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Early Childhood Cognitive Development and Parental Cognitive Stimulation: Evidence for Reciprocal Gene-Environment Transactions

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Abstract

Parenting is traditionally conceptualized as an exogenous environment that affects child development. However, children can also influence the quality of parenting that they receive. Using longitudinal data from 650 identical and fraternal twin pairs, we found that, controlling for cognitive ability at age 2 years, cognitive stimulation by parents (coded from video recorded behaviors during a dyadic task) at 2 years predicted subsequent reading ability at age 4 years. Moreover, controlling for cognitive stimulation at 2 years, children's cognitive ability at 2 years predicted the quality of stimulation received from their parents at 4 years. Genetic and environmental factors differentially contributed to these effects. Parenting influenced subsequent cognitive development through a family-level environmental pathway, whereas children's cognitive ability influenced subsequent parenting through a genetic pathway. These results suggest that genetic influences on cognitive development occur through a transactional process, in which genetic predispositions lead children to evoke cognitively stimulating experiences from their environments.

Keywords

Cognitive development; Gene-environment correlation; Cognitive stimulation; Parenting; Behavior genetics

"All the higher functions originate as actual relationships between individuals"

(Vygotsky, 1978, p. 57).

Developmentalists have long conceptualized children's learning and cognition as dependent on social interactions with adults, and have sought to understand specific parenting behaviors that maximize children's cognitive abilities. In particular, parental *cognitive stimulation*, defined as "parents' didactic efforts to enrich their children's cognitive and language development by engaging children in activities that promote learning and by offering language-rich environments to their children" (Lugo-Gil & Tamis-LeMonda, p. 1066), has been identified as a predictor of children's cognitive abilities (Berlin, Brooks-Gunn, Spiker, & Zaslow, 1995; Crosnoe, et al., 2010; Hubbs-Tait, McDonald, Culp, Culp, & Miller, 2002; Landry, Smith, & Swank, 2006; Landry, Smith, Swank, & Miller-Loncar, 2000; Smith, Landry, & Swank, 2000). Cognitive stimulation has also been of interest for researchers aiming to understand environmental (and potentially malleable) processes underlying socioeconomic disparities in children's cognitive outcomes. From an economic perspective, cognitive stimulation is one way in which parents invest financial and social

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resources in their children. Consistent with this perspective, parenting behaviors have been found to mediate the association between socioeconomic resources (e.g., poverty status, family income, maternal education) and children's cognitive ability and academic achievement (Duncan, Brooks-Gunn, & Klebanov, 1994; Garrett, Ng'andu, & Ferron, 1994; Lugo-Gil & Tamis-LeMonda, 2008; Yeung, Linver, & Brooks-Gunn, 2002). Thus, numerous studies of parenting behavior, rooted in socialization theories of cognitive development, have posited that parental cognitive stimulation is an important environmental determinant of children's cognitive abilities.

Historically, results from behavioral genetic research have been interpreted as a challenge to the importance of parenting and other socialization processes for children's cognitive abilities. Given that children's socialization experiences accumulate over time, one might anticipate environmental differences between families to account for increasing amounts of variance in cognitive outcomes as children age. However, research in developmental behavior genetics has consistently reported precisely the opposite pattern. Over the lifespan, the heritability of cognitive ability and academic achievement increases, while the contribution of between-family environmental differences appears to decrease (Bartels, Rietveld, van Baal, & Boomsma, 2002; Davis, Haworth, & Plomin, 2009; Fulker, Defries, & Plomin, 1988; McGue, Bouchard, Iacono, & Lykken, 1993; Petrill et al., 2004). By late adolescence, genetic differences between individuals account for more than 50% of the variance in important cognitive outcomes. Moreover, the longitudinal relations among genetic contributions to cognitive abilities across multiple ages tend to be very high (Bartels et al., 2002; Petrill, et al., 2004; Wadsworth, Corley, Hewitt, & DeFries, 2001), which suggests that increasing heritability over development largely represents an amplification of genetic variation that existed earlier, rather than the expression of new genes at later ages. A long-standing theoretical and empirical question, then, has been to understand how genetic potentials come to be realized – and amplified – over the course of child development.

In an attempt to reconcile research demonstrating the importance of parenting behaviors, on the one hand, and behavioral genetic results, on the other, previous authors have argued for an integrative approach that moves beyond a narrow conceptualization of genes and environments as independent forces, and towards more transactional models of human development (e.g., Bronfenbrenner & Ceci, 1994; Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Scarr, 1997; Scarr & McCartney, 1983). Central to transactional models is the concept of *gene-environment correlation (rGE)*, in which a child is systematically exposed to different environments as a function of his or her genotype (Scarr & McCartney, 1983). Evocative rGE, in which parenting behaviors are evoked by the child's own genetically influenced characteristics, may be a particularly important process in younger children, who cannot yet actively select environments for themselves. In this way, initial genetic differences lead children to become differentially exposed to learning environments (such as receiving high levels of cognitive stimulation by parents), which, in turn, causally affect children's cognitive skills. The net effect of this process is increasing phenotypic similarity between more genetically similar individuals: increasing heritability *through* environmental effects. This process was described in detail by Dickens and Flynn (2001), who proposed that "reciprocal causation produces a multiplier effect that inflates both genetic and environmental advantages by a process in which high IQ leads one into better environments causing still higher IQ, and so on" (p. 347). According to this argument, initial genetic differences may have very small effects, but these genetic differences are persistent over time, such that they systemically evoke high quality educational experiences from parents, peers, and educators. The result of this dynamic is an increasing "positive correlation between environment and genotype that masks the potency of environment."

There are two primary strands of research that provide support for the action of transactional processes. First, a large body of phenotypic research, originally synthesized over four decades ago in Bell's (1968) seminal paper, has clearly demonstrated that children's measured psychological characteristics predict the behavior of their caregivers (Crouter & Booth, 2003; Pardini, 2008). Of particular relevance for transactional models of children's cognitive development, Lugo-Gil and Tamis-LeMonda (2008) found bidirectional, cross-lagged associations between young children's cognitive abilities and parenting quality between the ages of 14 and 36 months. Even after accounting for previous levels of emotional support and cognitive stimulation, and for maternal economic and social resources, infants' cognitive abilities predicted future parenting. Second, previous behavioral genetic studies have found that a breadth of parenting behaviors do, in fact, reflect genetic differences between children, such that siblings experience more similar home environments with greater genetic relatedness (Dunn & Plomin, 1986; Elkins, McGue, & Iacono, 1997; Neiderhiser et al., 2004; Plomin, Reiss, Hetherington, & Howe, 1994; Rowe, 1981; Wade & Kendler, 2000; for reviews see McGuire, 2003; Plomin, 1994). For instance, Plomin et al. (1994) used a twin-sibling design to demonstrate that an average of over a quarter of the variation in indices of positive parenting, negative parenting, and parental monitoring was explained by genetic variation in the children they were raising. They noted that this finding appeared paradoxical because "environments have no DNA and thus cannot show genetic effects," (p. 32) but explained that heritability of environments could result from children selecting, modifying, creating, and evoking their environments based on their genetically influenced traits. Notably, however, there have been comparatively few studies that combine the above two strands of research, particularly for the study of cognitive development. That is, it is known that parenting behaviors are, generally speaking, influenced by children's characteristics, but no previous study has directly tested the cross-lagged bidirectional longitudinal association between children's cognitive ability and parenting behaviors using a genetically informative research design.

The current study focuses on early childhood as a period in development in which the cascade of gene-environment transactions is likely to begin. First, we examine the bidirectional phenotypic associations between parental cognitive stimulation and early cognitive ability in a sample of twins measured longitudinally at 2 years and 4 years. We predict that parenting behavior not only predicts future levels of cognitive ability, but that early cognitive ability also predicts ensuing changes in parenting behavior. Next, capitalizing on the varying degrees of genetic relatedness between identical and fraternal twins, we examine whether these bidirectional associations are mediated by genetic, family-level environmental, or unique environmental pathways. Consistent with theoretical models of gene-environment transaction, we predict that it is initial *genetic* differences in cognitive ability that predict future levels of parental cognitive stimulation (an evocative rGE). At the same time, we predict that parental cognitive stimulation predict future levels of child cognitive ability through environmental pathways. In other words, we predict that genetic influences on very early cognitive development lead children evoke stimulation of differing levels of quality from their parents, and that early levels stimulation of children by parents act as effectual environments in boosting their children's subsequent cognitive development.

Method

Participants

Data were drawn from the twin sample of the Early Childhood Longitudinal Study-Birth Cohort (ECLS-B; Snow et al., 2009), a nationally representative longitudinal study of children born in the United States in 2001, and followed through kindergarten entry. The current analyses are based on measures taken in 2003–2004, when the twins were approximately 2 years of age, and 2005–2006, when the twins were approximately 4 years

of age (preschool age). Data were available for approximately 1,300 twins (650 pairs).[1] Sixty-one percent of twins in the ECLS-B sample were White, 16% were African-American, 16% were Hispanic, 3% were Asian, 1% were Pacific Islander, American Indian, or Alaska Native, and 4% were multiracial. Fifty-one percent are male. Unlike many extant twin samples, which are drawn predominantly from the middle- to upper-classes, 25% of twin families lived below the poverty line at study entry.

Twin zygosity—During the 2-year wave, trained raters responded to six questions about same-sex twins regarding the similarity of their hair color, hair texture, complexion, facial appearance, and earlobe shape. Responses to each feature were coded as 1 (“no difference”), 2 (“slight difference”) or 3 (“clear difference”). Zygosity diagnoses based on physical similarity ratings have been consistently shown to be over 90% accurate when cross-validated against objective indices of zygosity, such as twin-pair genotyping (Forget-Dubois et al., 2003; Goldsmith, 1991; Price, Freeman, Craig, Petrill, Ebersole, & Plomin, 2000). Using the same procedure reported in Tucker-Drob et al. (2011), we summed scores for each pair to form a bimodal distribution of scores ranging from 6 to 18. Twin pairs with scores of 6, 7, or 8 were classified as monozygotic (MZ), and twin pairs with scores of 9 or above, along with opposite-sex twin pairs, were classified as dizygotic (DZ). Further, we eliminated same-sex pairs receiving a DZ diagnosis if their parents indicated that there was a medical reason for their dissimilarity. Our final working sample included 200 pairs of MZ twins and 450 pairs of DZ twins.

Measures

Bayley Scale (2 years)—During the 2-year wave, ECLS staff administered the Bayley Short Form-Research Edition (BSF-R), a shortened form of the Bayley Scales of Infant Development, Second Edition (Bayley, 1993). The BSF-R includes a mental scale and a motor scale. The current project made use of scores from the mental scale only. This scale is composed of 35 items that tap the quality of exploration of objects, early problem solving, the production of simple sound and gestures, and receptive and expressive communication with words. A two parameter (one parameter representing item difficulty, and one parameter representing item sensitivity) logistic item response theory model was applied to mental scale item responses from all children in the complete ECLS-B sample, and a mental scale score was then computed for each individual (for details see Andreassen & Fletcher, 2007). The reliability estimate for these mental scale scores was .88.

Early Readings Ability (4 years)—During the preschool wave children were directly measured on their early reading skills using a test composed of 37 multiple choice items representing the following content areas: receptive letter recognition, expressive letter recognition, letter sounds, recognition of simple words, phonological awareness, knowledge of print conventions, and matching words. A three parameter (one parameter representing item difficulty, one parameter representing item sensitivity, and one parameter accounting for probability of choosing the correct choice by guessing) logistic item response theory model was applied to all reading item responses from all children in the ECLS-B sample, and a reading score was then computed for each individual (for details see Najarian, Snow, Lennon, & Kinsey, 2010). The reliability estimate for this score was .84.

Parental Cognitive Stimulation (2 years and 4 years)—At both the 2-year wave and the 4-year wave, each twin participated separately in a 10-minute long videotaped semi-structured activity with his/her parent, called the *Two Bags Task*. The parent-child dyad was asked to play with two different sets of toys, each placed within a separate bag. Trained

[1]All sample sizes are rounded to the nearest 50 in accordance with ECLS-B data security regulations.

coders rated the videotaped interactions on a number of different dimensions using 7-point Likert-type scales adapted from Fauth, Brady-Smith, & Brooks-Gunn (2003). The current project used scores on *Parental Stimulation of Cognitive Development*, a scale that reflects the extent to which the parent demonstrates effortful teaching of the child to enhance cognitive, language, and perceptual development. The topic and method of teaching must be matched to both the child's developmental level and his or her level of interest in order for it to be rated as stimulating. The mean rating at 2 years was 4.13 (SD = 1.06), and the mean rating at 4 years was 4.31 (SD = 1.00). Inter-rater reliability of this rating was estimated at over 90% for both waves of data collection (Andreassen & Fletcher, 2007; Najarian, Snow, Lennon, & Kinsey, 2010).

Analyses

The first set of analyses tested the directionality of the association between parenting and early cognitive ability, using two regression models fit to longitudinal phenotypic data from one randomly selected twin per pair. The first regression model tested whether, controlling for Bayley scores at 2 years, parenting behavior at 2 years predicts early reading skills at 4 years. The second regression model tested the reciprocal association (Bayley scores at 2 years predicting parenting behavior at 4 years, controlling for baseline parenting behavior). In each model, the key parameter of interest was the cross-trait, cross-time regression coefficient, which can be used to infer the directionality of the association between parenting behavior and children's cognitive ability.

Next, we used data from both twins in each pair to estimate a series of behavioral genetic models. Following the conventions of the classical twin model, variance in each phenotype was decomposed into three components: additive genetic (*A*; correlated 1.0 in identical twins and 0.5 in fraternal twins), shared environmental (*C*, representing environmental influences that make twins similar to each other; correlated 1.0 in all twin types), and non-shared environmental (*E*, representing environmental influences that make twins less similar to each other, plus measurement error; uncorrelated across twins). The associations between the phenotypes were modeled using a Cholesky decomposition, in which each subsequent phenotype is regressed onto the *A*, *C*, and *E* components of all preceding phenotypes. In our parent→child model, we examined the cross-lagged association between variance in cognitive stimulation at age 2 years and subsequent reading ability at age 4 years, controlling for genetic and environmental variation in Bayley scores at 2 years. This allowed us to test the extent to which genetic and environmental differences in the quality of cognitive stimulation predict future reading. In particular, a significant shared environmental (*C*) pathway would be consistent with a socialization model. In our child→parent model, we examined the cross-lagged association between Bayley scores at age 2 years and subsequent levels of cognitive stimulation at age 4 years, controlling for genetic and environmental variation in cognitive stimulation at 2 years. This allowed us to test the extent to which genetic and environmental differences in children's early cognitive ability predict the quality of future parenting. In particular, a significant genetic (*A*) pathway would be consistent with an evocative gene-environment correlation, such that children with genetic predispositions for higher cognitive ability evoke greater cognitive stimulation from their parents.

Results

Phenotypic Evidence for Reciprocal Parent-Child Effects

Parameter estimates from the phenotypic regression models are summarized in Table 1. Two observations are of note. First, consistent with previous research, both cognitive ability and parenting behavior displayed significant longitudinal stability: Parent behavior at 2 years predicted parent behavior at 4 years, and Bayley scores at 2 years predicted reading skills at

4 years. Second, the cross-lagged associations indicated bidirectional effects between parents and children: Parenting behavior at 2 years predicted reading skills at 4 years, even after accounting for Bayley scores at 2 years, and Bayley scores at 2 years predicted parenting behavior at 4 years, even after accounting for parenting behavior at 2 years. These cross-lagged relations are highlighted in bold in Table 1.

Behavioral Genetic Model for Parent→Child Effects on Reading Ability at Age 4

Parameter estimates from the full behavioral genetic model of parent→child effects are presented in the top portion of Table 2, and parameter estimates from a reduced form of this model, in which only significant parameters were retained, are presented in the bottom portion of Table 2. As summarized in the top half of Table 3, the trimmed model fit the data as well as the full model. This model yielded four main results. First, Bayley scores at 2 years were primarily influenced by the shared environment (c^2 = 59%), but also influenced by genes (h^2 = 18%) and by the nonshared environment (e^2 = 23%). Second, the concurrent relation between Bayley scores and cognitive stimulation at 2 years was due entirely to shared environmental factors influencing both traits. Third, the stability between Bayley scores at 2 years and reading ability at 4 years was due to genetic and shared environmental factors. Fourth, and of greatest relevance, the association between cognitive stimulation at age 2 years and reading ability at age 4 years was entirely mediated by the shared environment (see the bolded parameter estimates in Table 2). This latter result suggests that parental stimulation affects children's cognitive development through a family-level process that equally affects both twins from a given pair. Figure 1 displays these latter findings. Dotted paths represent nonsignificant parameters, and solid lines represent significant parameters. The bolded pathway, with accompanying parameter estimates, represents the significant cross-lagged shared environmental association between parenting and cognitive development.

Behavioral Genetic Model for Child→Parent Effects on Cognitive Stimulation at Age 4

Parameter estimates for the behavioral genetic model of child→parent effects are presented in the top portion of Table 4, and parameter estimates from a reduced form of this model, in which only significant parameters were retained, are presented in the bottom portion of Table 4. As summarized in the bottom half of Table 3, the trimmed model fit the data as well as the full model. This model yielded three main results. First, consistent with the conceptualization of parenting as a family-level environment, most of the variance in cognitive stimulation at 2 years was due to environmental factors shared by twins (c^2 = 68%; h^2 = 7%, e^2 = 24%). Second, shared environmental factors were primarily responsible for the stability of cognitive stimulation between 2 years and 4 years. Third, and of greatest relevance, the relation between Bayley scores and later stimulation was entirely mediated by genetic variation (see the bolded parameters in Table 4). This latter result suggests that parents adjust the level of cognitive stimulation that they provide in response to their children's genetic predispositions for cognitive ability. In other words, genetic differences in early cognitive ability evoke differential levels of stimulation from parents. Figure 2 displays these latter findings. Dotted lines represent nonsignificant parameters, and solid lines represent significant parameters. The bolded pathway, with accompanying parameter estimates, represents the significant cross-lagged genetic association between early cognitive ability and parenting.

Discussion

The goal of this study was to test the predictions of a transactional model of cognitive development, in which initial genetic differences in children's cognitive ability evoke differential levels of cognitive stimulation from parents, while cognitive stimulation has an

environmental effect on children's future cognitive ability. Although transactional models of genetic predisposition and environmental experience have been suggested by numerous authors seeking to end the specious "nature vs. nurture" debate, and a volume of previous research has provided general support for both the bidirectionality of parent-child associations and for the existence of genetic differences in children's experiences with parenting, this study constituted the first direct test of gene-environment transactions in early childhood cognitive development using longitudinal, genetically informative data.

We found two main results. First, the longitudinal phenotypic associations between parental cognitive stimulation and child cognitive ability were reciprocal. Notably, the standardized regression coefficients from our phenotypic models were approximately equal, indicating that children's abilities predict their parents' future behavior as strongly as parents' behaviors predict their children's future abilities. Our study thus adds to a growing literature challenging the assumption that bidirectional parent-child associations are necessarily asymmetrical in favor of stronger parental influence (Pardini, 2008); across multiple domains, estimates of child→parent effects often equal or even exceed estimates of parent→child effects (e.g., Hipwell et al., 2008; Laird et al, 2003; Larsson, Viding, Rijsdjik, & Plomin, 2008). Second, our behavioral genetic models (illustrated in Figures 1 and 2) found that *genetic* differences in children's initial levels of cognitive ability predicted subsequent levels of cognitive stimulation by parents (even though genes accounted for a relatively small proportion of the overall variance in initial cognitive ability). At the same time, between-family *environmental* differences in cognitive stimulation predicted children's subsequent levels of reading ability. Thus, our results suggest that children's early environments may be indeed be the workhorses of cognitive development, but because exposure to these environments comes to be systematically correlated with genetic differences, environmental inputs ultimately amplify genetic variation.

One additional, null, result is also notable. Although there were substantial nonshared environmental influences on both parenting and cognition at 2 years and at 4 years, the nonshared environment played little, if any, role in the longitudinal relations across time points. That is, within MZ twin pair differences in cognition and parenting were not stable over time. Thus, our results indicate that while environmental influences that are unique to each twin may have large effects in the short-term, these non-shared experiences tend not to be recurring or systematic for an individual child over the course of development. This finding may help to explain why, even though unmeasured environments differentially experienced by children in the same family are typically estimated to have large effects (Plomin & Daniels 1987), attempts to identify large measurable nonshared environmental correlates of behavioral development have been unsuccessful (Turkheimer & Waldron, 2000). To the extent that nonshared environmental influences on development are temporally ephemeral and non-recurring, they may be effectively indistinguishable from random error of measurement (Dickens, Turkheimer, & Beam, 2011). Because environmental experiences that occur as functions of children's endogenous propensities may be more persistent and recurring than those that are exogenous (and hence less correlated with children's genotypes) they may have more systematic and lasting effects on development (Dickens & Flynn, 2001; Raine, Reynolds, Venables, & Mednick, 2002).

Two major strengths of the current study, which are comparatively rare in behavioral genetic research, are the use of a diverse and nationally representative sample of twins born in the United States, and the objective coding of parenting behavior observed during a dyadic task conducted separately with each twin, as opposed to self-reports of parenting behavior. However, the data analyzed were also limited in some respects. First, parenting and cognition data were only available for a relatively narrow period during early childhood: ages 2 years to 4 years. More longitudinal measurements over an extended age range would

be useful to examine how gene-environment transactions unfold over the entire span of child development. One might expect that, as parenting behaviors become less and less characteristics of the parents, and more and more a characteristics of children, the pathway through which experiences affect later learning would transition from a shared environmental pathway in early childhood to a genetic pathway in middle and late childhood. Second, our study only made use of one index of parenting quality, but cognitive stimulation is, of course, embedded in a larger matrix of parenting behaviors. For example, situational factors, such as having to work two jobs, could limit the amount of time that even "good" parents are able to spend with their children and thus reduce the amount of cognitive stimulation that children actually receive (Guryan, Hurst, & Kearney, 2008). Detailed information about how parents' time is spent in their day-to-day lives would be particularly useful in this respect. Third, while the current twin design was informative about the operation of children's genes, it was insufficient for making inferences about the operation of parents' genes. That is, while we found that parenting affected cognitive development through a family-level environmental pathway, our design was not capable of determining the extent to which parenting behaviors were themselves influenced by parents' genes.

In addition, the rapidity of cognitive change in early childhood has implications for how to best measure cognition at each age. Specifically, using the exact same measure of cognition for both 2-year-olds and 4-year-olds would be inappropriate. In other words, the Bayley test is an appropriate measure for 2 year olds, but would have been an inappropriate measure for 4 year olds, and similarly, a test of early reading is an appropriate measure for 4 year olds, but would have been an inappropriate measure for 2 year olds. Although the ability measured by the Bayley test and that measured by the reading test are conceptually distinguishable, past literature had indeed found a strong genetic link between general cognitive ability and multiple forms of achievement (Thompson, Detterman, & Plomin, 1991) suggesting that that the constructs are empirically similar. In the current study, Bayley scores at 2 years significantly predicted readings scores at 4 years, further supporting a relation between the two scales. Nevertheless, that repeated measures of the same form of cognition (e.g. general mental ability) were not available remains a limitation of the current study.

Although parent→child and child→parent influences were found to be equal in magnitude in this nationally representative sample of US-born twins (25% of whom were living below the poverty line), this result may not generalize to samples that include large proportions of children being raised under conditions of severe deprivation. For example, samples that include children being raised in orphanages and third world countries, in addition to children being raised in healthy-range environments, may contain substantially more heterogeneity in parenting quality, which could result in a standardized parent→child relation that exceeds the corresponding child→parent relation.

While the current study was concerned with documenting the pattern by which genes and environments come to be correlated over time, it did not specifically test whether genes and environments interact with one another to influence cognitive development. Belsky (2005) has hypothesized that differences in young children's genotypes may relate to differences in the extent to which they are susceptible to both positive and negative environmental inputs (such as parenting). Bronfenbrenner and Ceci (1994) have hypothesized that the realization of genetic potentials for healthy psychological development depends on environmental experience. Central to Bronfenbrenner and Ceci's framework is the concept of proximal processes, which they define as reciprocal interactions between the child and the caregiver that help to stimulate learning. Indeed, a number of studies (Friend, DeFries, & Olson, 2008; Harden, Turkheimer, & Loehlin, 2007; Rowe, Jacobson, & van den Oord, 1999; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003; Tucker-Drob et al., 2011)

have documented that the heritability of cognitive development is more pronounced in higher socioeconomic contexts, where proximal processes are presumed to be more abundant. An important direction for future research will be to examine whether the reciprocal gene-environment transactions documented in the current study vary as functions of macro-environmental contexts, such as socioeconomic status.

Finally, it is important to comment on the implications of the current findings for policy and intervention. Because we identified dyadic feedback processes between children and their parents, one possible implication is that early interventions for at-risk children may be most effective when they focus on both child functioning and parenting behaviors. As Huston et al. (2005) have commented, interventions could potentially have a “suntan” effect, whereby their benefits fade after exposure stops. They commented that a primary goal of intervention research is to identify interventions that work into person-environment feedback loops such that they are maintained or even amplified after exposure stops. The current findings suggest that early educational interventions may benefit from focusing on improving reciprocal interactions between the parent and child, rather than a more narrow focus on parent-to-child cognitive stimulation.

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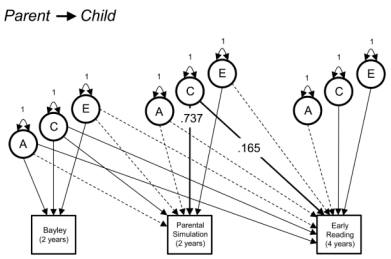

Figure 1.
Behavior Genetic Model of Parent → Child Effects on Reading Ability at 4 Years.
Note. Bolded paths represent significant cross-lagged associations.

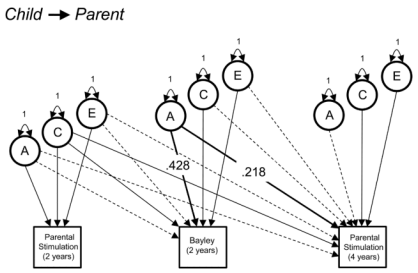

Figure 2.
Behavior Genetic Model of Child → Parent Effects on Cognitive Stimulation at 4 Years.
Note. Bolded paths represent significant cross-lagged associations.

Table 1

Standardized Parameter Estimates (and Standard Errors) from Phenotypic Regression Models.

	Outcome	
Predictor	**Early Reading(4 years)**	**Cognitive Stimulation(4 years)**
Parent → Child Regression Model		
Bayley(2 years)	.350 (.039)	
Cognitive Stimulation(2 years)	**.140 (.046)**	
Child → Parent Regression Model		
Bayley(2 years)		**.147 (.045)**
Cognitive Stimulation(2 years)		.230 (.048)

Note. All *P* values < .05. Model estimates using one twin per pair. Parameters representing the longitudinal cross-trait association are highlighted in bold font.

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Table 2

Standardized Parameter Estimates (and Standard Errors) from Behavioral Genetic Model of Parent→Child Effects on Reading Ability at Age 4.

	Bayley Scores (2 years)			Cognitive Stimulation (2 years)			Early Reading (4 years)		
	A	**C**	**E**	**A**	**C**	**E**	**A**	**C**	**E**
Full Model									
Bayley(2 years)	.420* (.076)	.769* (.033)	.481* (.027)						
Cognitive Stimulation(2 years)	.168 (.127)	.348* (.064)	.032 (.044)	.195 (.201)	.753* (.042)	.495* (.031)			
Reading (4 years)	.284* (.103)	.358* (.054)	.034 (.033)	**−.059 (.305)**	**.156* (.064)**	**−.018 (.036)**	.173 (.242)	.742* (.033)	.425* (.026)
Trimmed Model									
Bayley(2 years)	.427* (.063)	.767* (.030)	.479* (.024)						
Cognitive Stimulation(2 years)		.737* (.027)			.737* (.027)	.518* (.021)			
Reading (4 years)	.350* (.058)	.337* (.047)			**.165* (.048)**			.746* (.023)	.424* (.021)

Note. Rows = dependent variables; columns = predictor variables. Parameters representing cross-lagged associations are highlighted in bold.

*Parameter significant at $P < .05$.

Table 3

Fit Comparisons for Full versus Trimmed Behavioral Genetic Models.

Model	χ^2	*df*	*P*	CFI	TLI	AIC	BIC	RMSEA
Parent→Child Models								
Complete	27.38	33	0.74	1.00	1.00	7704.65	7799.17	0.00
Trimmed	**39.46**	**40**	**0.49**	**1.00**	**1.00**	**7702.72**	**7765.74**	**0.00**
Difference	12.07	7	>.05					
Child→Parent Models								
Complete	33.68	33	0.43	1.00	1.00	8628.81	8723.34	0.01
Trimmed	**46.79**	**40**	**0.21**	**0.99**	**1.00**	**8627.92**	**8690.93**	**0.02**
Difference	13.11	7	>.05					

Note. Preferred model is in bold.

Table 4

Standardized Parameter Estimates (and Standard Errors) from Behavioral Genetic Model of Child→Parent Effects on Cognitive Stimulation at Age 4

	Cognitive Stimulation (2 years)			Bayley Scores (2 years)			Cognitive Stimulation (4 years)		
	A	**C**	**E**	**A**	**C**	**E**	**A**	**C**	**E**
Full Model									
Cognitive Stimulation(2 years)	.305* (.102)	.818* (.030)	.487* (.028)						
Bayley (2 years)	.178 (.149)	.340* (.056)	.042 (.039)	.393* (.089)	.685* (.039)	.476* (.026)			
Cognitive Stimulation(4 years)	−0.128 (.148)	.343* (.062)	.075 (.045)	**.253* (.119)**	**.060 (.408)**	**−.014 (.754)**	.001 (.026)	.628* (.057)	.631* (.030)
Trimmed Model									
Cognitive Stimulation(2 years)	.267* (.134)	.827* (.033)	.494* (.031)						
Bayley (2 years)		.405* (.040)		.428* (.072)	.649* (.042)	.481* (.026)			
Cognitive Stimulation(4 years)		.325* (.043)		**.218* (.070)**				.655* (.030)	.646* (.023)

Note. Rows = dependent variables; columns = predictor variables. Parameters representing cross-lagged associations are highlighted in bold.

*Parameter significant at $P < .05$.