248) = 788.28, p < .01, MLR scaling = .84, AIC = 51,423.07, BIC = 51,843.68, RMSEA = .07, CFI = .92, TLI = .89.

Table 18. Hypothesized Correlations between Parenting and Child Phenotypes at Subordinate Factor and Measurement Levels.

Child Outcome	Element of Child Agency	Actions elicited from caregivers	Parenting Behavior
Communica-tion	receptive and expressive language ability	adjusting the amount and complexity of cognitive stimulating materials to a child's language ability to foster verbal and nonverbal concept learning	Cognitive Stimulation
		adjusting the complexity of rules to a child's language ability to facilitate learning of rules	Setting Limits
Problem-Solving	ability to comprehend and solve day-to-day quests	adjusting the complexity of cognitive stimulating activities to a child's comprehension level to foster effective learning	Cognitive Stimulation
		adjusting the predictability in a child's environment to a child's need for effective learning of rules and logic	Structured Parenting
		extrinsic and intrinsic positive reinforcement for performance meeting parental expectations	Support Positive Behavior
Personal-Social	ability to comprehend and adaptively approach personal-care and social situations	extrinsic and intrinsic positive reinforcement for performance meeting parental expectations	Support Positive Behavior
		adjusting the extent of consistent and elaborative instructions to a child's need to foster the understanding of norms and expectations	Setting Limits
		adjusting the amount of preparation needed for day-to-day tasks and activity transitions to foster the learning of techniques and procedures	Proactive Parenting
Social-Emotional	display of adaptive behavior in social and emotional situations	adjusting the extent of consistent and elaborative instructions to a child's need to foster the understanding of appropriate behaviors	Setting Limits
		adjusting the amount of preparation needed for day-to-day tasks and activity transitions to foster the learning of appropriate behaviors	Proactive Parenting
Externalizing	display of problem behavior that interfere with own and others' welfare	adjusting the extent of consistent and elaborative instructions to a child's need to foster the understanding of appropriate behaviors	Setting Limits
		adjusting the amount of preparation needed for day-to-day tasks and activity transitions to foster the learning of appropriate behaviors	Proactive Parenting
Problem Behavior	overall emotional and behavioral negativity	immediate and quick (though ineffective in a long-run) attempt to contain child's negativity	Maladaptive Emotional Socialization

When correlations between parenting and child phenotypes at subordinate factor and measurement levels were also estimated, those at the highest-order factor level remained consistent with those observed in Model P1. At the subordinate levels, we observed a correlation of .09 between *Cognitive Stimulation* at the latent factor level and *Communication* at the measurement level. This suggests that, in addition to the association between *Positive Parenting* and *Broad Ability*, greater degree of parental cognitive stimulation is specifically associated with more advanced communication skills. At the measurement level, *Setting Limits* was correlated with *Communication* at -.19, *Personal-Social* at -.17, and *Externalizing* at -.20. Although setting rules in a clear and non-coercive manner is considered a practice of positive parenting, which is associated with children's greater overall competence, results suggest that clear and non-coercive rule-setting is also specifically associated with less advanced child development in communication and personal-care as well as social skills. Moreover, above and beyond the association between positive parenting style and children's overall emotional or behavioral maladjustment, clear and non-coercive rule-setting was also specifically associated with less behavioral problems. These findings from Model P2 together illustrate that associations between parenting and child phenotypes are distributed across multiple dimensions of generality or specificity.

To identify other possible associations between specific parenting variables and child outcomes that are not tested on the basis of our a priori hypotheses, we examined the modification indices generated in Model P2 for associations that fit the general categories listed in Bell and Chapman (1986). *Problem-Solving*, an essential skill for children to be practically independent, might be associated with broad parenting styles beyond their associations with *Broad Ability*. Moreover, instead of the hypothesized association between *Problem-Solving* and *Structured Parenting*, *Problem-Solving* might

also be associated specifically with the quality of instructions received (i.e., *Setting Limits*) rather than general environmental structure that included predictability and consistency of parental guidance. Similarly, *Social-Emotional* in specific, instead of general child negativity, might be associated with ineffective response to children's display of negative emotions (i.e., *Maladaptive Emotinoal Socialization*). Modification indices from Model P2 also suggested that *Externalizing* might be associated with negative parenting style, possibly as a general tendency to exert control over children's disruptive behavior immediately even when it might not be effective in a long-run. Furthermore, *Problem Behavior* might be associated specifically with *Emotional Magnification* (i.e., parental mirroring of child's negativity; see Patterson, DeBaryshe, & Ramsey [1989] for a brief discussion on coercive family process) instead of general *Maladaptive Emotional Socialization*. Table 19 lists these post-hoc correlations identified from modification indices and the corresponding rationales. We tested a model with nonsignificant a priori hypothesized correlations dropped and these post-hoc correlations added (Model P3; see Figure 12). Model fit statistics suggested that this model fit our data reasonably well, χ^2 (130, N = 1,248) = 780.48, $p < .01$, MLR scaling = .84, AIC = 51,410.39, BIC = 51,815.61, RMSEA = .06, CFI = .92, TLI = .90.

Table 19. Post-hoc Correlations Added to the Phenotypic Model of Associations between Parenting and Child Phenotypes.

Child Outcome	Element of Child Agency	Actions Elicited from Caregivers	Parenting Behavior
Problem-Solving	ability to comprehend and solve day-to-day quests	adjusting general positivity (instead of specific acts of using elaborative guidance and positive reinforcement as hypothesized) to children's level of practical indepedence	Positive Parenting
		adjusting general negativity (e.g., as an immediate but ineffective strategy to impose independence) to children's level of practical indepedence	Negative Parenting
		adjusting the extent of consistent and elaborative instructions (instead of general predictability in a child's environment as hypothesized) to a child's need to foster effective learning of rules and logic	Setting Limits
Social-Emotional	display of adaptive behavior in social and emotional situations	immediate and quick (though ineffective in a long-run) attempt to induce children's self-regulation in social and emotional situations (instead of an attempt to contain child negativity in general)	Maladaptive Emotinoal Socialization
Externaliz-ing	display of problem behavior that interfere with own and others' welfare	general tendency to exert control over children's disruptive behavior immediately (though ineffective in a long-run)	Negative Parenting
Problem Behavior	overall emotional and behavioral negativity	maladaptively matching children's negativity (instead of general attempt to immediately contain child's negativity as hypothesized)	Emotional Magnification

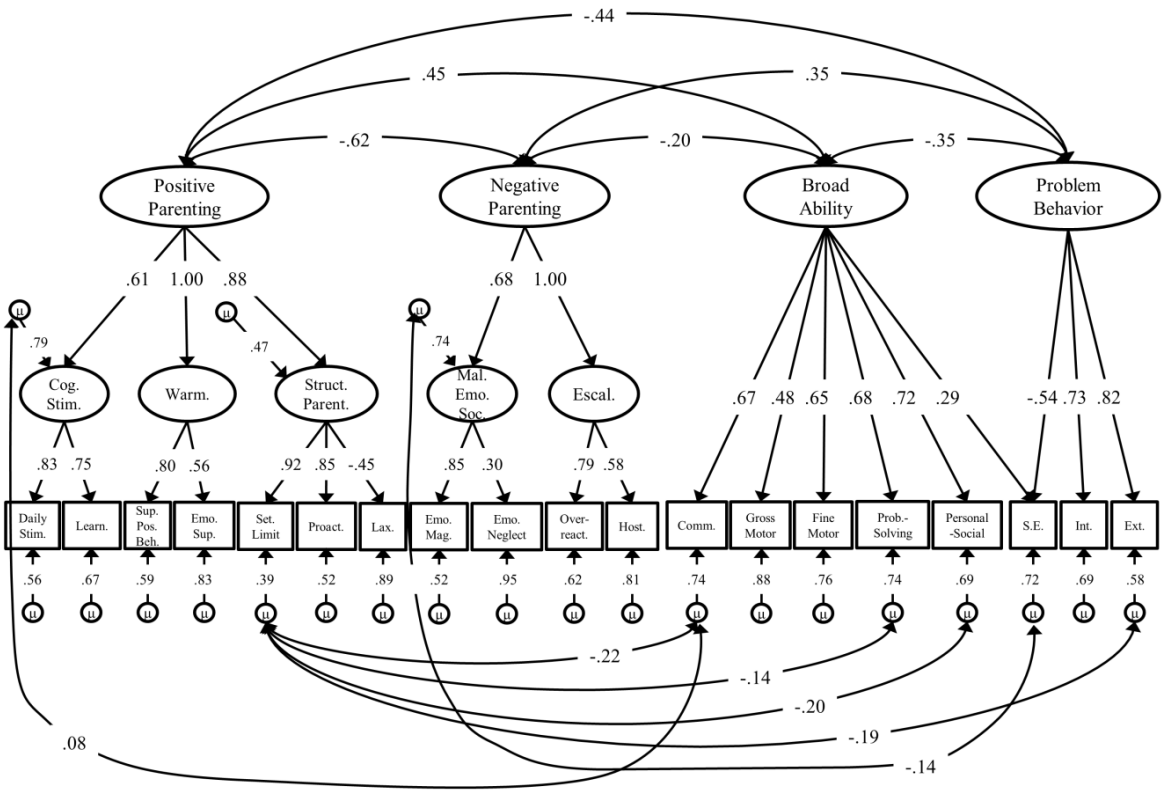

Figure 12. Preferred Phenotypic Model of Associations between Parenting and Early Childhood Phenotypes (Model P3). Cog. Stim. = Cognitive Stimulation. Warm. = Warmth. Struct. Parent. = Structured Parenting. Mal. Emo. Soc. = Maladaptive Emotional Socialization. Escal. = Escalation. Daily Stim. = Daily Stimulating Activities. Learn. = Formal Learning. Sup. Pos. Beh. = Supporting Positive Behavior. Emo. Sup. = Emotional Support. Set. Limit = Setting Limits. Proact. = Proactive Parenting. Lax. = Laxness-Consistent Parenting. Emo. Mag. = Emotional Magnification. Emo. Neglect = Emotional Neglect. Overreact. = Overreactivity. Host. = Hostility. Comm. = Communication. Prob.-Soling = Problem-Solving. S.E. = Social-Emotional. Int. = Internalizing. Ext. = Externalizing. Only paths that reached statistical significance ($p < .05$) are shown. All coefficients shown are standardized estimates.

Correlations that reached statistical significance in Model P1 and Model P2 remained the same in Model P3. Moreover, two post-hoc correlations reached statistical significance in Model P3, both of which appeared to be an alternative manifestation of a

hypothesized association that did not reach statistical significance in Model P2. First, although the correlation between *Maladaptive Emotional Socialization* and *Problem Behavior* did not reach statistical significance in Model P2, post-hoc correlation between *Maladaptive Emotional Socialization* and *Social-Emotional* did in Model P3 (r = -.14, p = .01); this suggests that caregivers are more likely to engage in maladaptive responses to children's expression of negative emotions toward children who are less competent in managing own emotions and behaviors (e.g., difficulty calming down quickly) rather than those who exhibit elevated emotional and behavioral problems. Second, whereas *Structure Parenting* was not significantly correlated with *Problem-Solving* in Model P2, the post-hoc correlation between *Setting Limits* and *Problem-Solving* reached statistical significance in Model P3 (r = -.14, $p < .01$); this suggests that children's development of reasoning skills may be associated specifically with caregivers' clear and non-coercive implementation of rules rather than parenting behaviors that are thought to generally increase the predictability of the childrearing environment.

Results from chi-square goodness-of-fit comparison tests indicated a significant loss of model fit when correlations at the subordinate levels were excluded (i.e., Model P1 fit our data worse than Model P2 and Model P3; $\Delta\chi^2 = 55.97$, $\Delta df = 13$, $p < .01$ and $\Delta\chi^2 = 64.26$, $\Delta df = 10$, $p < .01$, respectively). Model P2 and Model P3 (models with correlations at all levels) fit our data equivalently well ($\Delta\chi^2 = -7.93$, $\Delta df = 3$, $p > .05$). Model P3, being more parsimonious, was therefore considered the preferred phenotypic model (see Figure 12). Tables 20 and 21 list the standardized parameter estimates from all three phenotypic models.

Table 20. Standardized Factor Loadings Estimated (with Confidence Intervals in Brackets) in Phenotypic Models of Associations between Parenting and Child Phenotypes.

Measures	Factor	Model P1	Model P2	**Model P3**
Cognitive Stimulation	Positive Parenting	.62 [.57, .66]	.61 [.57, .65]	**.61 [.57, .65]**
Warmth		1.00 [>.99, <1.01]	1.00 [>.99, <1.01]	**1.00 [>.99, <1.01]**
Structured Parenting		.88 [.85, .90]	.88 [.85, .91]	**.88 [.85, .91]**
Maladaptive Emotional Socialization	Negative Parenting	.68 [.59, .78]	.68 [.59, .78]	**.68 [.59, .77]**
Escalation		1.00 [>.99, <1.01]	1.00 [>.99, <1.01]	**1.00 [>.99, <1.01]**
Daily Stimulating Activities	Cognitive Stimulation	.82 [.79, .86]	.83 [.80, .87]	**.83 [.79, .86]**
Formal Learning		.75 [.72, .79]	.74 [.71, .78]	**.75 [.71, .78]**
Support Positive Behavior	Warmth	.81 [.79, .84]	.81 [.78, .83]	**.80 [.78, .83]**
Emotional Support		.56 [.52, .60]	.56 [.52, .61]	**.56 [.52, .61]**
Setting Limits	Structured Parenting	.92 [.90, .93]	.92 [.89, .94]	**.92 [.90, .94]**
Proactive Parenting		.86 [.83, .88]	.86 [.83, .88]	**.85 [.83, .87]**
Laxness-Consistent Parenting		-.44 [-.49, -.39]	-.44 [-.49, -.39]	**-.45 [-.49, -.40]**
Emotional Magnification	Maladaptive Emotional Socialization	.87 [.76, .97]	.87 [.76, .98]	**.85 [.75, .96]**
Emotional Neglect		.30 [.24, .35]	.30 [.24, .35]	**.31 [.24, .36]**
Overreactivity	Escalation	.78 [.73, .82]	.78 [.73, .82]	**.79 [.74, .83]**
Hostility		.59 [.53, .64]	.59 [.54, .64]	**.58 [.53, .63]**
Communication	Broad Ability	.67 [.63, .71]	.67 [.63, .71]	**.67 [.63, .71]**
Gross Motor		.48 [.43, .52]	.48 [.43, .52]	**.48 [.43, .52]**
Fine Motor		.66 [.62, .69]	.65 [.61, .69]	**.65 [.61, .69]**
Problem-Solving		.68 [.65, .72]	.68 [.64, .72]	**.68 [.64, .72]**
Personal-Social		.72 [.69, .75]	.72 [.69, .75]	**.72 [.69, .75]**
Social-Emotional		.30 [.24, .36]	.30 [.24, .36]	**.29 [.23, .35]**
	Problem Behavior	-.53 [-.58, -.48]	-.53 [-.58, -.48]	**-.54 [-.59, -.49]**
Internalizing		.72 [.67, .77]	.72 [.67, .77]	**.73 [.68, .77]**
Externalizing		.83 [.78, .87]	.82 [.78, .86]	**.82 [.77, 86]**

Note. Bolded = preferred model. Model P1 = Model with only correlations at the highest-order factor level. Model P2 = Model with hypothesized correlations added at the subordinate levels. Model P3 = Model with post-hoc correlations added at the subordinate levels. All estimates are significant at $p < .001$.

Table 21. Correlations Estimated (with Confidence Intervals in Brackets) in Phenotypic Models of Associations between Parenting and Child Phenotypes.

Correlation between		Model P1	Model P2	**Model P3**
Positive Parenting	Negative Parenting	-.62 [-.67, -.57]	-.62 [-.67, -.57]	**-.62 [-.67, -.58]**
	Broad Ability	.44 [.38, .50]	.45 [.39, .52]	**.45 [.39, .51]**
	Problem Behavior	-.44 [-.50, -.38]	-.43 [-.50, -.37]	**-.44 [-.51, -.38]**
Negative Parenting	Broad Ability	-.17 [-.25, -.10]	-.19 [-.27, -.12]	**-.20 [-.28, -.12]**
	Problem Behavior	.41 [.35, .48]	.39 [.32, .45]	**.35 [.27, .42]**
Broad Ability	Problem Behavior	-.34 [-.41, -.26]	-.34 [-.42, -.27]	**-.35 [-.42, -.27]**
Maladaptive Emotional Socialization	Problem Behavior		(.04 [-.06, .13]) [a]	-
Cognitive Stimulation	Communication		.09 [.01, .17] [a]	**.08 [.01, .16]** [a]
	Problem-Solving		(.07 [-.01, .15]) [a]	
Structured Parenting	Problem-Solving		(-.05 [-.17, .08]) [a]	
Support	Problem-Solving		(-.05 [-.14, .04]) [a]	-
Positive Behavior	Personal-Social	-	(.06 [-.03, .14]) [a]	
Setting Limits	Communication		-.19 [-.28, -.10] [a]	**-.22 [-.32, -.13]** [a]
	Personal-Social		-.18 [-.31, -.04] [a]	**-.20 [-.31, -.09]** [a]
	Social-Emotional		(.07 [-.05, .18]) [a]	-
	Externalizing		-.20 [-.38, -.02] [a]	**-.19 [-.32, -.06]** [a]
Proactive Parenting	Personal-Social		(-.06 [-.16, .03]) [a]	
	Social-Emotional		(.04 [-.06, .13]) [a]	-
	Externalizing		(.04 [-.11, .19]) [a]	
Positive Parenting	Problem-Solving			**(.03 [-.03, .10])** [a]
Negative Parenting	Problem-Solving			**(.02 [-.05, .09])** [a]
	Externalizing			**(.09 [-.01, .19])** [a]
Maladaptive Emotional Socialization	Social-Emotional	-	-	**-.14 [-.24, -.04]** [a]
Emotional Magnification	Problem Behavior			**(.01 [-.10, .12])** [a]
Setting Limits	Problem-Solving			**-.14 [-.24, -.04]** [a]

Note. Bolded = preferred model. Model P1 = Model with only correlations at the highest-order factor level. Model P2 = Model with hypothesized correlations added at the subordinate levels. Model P3 = Model with post-hoc correlations added at the subordinate levels. Estimates in paratheses did not reach statistical significance ($p > .05$).
[a] Residaul correlations after accouting for the correlations via highest-order factors.

Behavioral genetic links between parenting and early childhood phenotypes

Having identified phenotypic associations between parenting and child outcomes at both general and specific levels, we then decomposed the variance in Model P1 (i.e., the phenotypic model with correlations estimated at the highest-order factor level only; Model BG1) and those in Model P3 (i.e., the preferred phenotypic model with correlations also estimated at subordinate factor and measurement levels; Model BG2) into biometric components *A*s, *C*s, and *E*s. Total variance of the highest-order factors, domain-specific (residual) variance at the subordinate factor level, and measure-specific (residual) variance at the measurement level were each constrained to be fully explained by 3 biometric components *A*, *C*, and *E*. Cross-twin correlation between corresponding *A*s for each factor or measure was fixed at 1 for MZ twins as they shared nearly all of their genetic material and at 0.5 for DZ twins as they shared approximately half of their segregating genetic material. Cross-twin correlation between corresponding *C*s for each factor or measure was fixed at 1 for all twin pairs as *C*s represent environmental influences shared by twins in a pair, whereas that between corresponding *E*s was fixed at 0 for all twin pairs because *E*s contribute to within-pair dissimilarities. Correlations between corresponding *A*s, *C*s, and *E*s of parenting and early child phenotypes were estimated to examine whether each phenotypic association observed was mediated by child genetic factors, shared child environmental factors, or nonshared child environmental factors, respectively.

Model fit statistics suggested that both models fit the data well (Model BG1: χ^2 [1421, N = 700] = 2692.05, $p < .01$, MLR scaling = 1.02, AIC = 44,270.40, BIC = 44,893.90, RMSEA = .05, CFI = .93, TLI = .93; Model BG2: χ^2 [1403, N = 700] = 2618.51, $p < .01$, MLR scaling = 1.02, AIC = 44,216.27, BIC = 44,921.69, RMSEA = .05, CFI = .93, TLI = .93). Results from chi-square goodness-of-fit comparison test

suggested that model fit significantly improved with the inclusion of correlations at subordinate factor and measurement levels ($\Delta\chi^2 = 61.29$, $\Delta df = 18$, $p < .01$). Tables 22 – 25 list the standardized parameter estimates from both behavioral genetic models. Estimates on factor loadings, domain- and measure-specific (residual) variance, and correlations at the highest-order factor level were generally consistent across Model BG1 and Model BG2; we focus our discussion on genetic and environmental correlations between parenting and child phenotypes observed in Model BG2 (see Figure 13).

Table 22. Standardized Factor Loadings Estimated (with Confidence Intervals in Brackets) in Behavioral Genetic Models of Associations between Parenting and Child Phenotypes.

Measures	Factor	Model BG1	**Model BG2**
Cognitive Stimulation	Positive Parenting	.57 [.48, .66]	**.56 [.48, .65]**
Warmth		.98 [.94, 1.01]	**.97 [.94, 1.01]**
Structured Parenting		.89 [.85, .93]	**.89 [.85, .93]**
Maladaptive Emotional Socialization	Negative Parenting	.73 [.60, .87]	**.72 [.57, .86]**
Escalation		.99 [.96, 1.02]	**.99 [.96, 1.02]**
Daily Stimulating Activities	Cognitive Stimulation	.85 [.79, .91]	**.85 [.79, .92]**
Formal Learning		.72 [.65, .78]	**.72 [.65, .78]**
Support Positive Behavior	Warmth	.82 [.77, .86]	**.81 [.77, .85]**
Emotional Support		.58 [.52, .64]	**.58 [.52, .65]**
Setting Limits	Structured Parenting	.91 [.88, .94]	**.92 [.89, .95]**
Proactive Parenting		.85 [.82, .88]	**.85 [.81, .88]**
Laxness-Consistent Parenting		-.44 [-.52, -.36]	**-.45 [-.53, -.38]**
Emotional Magnification	Maladaptive Emotional Socialization	.82 [.68, .96]	**.83 [.68, .99]**
Emotional Neglect		.28 [.21, .36]	**.28 [.19, .36]**
Overreactivity	Escalation	.76 [.69, .83]	**.76 [.69, .83]**
Hostility		.58 [.50, .65]	**.58 [.51, .65]**
Communication	Broad Ability	.67 [.62, .72]	**.63 [.57, .69]**
Gross Motor		.50 [.45, .55]	**.50 [.45, .55]**
Fine Motor		.67 [.63, .72]	**.68 [.63, .73]**
Problem-Solving		.70 [.65, .74]	**.68 [.63, .74]**
Personal-Social		.74 [.70, .78]	**.74 [.69, .78]**
Social-Emotional		.32 [.25, .38]	**.30 [.23, .36]**
	Problem Behavior	-.52 [-.58, -.47]	**-.52 [-.58, -.46]**
Internalizing		.72 [.67, .77]	**.73 [.68, .78]**
Externalizing		.83 [.79, .87]	**.83 [.79, .87]**

Note. Bolded = preferred model. Model BG1 = Behavioral genetic model with correlations only at the highest-order factor level. Model BG2 = Behavioral genetic model with correlations at the subordinate factor and measurement levels added. All estimates are significant at $p < .001$.

Table 23. Standardized Factor-Specific Genetic and Environmental Estimates (with Confidence Intervals in Brackets) from Behavioral Genetic Models of Associations between Parenting and Child Phenotypes.

	Model BG1			**Model BG2**		
	A	*C*	*E*	***A***	***C***	***E***
Positive Parenting	.14 [.08, .20]	.99 [.98, 1.00]	.07 [.04, .10]	**.14 [.07, .21]**	**.99 [.98, 1.00]**	**.07 [.04, .10]**
Negative Parenting	.12 [.03, .20]	.98 [.96, .99]	.18 [.13, .23]	**.11 [.01, .20]**	**.98 [.96, .99]**	**.19 [.14, .24]**
Broad Ability	.58 [.42, .75]	.79 [.67, .90]	.21 [.15, .27]	**.60 [.43, .77]**	**.77 [.65, .90]**	**.22 [.15, .28]**
Problem Behavior	`.69 [.53, .86]	.68 [.53, .83]	.25 [.10, .39]	**.69 [.50, .89]**	**.68 [.51, 84]**	**.24 [.07, 42]**
Cognitive Stimulation	.17 [.08, .26]	.80 [.74, .86]	(-.06 [-.03, .16])	**.17 [.08, .26]**	**.81 [.75, .87]**	**(.06 [-.04, .15])**
Warmth	(.18 [-.07, .42])	(<.01 [>-.01, <.01])	.14 [.01, .27]	**(.19 [-.05, .42])**	**(<.01 [>-.01, <.01])**	**.14 [.01, .27]**
Structured Parenting	.17 [.04, .31]	.43 [.35, .51]	(<.01 [>-.01, <.01])	**.17 [.03, .30]**	**.43 [.35, .52]**	**(<.01 [>-.01, <.01])**
Maladaptive Emotional Socialization	(.19 [-.14, .52])	.64 [.46, .82]	(.13 [-.12, .39])	**(.18 [-.19, .56])**	**.65 [.46, .85]**	**(.15 [-.08, .39])**
Escalation	(.16 [-.03, .34])	(<.01 [>-.01, <.01])	(<.01 [>-.01, <.01])	**(.14 [-.07, .35])**	**(<.01 [>-.01, <.01])**	**(<.01 [>-.01, <.01])**

Note. Bolded = preferred model. Model BG1 = Behavioral genetic model with correlations only at the highest-order factor level. Model BG2 = Behavioral genetic model with correlations at the subordinate factor and measurement levels added. Estimates in paratheses did not reach statistical significance ($p > .05$).

Table 24a. Standardized Genetic and Environmental Estimates unique to Parenting Measures (with Confidence Intervals in Brackets) from Behavioral Genetic Models of Associations between Parenting and Child Phenotypes.

	Model BG1			**Model BG2**		
	A	*C*	*E*	***A***	***C***	***E***
Daily Stimulating Activities	.17 [.09, .26]	.48 [.37, .59]	.13 [.09, .17]	**.17 [.08, .26]**	**.47 [.36, .58]**	**.14 [.10, .18]**
Formal Learning	(.12 [-.07, .30])	.66 [.59, .73]	.20 [.15, .24]	**(.12 [-.05, .29])**	**.66 [.59, .73]**	**.20 [.15, .24]**
Support Positive Behavior	.34 [.21, .47]	.42 [.32, .51]	.22 [.16, .29]	**.33 [.20, .46]**	**.43 [.34, .52]**	**.22 [.16, .29]**
Emotional Support	.34 [.16, .52]	.66 [.58, .74]	.33 [.26, .41]	**.34 [.16, .51]**	**.66 [.58, .74]**	**.34 [.26, .41]**
Setting Limits	(.16 [-.05, .36])	.29 [.19, .39]	.25 [.20, .30]	**.18 [.02, .33]**	**.25 [.14, .36]**	**.25 [.20, .29]**
Proactive Parenting	.21 [.07, .35]	.43 [.35, .50]	.22 [.17, .27]	**.22 [.08, .35]**	**.44 [.36, .51]**	**.22 [.17, .27]**
Laxness-Consistent Parenting	(<.01 [-.22, .22])	.84 [.80, .88]	.31 [.28, .34]	**(.02 [-3.05, 3.09])**	**.84 [.78, .90]**	**.31 [.23, .39]**
Emotional Magnification	(<.01 [>-.01, <.01])	.46 [.22, .71]	.33 [.25, .41]	**(<.01 [-.05, .05])**	**.45 [.17, .73]**	**.32 [.25, .40]**
Emotional Neglect	(.22 [-.01, .46])	.87 [.82, .92]	.34 [.27, .40]	**(.22 [-.03, .47])**	**.87 [.82, .93]**	**.34 [.27, .41]**
Overreactivity	.35 [.22, .48]	.50 [.38, .62]	.23 [.17, .29]	**.36 [.23, .48]**	**.49 [.37, .61]**	**.22 [.17, .28]**
Hostility	.33 [.08, .58]	.68 [.58, .79]	.30 [.22, .39]	**.33 [.08, .58]**	**.68 [.58, .79]**	**.30 [.22, .39]**

Note. Bolded = preferred model. Model BG1 = Behavioral genetic model with correlations only at the highest-order factor level. Model BG2 = Behavioral genetic model with correlations at the subordinate factor and measurement levels added. Estimates in paratheses did not reach statistical significance ($p > .05$).

Table 24b. Standardized Genetic and Environmental Estimates unique to Child Measures (with Confidence Intervals in Brackets) from Behavioral Genetic Models of Associations between Parenting and Child Phenotypes.

	Model BG1			**Model BG2**		
	A	*C*	*E*	***A***	***C***	***E***
Communication	.41 [.26, .56]	.50 [.40, .60]	.35 [.29, .42]	**.43 [.29, .57]**	**.54 [.44, .64]**	**.36 [.29, 42]**
Gross Motor	.62 [.43, .81]	.35 [.11, .58]	.50 [.40, .59]	**.62 [.43, .81]**	**.35 [.11, .58]**	**.50 [.40, .59]**
Fine Motor	(.21 [-.15, .57])	.56 [.47, .66]	.43 [.36, .51]	**(.20 [-.17, .58])**	**.56 [.46, .66]**	**.43 [.36, .51]**
Problem-Solving	.40 [.21, .60]	.45 [.32, .58]	.39 [.31, .48]	**.39 [.18, .59]**	**.48 [.35, .61]**	**.39 [.31, .48]**
Personal-Social	.39 [.28, .50]	.47 [.39, .56]	.28 [.23, .34]	**.38 [.26, .50]**	**.48 [.40, .57]**	**.28 [.23, .34]**
Social-Emotional	.43 [.27, .59]	.42 [.29, .54]	.39 [.33, .46]	**.44 [.26, .61]**	**.42 [.28, .56]**	**.40 [.32, .47]**
Internalizing	.39 [.26, .53]	(<.01 [>-.01, <.01])	.57 [.50, .65]	**.39 [.26, .52]**	**(<.01 [>-.01, <.01])**	**.57 [.49, .64]**
Externalizing	(<.01 [-.08, .08])	.24 [.10, .37]	.50 [.44, .56]	**(.12 [-1.52, 1.76])**	**(.22 [-.30, .74])**	**.50 [.34, .67]**

Note. Bolded = preferred model. Model BG1 = Behavioral genetic model with correlations only at the highest-order factor level. Model BG2 = Behavioral genetic model with correlations at the subordinate factor and measurement levels added. Estimates in paratheses did not reach statistical significance ($p > .05$).

Table 25a. Model-Estimated Genetic and Environmetal Corrleations between Parenting and Child Phenotypes at the Highest-Order Factor Level (with Confidence Intervals in Brackets) from Behavioral Genetic Models of Associations between Parenting and Child Phenotypes.

		Model BG1			**Model BG2**		
		rA	*rC*	*rE*	***rA***	***rC***	***rE***
Positive Parenting	Negative Parenting	-.61 [-.04, -1.18]	-.62 [-.53, -.71]	-1.00 [-.95,-1.04]	**(-.56 [-1.21, .10])**	**-.62 [-.54, -.71]**	**-.99 [-.91, -1.07]**
	Broad Ability	.53 [.18, .89]	.52 [.42, .61]	(.38 [-.10, .86])	**.61 [.19, 1.02]**	**.55 [.44, .65]**	**(.29 [-.19, .77])**
	Problem Behavior	-.97 [-.85,-1.08]	-.46 [-.33, -.59]	-.73 [-.17, -1.29]	**-.93 [-.54, -1.33]**	**-.47 [-.33, -.61]**	**(-.52 [-1.14, .09])**
Negative Parenting	Broad Ability	(.35 [-.25, .94])	-.21 [-.09, -.33]	(-.39 [-.78, <.01])	**(.32 [-.37, 1.00])**	**-.24 [-.12, -.37]**	**(-.36 [-.75, .02])**
	Problem Behavior	.79 [.43, 1.15]	.46 [.33, .60]	.79, [.35, 1.22]	**.80 [.38, 1.22]**	**.43 [.29, .57]**	**.64 [.19, 1.09]**
Broad Ability	Problem Behavior	-.30 [-.07, -.53]	-.36 [-.17, -.54]	(-.47 [-.96, .03])	**-.31 [-.07, -.55]**	**-.42 [-.21, -.63]**	**(-.43 [-.98, .12])**

Note. Bolded = Preferred model. Model BG1 = Behavioral genetic model with correlations only at the highest-order factor level. Model BG2 = Behavioral genetic model with correlations at the subordinate factor and measurement levels added. Estimates in paratheses did not reach statistical significance ($p > .05$).

Table 25b. Model-Estimated Genetic and Environmetal Corrleations between Parenting and Child Phenotypes at the Subordinate Factor and Measurement Levels (with Confidence Intervals in Brackets) from the Preferred Behavioral Genetic Model of Associations between Parenting and Early Childhood Phenotypes (Model BG2).

		rA[a]	*rC*[a]	*rE*[a]
Cognitive Stimulation	Communication	(-.28 [-.74, .19])	.18 [.04, .32]	(.43 [-.49, 1.35])
Maladaptive Emotional Socialization	Social-Emotional	(.36 [-1.61, 2.32])	(-.28 [-.59, .03])	(-.46 [-1.30, .39])
Setting Limits	Communication	(-.16 [-.45, .13])	-.40 [-.16, -.64]	(-.09 [-.27, .09])
	Problem-Solving	(.25 [-.38, .88])	-.62 [-.19, -1.05]	(.06 [-.15, .27])
	Personal-Social	(-.77 [-1.84, .31])	(-.17 [-.48, .15])	(.09 [-.16, .34])
	Externalizing	(-.88 [-12.65, 10.90])	(-.22 [-.96, .51])	-.25 [-.05, -.46]

Note. Estimates in paratheses did not reach statistical significance ($p > .05$). [a] Residual correlations after accouting for the correlations via highest-order factors.

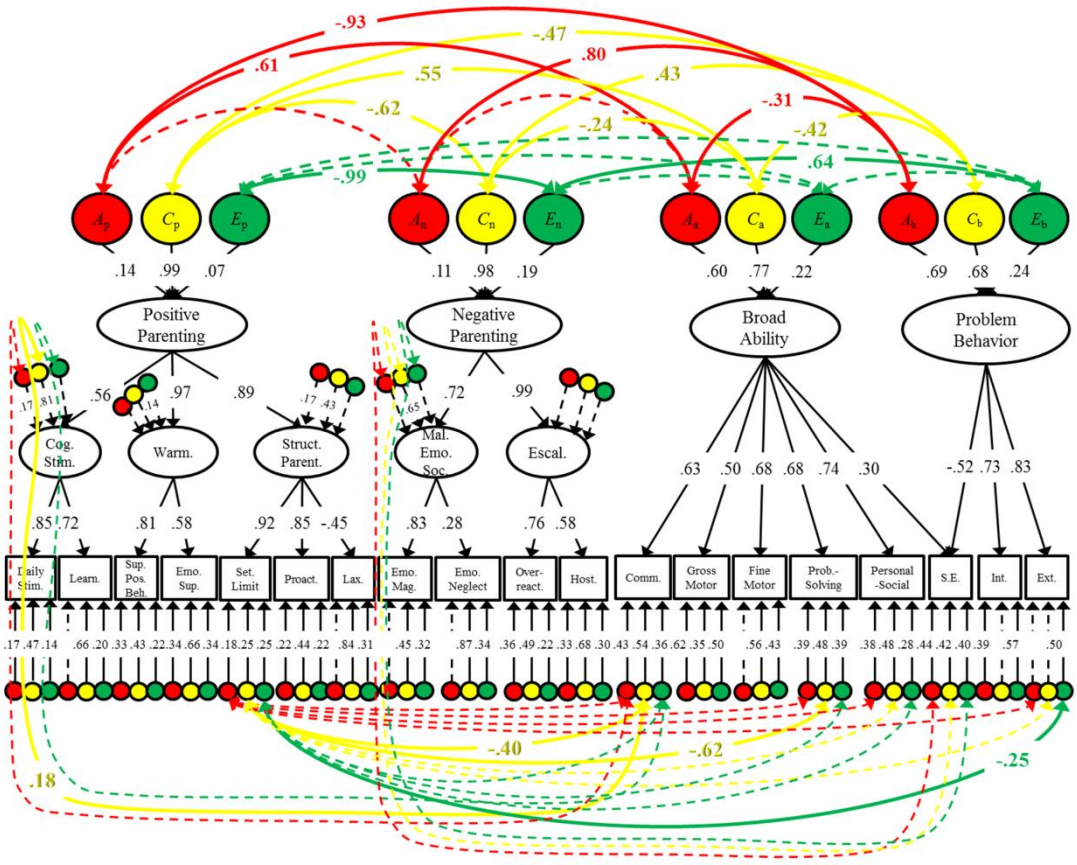

Figure 13. Preferred Behavioral Genetic Model of Associations between Parenting and Early Childhood Phenotypes (Model BG2). Cog. Stim. = Cognitive Stimulation. Warm. = Warmth. Struct. Parent. = Structured Parenting. Mal. Emo. Soc. = Maladaptive Emotional Socialization. Escal. = Escalation. Daily Stim. = Daily Stimulating Activities. Learn. = Formal Learning. Sup. Pos. Beh. = Supporting Positive Behavior. Emo. Sup. = Emotional Support. Set. Limit = Setting Limits. Proact. = Proactive Parenting. Lax. = Laxness-Consistent Parenting. Emo. Mag. = Emotional Magnification. Emo. Neglect = Emotional Neglect. Overreact. = Overreactivity. Host. = Hostility. Comm. = Communication. Prob.-Soling = Problem-Solving. S.E. = Social-Emotional. Int. = Internalizing. Ext. = Externalizing. Only the portion for one twin is shown for easy interpretation. Only paths that reached statistical significance ($p < .05$) are shown. All coefficients shown are standardized estimates.

Shared child environmental influences at the broadest dimension were large (c^2 = .46-.98) and moderately overlapped across parenting and child phenotypes ($|rC|$ =.24-.62). Results suggested that broad contextual and parental characteristics that fostered positive parenting also discouraged negative parenting, facilitated early development across multiple domains of functioning, and deterred the development emotional and behavioral difficulties; at the same time, broad contextual and parental characteristics contributing to negative parenting also impeded early development across multiple domains of functioning and contributed to more emotional and behavioral difficulties. Moreover, we observed a substantial amount of shared child environmental influences at the subordinate factor level ($c^2_{Cognitive\ Stimulation}$ = .65; $c^2_{Structured\ Parenting}$ = .19; $c^2_{Maladaptive\ Emotional\ Socialization}$ = .43) and the measurement level ($c^2_{parenting}$ = .06-76; $c^2_{child\ outcomes}$ = .12-.31). Unique of broad contextual and parental characteristics contributing broadly and simultaneously to positive parenting style and overall development of early abilities, there was another set of shared environmental factors that specifically prompted caregivers to provide more cognitive stimulation to their children and, at the same time, facilitated children's development of communication skills ($rC_{Cognitive\ Stimulation,Communication}$ = .18). Additionally, there was a moderate association between shared environmental factors that prompted caregivers' use of clear and non-coercive instruction and those that impeded children's development in communication ($rC_{Setting\ Limits,Communication}$ = -.40) as well as reasoning ($rC_{Setting\ Limits,Problem\text{-}Solving}$ = -.62).

Child genetic influences on children's general outcomes were moderate ($a^2_{Broad\ Ability}$ = .36; $a^2_{Problem\ Behavior}$ = .48), whereas those on broad parenting styles were much more modest ($a^2_{Positive\ Parenting}$ = .02; $a^2_{Negative\ Parenting}$ = .01). Results indicated large associations between child genetic influences on broad parenting styles and genetic risks for emotional-behavioral difficulties ($rA_{Positive\ Parenting,Problem\ Behavior}$ = -.93;

$rA_{Negative\ Parenting,Problem\ Behavior}$ = .80). At the same time, children with genetic dispositions for more advanced development of early abilities were more likely to receive positive parenting ($rA_{Positive\ Parenting,Broad\ Ability}$ = .61). There were nontrivial amounts of child genetic influences at the subordinate factor level ($a^2_{Cognitive\ Stimulation}$ = .03; $a^2_{Structured\ Parenting}$ = .03) and the measurement level ($a^2_{\text{parenting}}$ = .03-.13; $a^2_{\text{child outcomes}}$ = .15-.38). Yet, none of the genetic correlations between parenting and child phenotypes at the subordinate factor or measurement level reached statistical significance.

Nonshared child environmental influences were more modest both at the broad dimension ($e^2_{Positive\ Parenting}$ = <.01; $e^2_{Negative\ Parenting}$ = .03; $e^2_{Broad\ Ability}$ = .05; $e^2_{Problem\ Behavior}$ = .06) and at subordinate levels ($e^2_{\text{parenting}}$ = .02-12; $e^2_{\text{child outcomes}}$ = .08-.32). Nonetheless, results suggested that caregivers tended to engage in negative parenting toward children displaying more emotional-behavioral maladjustment due to nongenetic factors that were unique to a child, which might indicate parent-driven or child-driven processes ($rE_{Negative\ Parenting,Problem\ Behavior}$ = .64). At the measurement level, the correlated nonshared child environmental influences on *Setting Limits* and *Externalizing* (*rE* = -.25) suggested that caregivers were less likely to implement rules in a clear and non-coercive manner with a child 1) for idiosyncratic reasons such as less patience with this child and this differential treatment in turn contributed to the child's behavioral difficulties (i.e., parent-driven processes) or 2) in response to the child's higher level of behavioral difficulties that were driven by other environmental factors unique to this child such as having negative interactions with other family members (i.e., child-driven processes).

Tables 26 and 27 list the genetically- and environmentally-mediated correlations calculated using estimates from Model BG2. At the broadest dimension, parenting and early child outcomes were modestly to moderately correlated ($|r_{\text{phenotypic}}|$ = .18-.47). The correlation between *Positive Parenting* and *Broad Ability* and that between *Negative*

Parenting and *Broad Ability* was 88% and 84%, respectively, mediated by shared child environmental factors. Similarly, the correlation between *Positive Parenting* and *Problem Behavior* and that between *Negative Parenting* and *Problem Behavior* were both 76% mediated by shared child environmental factors. These results suggested that broad contextual and parental characteristics contributed the most to the association between general parenting style and children's overall development, suggesting strong parent-driven processes (see Figure 14). At the same time, we observed a nontrivial role of child genetic factors in explaining the association between *Positive Parenting* and *Broad Ability* (11%) and that between *Positive Parenting* and *Problem Behavior* (22%), suggesting genetically-based child-driven processes in differential positive parenting.

Table 26. Model-Estimated Genetically- and Environmetally-Mediated Corrleations between Parenting and Child Phenotypes at the Highest-Order Factor Level (with Confidence Intervals in Brackets) from the Preferred Behavioral Genetic Model of Associations between Parenting and Early Childhood Phenotypes (Model BG2).

			a-mediated	c-mediated	e-mediated
Positive Parenting	With	Negative Parenting	(-.01 [-.02, .01])	-.60 [-.68, -.52]	-.01 [-.02, -.01]
		Broad Ability	.05 [<.01, .10]	.42 [.33, .50]	(<.01 [>-.01, .01])
		Problem Behavior	-.09 [-.16, -.02]	-.32 [-.40, -.23]	(-.01 [-.02, <.01])
Negative Parenting		Broad Ability	(.02 [-.04, .08])	-.18 [-.29, -.08]	(-.01 [-.04, .01])
		Problem Behavior	(.06 [-.01, .13])	.29 [.19, .38]	(.03 [-.01, .07])
Broad Ability		Problem Behavior	(-.13 [-.47, .22])	-.22 [-.33, -.11]	(-.02 [-.20, .15])

Note. Estimates in paratheses did not reach statistical significance ($p > .05$).

Table 27. Model-Estimated Genetically- and Environmetally-Mediated Corrleations between Parenting and Child Phenotypes at the Subordinate Factor and Measurement Levels (with Confidence Intervals in Brackets) from the Preferred Behavioral Genetic Model of Associations between Parenting and Early Childhood Phenotypes (Model BG2).

Correlation between		via paths at the highest-order factor level			via paths at the subordinate factor or measurement levels		
		a-mediated	c-mediated	e-mediated	a-mediated	c-mediated	e-mediated
Cognitive Stimulation	Communication	(.02 [>-.01, .04])	.15 [.11, .19]	(<.01 [>-.01, <.01])	(-.02 [-.05, .01])	.08 [.02, .14]	(.01 [-.02, .03])
Maladaptive Emotional Socialization	Social-Emotional	(-.02 [-.32, .29])	(-.15 [-.41, .12])	(-.01 [-.31, .28])	(.03 [-.13, .19])	(-.08 [-.16, .01])	(-.03 [-.10, .04])
Setting Limits	Communication	(.03 [>-.01, .05])	.21 [.16, .26]	(<.01 [>-.01, .01])	(-.01 [-.04, .01])	-.05 [-.10, -.01]	(-.01 [-.02, .01])
	Problem-Solving	.03 [<.01, .06]	.23 [.18, .29]	(<.01 [>-.01, .01])	(.02 [-.03, .06])	-.08 [-.14, -.01]	(.01 [-.01, .03])
	Personal-Social	.03 [<.01, .06]	.25 [.19, .31]	(<.01 [>-.01, .01])	(-.05 [-.14, .03])	(-.02 [-.06, .02])	(.01 [-.01, .02])
	Externalizing	-.06 [-.11, -.02]	-.21 [-.27, -.16]	(-.01 [-.01, <.01])	(-.02 [-.05, .02])	(-.01 [-.04, .02])	-.03 [-.06, <.00]

Note. Estimates in paratheses did not reach statistical significance ($p > .05$).

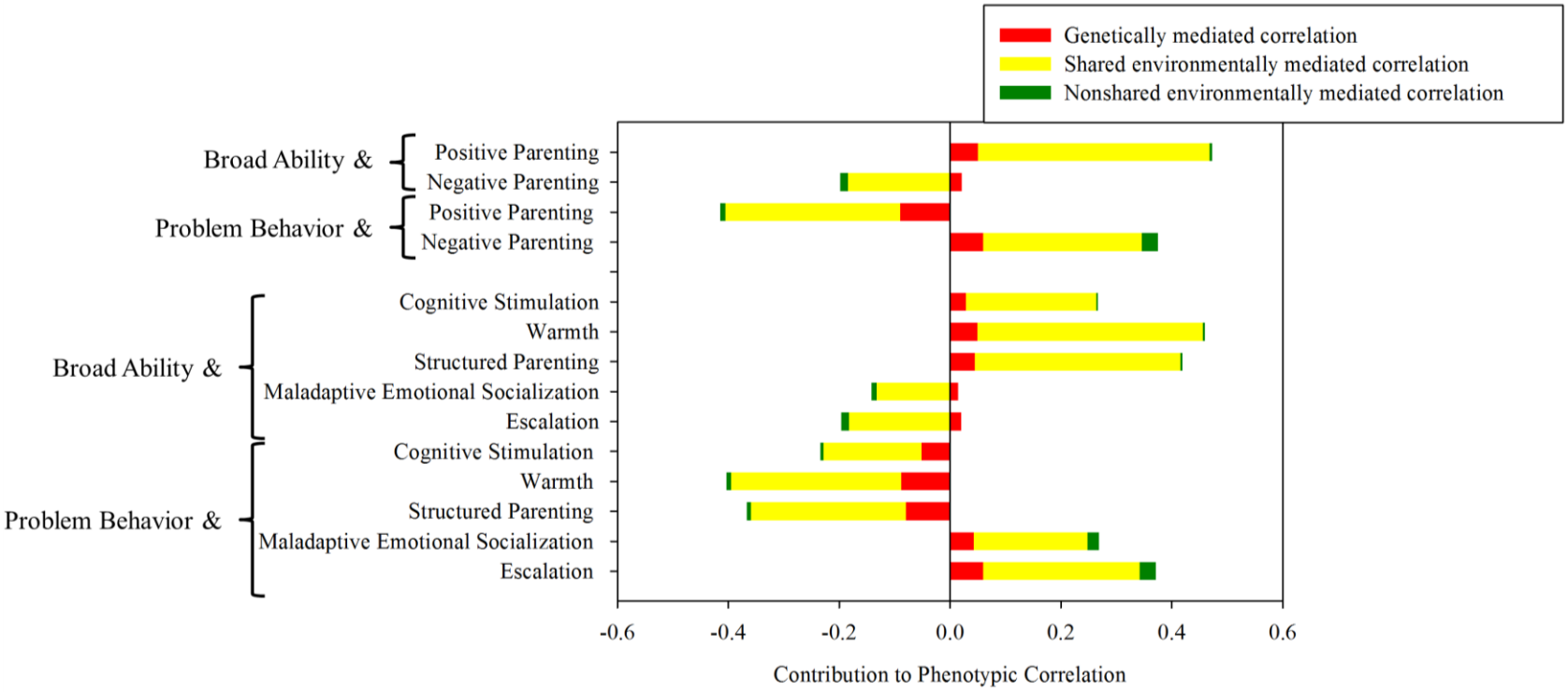

Figure 14. Model-Estimated Genetically and Environmentally Mediated Correlations between Broad Factors of Parenting and Early Child Outcomes. These correlations are based on estimates from the preferred behavioral genetic model (Model BG2).

Specific domains of parenting were also modestly to moderately correlated with general child functioning ($|r_{phenotypic}|$ = .13-.46). Each correlation was fully mediated by child genetic and environmental factors operating at the highest-order factor level. For example, the correlation between *Cognitive Stimulation* and *Broad Ability* was 88% mediated by shared child environmental factors that also explained 88% of the correlation between *Warmth* and *Broad Ability* and that between *Structured Parenting* and *Broad Ability*. Similarly, the correlation between *Maladaptive Emotional Socialization* and *Problem Behavior* was 76% mediated by shared child environmental factors that also explained 76% of the correlation between *Escalation* and *Problem Behavior*. These results suggest strong parent-driven processes that are common to multiple associations between different parenting domains and general child functioning.

We also observed modest but nontrivial child genetic mediations that were common to multiple associations between different domains of parenting and general child functioning. The correlation between *Cognitive Stimulation* and *Broad Ability* was 11% mediated by child genetic factors that also explained 11% of the correlation between *Warmth* and *Broad Ability* and that between *Structured Parenting* and *Broad Ability*. Likewise, the correlation between *Cognitive Stimulation* and *Problem Behavior* was 22% mediated by child genetic factors that also explained 22% of the correlation between *Warmth* and *Problem Behavior* and that between *Structured Parenting* and *Problem Behavior*. Additionally, the correlation between *Maladaptive Emotional Socialization* and *Problem Behavior* was 16% mediated by child genetic factors that also explained 16% of the correlation between *Escalation* and *Problem Behavior*. These indicated that child genetic influences on early child overall competence and those on emotional-behavioral maladjustment both contributed to variation in multiple distinct but related domains of parenting. Figure 14 shows the proportion of model-estimated phenotypic correlations

between broad parenting domains and general child functioning mediated by child genetic and environmental factors at the highest-order factor level.

Among the 6 pairs of parenting and child measures that were correlated at both broad and specific dimensions, model-estimated total phenotypic correlations were modest in size ($|r_{\text{phenotypic}}|$ = .17-.34) and mostly mediated by factors at the broadest dimension (see Table 27). The same set of shard child environmental factors that explained 54% of the correlation between *Cognitive Stimulation* and *Communication* also explained 68% of the correlation between *Setting Limits* and *Communication*, 64% of the correlation between *Setting Limits* and *Problem-Solving*, and 69% of the correlation between *Setting Limits* and *Personal-Social*. We also observed that the same set of child genetic factors mediating 8% of the correlation between *Setting Limits* and *Problem-Solving* also mediated 8% of that between *Setting Limits* and *Personal-Social*. The correlation between *Setting Limits* and *Externalizing* was 62% mediated by shared child environmental factors and 18% mediated by child genetic factors that explained the correlation between positive broad parenting style and general emotional-behavioral maladjustment.

Only 3 shared-environmentally-mediated correlations at the subordinate levels reached statistical significance – $r_{Cognitive\ Stimulation,Communication}$, $r_{Setting\ Limits,Communication}$, and $r_{Setting\ Limits,Problem\text{-}Solving}$ were 28%, 17%, and 21%, respectively, mediated by shared child environmental factors unique to each corresponding association. None of the genetically-mediated correlations at the specific dimensions reached statistical significance. The only association-specific nonshared-environmentally-mediated correlation that reached statistical significance was the one between *Setting Limits* and *Externalizing*, which explained 9% of their model-estimated total phenotypic correlation. In addition to suggesting strong parent-driven processes and modest genetically-based child-driven

processes, results suggest that much of an association between specific measures of parenting and early child outcome is attributed to child genetic and environmental factors that also explain the associations between other distinct but related constructs of parenting and early child outcomes.

DISCUSSION

Extensive theoretical and empirical evidence supports transactional associations between parenting and child development. However, the literature on the associations between parenting and child phenotypes has mostly been limited to specific measures. The current study employed a multidimensional approach to examining general and specific genetic and environmental associations between parenting and child cognitive, motor, social-emotional, and behavioral outcomes.

Results indicate that shared environmental influences, which represent the influences of broad contextual and parental characteristics on child outcomes, are substantial at all dimensions of parenting and early childhood phenotypes. Majority of each association between parenting and child functioning can be explained by socialization processes that also explain the associations between other distinct but related constructs of parenting and early child outcomes. Child environmental factors shared across siblings that contribute to various domains of positive parenting also facilitate early child development in overall functioning and protect against emotional-behavioral maladjustment, whereas those contributing to various domains of negative parenting also impede the development of early child functioning while contributing to emotional-behavioral maladjustment. Association-specific shared child environmental mediations were relatively modest and uncommon. At the subordinate level, we only observed shared child environmental mediation of the correlation between *Cognitive*

Stimulation and *Communication*, that between *Setting Limits* and *Problem-Solving*, and that between *Setting Limits* and *Personal-Social*. These results suggest that much of the association between parenting and early child outcome at various dimensions is attributed to broad contextual and parental characteristics affecting multiple domains of parenting and various aspects of early child development.

We also observed a large overlap between child genetic influences on broad parenting styles and those on children's overall development across multiple domains. Our results indicate that, when caregivers adjust their parenting behavior to early child outcomes, almost all of these child-driven processes are attributed to genetic factors shared across multiple developmental domains. Caregivers are more likely to engage in positive parenting toward children with genetic potential for more advanced development across domains of early abilities. At the same time, caregivers tend to engage less in positive parenting and more in negative parenting toward children with genetic risks for emotional-behavioral difficulties.

Despite the large and positive overlap between nonshared child environmental influences on *Negative Parenting* and those on *Problem Behavior*, the resulting nonshared-environmentally-mediated correlation did not reach statistical significance. At the measurement level, the only statistically significant nonshared-environmentally-mediated correlation was rather modest in size. In general, results suggest that individual differences in children driven by their unique experiences (including those initiated by the caregivers themselves) may be relatively less critical in explaining differential parenting across siblings.

Our study is the first to use a multidimensional approach to examine the complex associations between parenting and child development at the behavioral genetic level. Results indicate that child genetic and environmental mediation pathways overlap across

multiple associations between different parenting domains and various aspects of general child functioning. This means that associations between parenting and early child outcomes can be specified to occur through broad constructs. This complexity in associations between parenting and early child development would not be captured when using more conventional bivariate approaches. Through examining parent- and child-driven processes from a multidimensional perspective, we can identify the different patterns of socialization and child-driven differential parenting across measures or dimensions. Moreover, much of the residual genetic and environmental variation in the more specific constructs left unexplained in our models. It is possible that such child-level variation in specific parenting practices is associated with child characteristics not measured in this study.

Although these shared environmental correlations at the broadest dimension are in the expected direction, in which broad contextual and parental characteristics that contribute to positive parenting also contribute to more advanced overall functioning and less emotional-behavioral difficulties, there is a nontrivial amount of residual shared-environmentally-mediated correlations in the opposite direction at the more specific dimensions. Specifically, accounting for the positive correlation between *Positive Parenting* and *Broad Ability*, broad contextual and parental characteristics that uniquely encourage caregivers' use of clear and non-coercive rules (i.e., *Setting Limits*) actually impede children's development in receptive as well as expressive language skills (i.e., *Communication)* and reasoning skills (i.e., *Problem-Solving*). Whereas the use of clear and non-coercive instructions is a positive parenting skill, parent-initiated use of particularly detailed instructions may deprive children of the important learning opportunity to communicate and independently process information of varying complexity.

Limitations

It is important that our conclusions be considered in light of this study's limitations. First, caregivers reported their own parenting behaviors as well as their children's abilities and behaviors. To minimize potential biases such as self-enhancement or social desirability, caregivers were encouraged to complete their questionnaires in the privacy of their home and multiple physical and technical measures were taken to protect the confidentiality of their information. These procedures have been shown to effectively reduce biases such as social desirability in reporting personal behaviors (Richman, Kiesler, Weisband, & Drasgow, 1999). Moreover, most items in ASQ and ASQ:SE provide concrete examples or situations for caregivers to assess their children's performance in these specific tasks (e.g., *Does your child count up to 15 without making mistakes? If so, mark "yes." If your child counts to 12 without making mistakes, mark "sometimes."*). This approach reduces potential biases in caregivers' report due to subjective impressions.

Second, genetic and environmental estimates may vary across informants or assessment methods. For example, for both parenting and child outcomes, the use of parent-report tends to result in higher genetic estimates and lower nonshared environmental estimates than when using observational data (e.g., Avinun & Knafo, 2013; Burt, 2009; Klahr & Burt, 2014). Furthermore, caregivers' self-report on their parenting behaviors may result in higher shared environmental estimates than when using child-report as they may tend to report fair or similar treatment of their children for reasons such as social desirability. Although genetic and environmental estimates vary by informants or assessment methods, these estimates are generally nontrivial and statistically significant across measurement approaches (Avinun & Knafo, 2013; Burt, 2009; Klahr & Burt, 2014).

Third, being the first multidimensional study of genetic and environmental associations between parenting and early childhood phenotypes, we were constrained by the scarcity of existing multivariate literature to guide our hypotheses on correlations at the subordinate factor and measurement levels. In addition to correlations based on previous review of evidence for child effects on caregivers' behaviors, we tested more correlations at the subordinate factor and measurement levels after examining the modification indices generated in one of our phenotypic models. Identified correlations, particularly those at the subordinate levels, need replications to establish reliability of such associations above and beyond those operating at the broadest dimension. Importantly, the current study focused on the overall pattern of genetic and environmental links between parenting and early child outcomes within a hierarchical structure rather than significance testing of any given parameter.

CONCLUSION

The existing literature on the mutual association between parenting and early child outcomes has mostly been limited to bivariate findings. Using a multivariate behavioral genetic approach, we found that more than half of a given correlation between parenting and early childhood outcomes operated via family-level environmental factors that affected broad constructs and about one-fifth of it operated via child genetic factors common to multiple developmental domains. Child genetic influences were also observed at the more specific dimension of parenting but these genetic factors were not correlated with those operating on child outcomes. This suggests that caregivers may adjust their specific practices to children's genetically driven characteristics that are less outcome-oriented. Ongoing research in this area should combine multivariate methods, such as those employed here, with longitudinal data to distinguish the extent to which mutual

processes between parents and children occur through broad and specific dimensions, and whether such processes change with advancing age.

Chapter 4: General Summary

This dissertation is motivated by a model in which caregivers, in addition to exerting their influences on early child development via socialization, modify their parenting practices in response to their children's genetically driven characteristics. Substantial phenotypic and behavioral genetic evidence supports the mutual influences of parenting and early child development on one another. Yet, previous effort has focused on the associations between specific measures of parenting and child outcomes. This dissertation aimed to understand the broader picture of these complex associations between parenting and early child development. All three studies capitalized on a population-based genetically informative sample of young children and utilized a multivariate approach to examine the genetic and environmental links between parenting and early child development at both general and specific dimensions. Results from Study 1 suggest that caregivers, to some extent, modify their general parenting approaches and specific practices in response to different sets of genetically driven characteristics of their children. Study 2 presents evidence for age-related increases in genetic covariance across multiple domains of early abilities, providing additional support for the complex dynamics between a child's genetic dispositions and his or her environment during first years of life. Examining the genetic and environmental links between an array of parenting practices and child outcomes within a multidimensional structure, results from Study 3 suggest that most transactional associations between specific parenting and early child outcomes overlap and are mostly attributed to parent- and child-driven processes occurring at the broad dimensions.

These studies together highlight the complexity and dynamic nature of associations between parenting and early child development. Results presented in this

dissertation underscore the importance of examining the mutual influences of parenting and child development on one another from a multidimensional perspective. These results also demonstrate that these parent- and child-driven processes in parenting and child development occur as early as infancy.

Future research should examine these complex dynamics between parenting and child development at different life stages. As children grow older, they may become more active in molding and selecting environmental input of their choice (Scarr & McCartney, 1983). One may anticipate a reduction in child effect on parenting as children pursue more extra-familial experiences with age; at the same time, it is likely that such developmentally appropriate child behavior may prompt caregivers to continue to adjust their parenting approach, for example, from providing more hands-on guidance to more hands-off supportive monitoring.

The quality of parent-child relationship may also moderate the genetic and environmental links between parenting and child phenotypes. For example, in the context of a close relationship, a child may be more assertive in expressing their interests or needs and, at the same time, a caregiver may be more responsive to the child's requests and more likely to explain to the child in an open and non-coercive way when declining a request. Furthermore, the quality of parent-child relationship likely changes with child age as well; young children may experience a stronger need for approval by their caregivers, whereas adolescents have a stronger need for autonomy from their parents.

Parental characteristics may not only have a direct impact on parenting and child development but also moderate child influences on parenting. For example, there are likely individual differences in parental tolerance for negative child behaviors (Jenkins et al., 2015) and hence the extent to which parents may modify their behavior toward misbehaving children. Similarly, different caregivers may interpret a given child behavior

differently (Bell & Chapman 1986; Karraker & Coleman, 2005), which may then affect their subsequent approach to the child behavior. For example, a parent may perceive the nonstop crying of his or her baby as a signal of parental attention mismatching the baby's need and therefore continues to adjust one's behavior toward the baby; while another parent may perceive that as a nagging behavior and does not see a need to modify one's parenting behavior. Parental characteristics such as personality and mental or physical flexibility likely moderate the extent to which a child may affect a parent's behavior.

In conclusion, this dissertation highlights the importance of using a multidimensional approach to study parent- and child-driven processes during early childhood. Future research should also investigate the moderation effects of age, parent-child relationship, the interaction of the two, and parental characteristics on the genetic and environmental links between parenting and child phenotypes. Increasing emphasis and clarity on the mutual influences of parenting and child development on one another may help lighten the burden on parents as the sole agent in child development and, in turn, allow parents more mental capacities to engage in better parenting (Karraker & Coleman, 2005). As Putnam, Sanson, and Rothbart (2002) puts it, "any program giving prescriptions about 'the right way to do it' will clearly be deficient if it does not also direct parents' attention to individuality and to the need to be flexible in their approach to parenting" (p.270). Continued research effort in understanding the complexity in the mutual associations between parenting and child development across life stages may eventually inform the development of interventions and policies that target such dynamic feedback processes to foster more positive behavioral repertoires on the parts of both parents and young children.

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