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SES and CHAOS as environmental mediators of cognitive ability: A longitudinal genetic analysis[★]

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Abstract

This study examined shared environmental influences on the longitudinal stability of general cognitive ability, as mediated by socioeconomic status and chaos in the home, using 287 pairs of elementary school-age twins drawn from the Western Reserve Reading Project (WRRP). General cognitive ability was evaluated at two annual assessments using the Stanford-Binet Intelligence Test. SES was examined using the highest level of education achieved by the mother of the twins, and chaos by a 6-item parent-report questionnaire. Results suggest that SES and CHAOS not only account for independent sources of shared environmental influences related to general cognitive ability at a given measurement occasion, but these effects also account for a portion of the longitudinal stability of general cognitive ability in early childhood.

Keywords

General cognitive ability; Twins; SES; CHAOS

1. Introduction

It is now commonly accepted that both genetic and environmental influences contribute to individual differences in general cognitive ability. On average, about 50% of the variance in intelligence is due to genetic differences, whereas shared environmental influences (factors that make family members more similar) account for around a quarter of the variance, with the remaining 25% of the variance accounted for by nonshared environmental influences and error (Jensen, 1981; Plomin, DeFries, McClearn, & McGuffin, 2001; Vernon, 1997). However, these estimates are not static. Genetic influences on intelligence increase as a function of age, starting at around 40% in childhood, to 60% in adulthood, to around 80% by old age (McGue, Bouchard, Iacono, & Lykken, 1993; Pederson, Plomin, Nesselroade, & McClearn, 1992). Conversely, shared environmental influences are most important in early childhood and decline to zero by adolescence; thus, studies have begun to identify measures of the environment that affect intellectual development early in life. Previous phenotypic research on cognitive development has consistently shown that deprived family environments (e.g., low quality of home environment, poor non-maternal care, low family income, low mother aptitude, etc.), can lead to increasingly poor cognitive functioning (Bradley & Caldwell, 1980; Carlson & Corcoran, 2001; Feinstein & Bynner, 2004; Neiss & Rowe, 2000; NICHD Early Child Care Research Network, 2001; Petrill, Lipton et al., 2004; Petrill, Pike, Price, & Plomin, 2004). These findings

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are consistent across age and ethnic groups (Bradley et al., 1989). More specifically, children from low-SES groups are more likely to experience academic problems, such as low test scores, increased course failure, and retaking of grades, as well as lower levels of educational attainment (Ellis & Bonin, 2003; Gutman, Sameroff, & Cole, 2003; Jimerson, Egeland, Sroufe, & Carlson, 2000; McLoyd, 1998; Rumberger, 1987).

Beyond SES, researchers have begun to look for other characteristics of the home and family environment that account for individual differences in cognitive functioning (van den Oord & Rowe, 1999). One such environmental mediator under recent study is the concept of “environmental confusion” (i.e., noise, crowding, and traffic within the home), which can be measured by a parental questionnaire entitled “The Confusion, Hubbub, and Order Scale” (CHAOS; Matheny, Wachs, Ludwig, & Phillips, 1995). Phenotypic studies have shown that adolescents from low income families experience a higher amount of environmental confusion at home, and this CHAOS mediates the SES effects of socioemotional development (Evans, Gonnella, Marcynyszyn, & Salpekar, 2005). Additionally, Dumas et al. (2005) showed that the CHAOS scale measures a construct that is distinct from known destructive social and psychological mediators (e.g., parenting stress, neighborhood attributes, etc.).

While these phenotypic studies have provided an important foundation, genetically sensitive designs have the power to extend our understanding of the etiology of the impact of measured aspects of the home environment on early cognitive development. In particular, Petrill, Pike et al. (2004) examined whether SES and CHAOS account for a proportion of the shared environmental variance associated with general cognitive ability in a longitudinal study of twins assessed at ages 3 and 4 years. SES and CHAOS mediated a small but significant portion of the shared environment of the cognitive measures at both ages. Specifically, the total measured environment accounted for about 6% of the total variance at ages 3 and 4 years for both verbal and nonverbal measures of cognitive ability. Also, this study showed that CHAOS predicted independent shared environmental influences on cognitive ability separate from SES, and therefore influenced a separate portion of the shared environmental variance of the cognitive measures. Furthermore, this study showed that SES and CHAOS contributed to stability in cognitive performance from ages 3 to 4 years.

However, results in Petrill, Pike et al. (2004) were based on the parent-report component of the Twins Early Development Study (TEDS), where all data (CHAOS, SES, and cognitive ability) was obtained from parent-report questionnaires. Thus, it is unclear whether the shared environmental overlap between SES, CHAOS, and cognitive ability was a function of “true” shared environmental influences or a function of the fact that all measures came from the same informant (generally the mother). Moreover, it is unclear whether the effects of SES and, in particular, CHAOS are generalizable beyond the very young age of the TEDS sample.

For this reason, the goal of the current study was to examine the links between SES, CHAOS and cognitive ability in a sample of kindergarten and 1st grade twins using the Western Reserve Reading Project (WRRP; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, 2006). The current study has three primary goals. First, we examine whether SES and CHAOS account for a significant portion of the shared environmental variance that affects general cognitive ability. Second, we examine whether SES and CHAOS account for a portion of the longitudinal stability in general cognitive ability. Finally, we test whether CHAOS explains a portion of shared environmental variance affecting cognitive ability independent from SES. Given Petrill, Pike et al. (2004) and the larger literature, we hypothesize that the SES and CHAOS will each predict a significant and independent proportion of the shared environmental variance in cognitive ability, and the effects will be stable across the measured years.

2. Method

2.1. Participants

Participants were drawn from the Western Reserve Reading Project (WRRP), an ongoing study of 350 twin pairs. WRRP is a longitudinal genetically sensitive project looking at the development of reading and other cognitive skills across four annual assessments. The twins are from the Greater Cleveland, Columbus and Cincinnati metropolitan areas, with a small minority coming from other areas of Ohio and Western Pennsylvania. The twins were recruited primarily through school nominations ($n=273$ schools, resulting in $>80\%$ of subsequently enrolled families). These schools were asked to send an information package to all parents of twins who had entered kindergarten but have not yet finished the first grade. The information to the parents included a letter and brochure discussing the study, as well as a stamped postcard of interest. All parents that returned the postcards were contacted by phone, and if interested, sent an additional information packet. This packet consisted of a demographic questionnaire asking for additional contact information, names and ages of twins and other children living at home, parental education, parental occupation, and ethnicity. Families were considered enrolled in the Project once this packet had been returned. All other families were recruited via Ohio State birth records, mother of twin clubs, and media advertisement. Zygosity was determined using DNA analysis via a cheek swab. For the handful of cases where parents did not consent to genotyping, zygosity was determined using a parent questionnaire on the twins' physical similarity (Goldsmith, 1991). Although somewhat skewed, parent education levels varied widely and was similar for fathers and mothers: 12% high school or less, 18% some college, 30% bachelor's degree, 24% some post-graduate education or degree, 5% not specified. Most families were two-parent households (6% single mothers) and nearly all were White (91%). Almost all the two-parent families represented intact families, in that both parents were biologically related to the twins (99%).

All twins were in kindergarten or first grade in wave 1, and in the first or second grade in wave 2. Complete data from 123 MZ pairs and 164 same-sex DZ pairs for wave 1 Stanford-Binet SAS was available (age $M=6.0$ yrs, $SD=.70$ yrs, range=4.3–7.9 yrs). Concurrently, 88 MZ twin pairs and 123 same-sex DZ twin pairs from wave 2 had complete data at time of analysis (age $M=7.2$ yrs, $SD=.67$ yrs, range=6.0–8.8 yrs).

3. Material

3.1. Socioeconomic status

Socioeconomic status (SES) was measured using mothers' highest level of education reported in the initial demographic questionnaire. This was a scale from 1–9 (1=less than high school, 9=post-graduate degree).

3.2. Chaos

Chaos in the home was measured via maternal questionnaire in both waves 1 and 2. This questionnaire was derived from the "Confusion, Hubbub, and Order Scale" (CHAOS; Matheny et al., 1995; Petrill, Pike et al., 2004). The questionnaire consisted of six questions concerning the level of chaos in the home, reported on a five-point likert scale (1="definitely untrue", 5="definitely true"). Sample questions include "It's a real zoo in our home" and "The atmosphere in our house is calm" (see Table 1 for full questionnaire). A composite CHAOS score was created by taking the mean of the items, identical to what was done in the Petrill, Pike et al. (2004) study. Inter-item reliability of the CHAOS scale for our study was moderate ($\alpha=.68$). Although only mother's report of CHAOS is used for the present analyses, father's report on the measure was also collected for 137 families. Therefore, using these data, inter-rater reliability was shown to be moderate ($r=.77$).

3.3. General cognitive ability

General cognitive ability was assessed using a short form of the Stanford-Binet Intelligence Scale (Thorndike, Hagen, & Sattler, 1986a), including tests of Vocabulary, Pattern Analysis, Memory for Sentences, Memory for Digits, and Quantitative Subtests. These subtests were summed and standardized to form the Composite Summary of Area Score (SAS), forming a measure with a mean of 100 and a standard deviation of fifteen. Published reliability of SAS scores for the abbreviated battery at the age 7 is high ($\alpha = .95$; Thorndike, Hagen, & Sattler, 1986b).

4. Results

4.1. Phenotypic analysis

Means and standard deviations for SES, wave 1 and wave 2 CHAOS, and wave 1 and wave 2 Stanford-Binet SAS are presented in Table 2. Pearson correlations between wave 1 and wave 2 Stanford-Binet SAS, SES, and wave 1 and 2 CHAOS, are presented in Table 3. Wave 1 and wave 2 CHAOS were reverse coded so that all variables were in the same direction (high score=positive). Wave 1 Stanford-Binet SAS was highly correlated to wave 2 Stanford-Binet SAS ($r(409) = .72, p < .01$). Similarly, wave 1 and wave 2 CHAOS were highly correlated to each other ($r(364) = .81, p < .01$). SES and both waves of CHAOS were minimally correlated to one another, $r(530) = .21$ and $r(372) = .21$, respectively. SES and CHAOS are also minimally correlated with Stanford Binet SAS ($r = .17-.24$ between SES and Stanford-Binet SAS, and $r = .27-.29$ between CHAOS and Stanford-Binet SAS).

4.2. Quantitative genetic analysis

4.2.1. Intraclass and cross-twin correlations—Table 4 presents twin intraclass correlations for the wave 1 and wave 2 Stanford-Binet SAS scores. If the MZ twin correlation exceeds the DZ twin correlation, then genetic influences are implicated (as MZ twins share 100% of their genes, whereas DZ twins share an average of 50% of their genes). Shared environmental effects are implicated if MZ and DZ correlations are similar. Finally, nonshared environmental influences (and error) are implicated to the extent that the MZ twin correlation is less than one. Table 4 presents trends for higher MZ versus DZ correlations, suggesting genetic influences. However, because the MZ correlations are not greater than two times the difference between the MZ and DZ correlations, there is also evidence for shared environmental influences. Cross-twin correlations (twin 2 wave 1 Stanford-Binet SAS with twin 1 wave 2 Stanford-Binet SAS) further suggest the importance of genetic influences on the longitudinal stability of general cognitive ability, as the MZ cross-correlations appear to be larger than the DZ cross-correlations ($r_{MZ} = .72$ versus $r_{DZ} = .48$).

4.2.2. Univariate genetic analyses—Going beyond estimates of genetic, shared environmental and nonshared environmental influences, the main goal of this study is to identify the proportion of the shared environment explained by measures of SES and CHAOS. Therefore, this study employs a model based on that of Petrill, Pike et al. (2004) and Neale, Boker, Xie, and Maes (1999). This univariate model measures genetic (A), shared environmental (C), and nonshared environmental (E) influences on Stanford-Binet SAS, as well as variance due to an “identified” measure of shared environment (i.e., SES and CHAOS; see Fig. 1). In all, four separate univariate analyses were conducted: (1) wave 1 Stanford-Binet SAS and SES; (2) wave 2 Stanford-Binet SAS and SES; (3) wave 1 Stanford-Binet SAS and wave 1 CHAOS; and (4) wave 2 Stanford-Binet SAS and wave 2 CHAOS. The models all used 95% confidence intervals to test for significance of parameter estimates. All available data were analyzed using raw data in Mx (Neale et al., 1999).

Univariate analyses suggest moderate heritability and shared environment for both wave 1 and wave 2 Stanford Binet SAS (see Table 5). Genetic influences accounted for between 27–51% of the total variance. Shared environmental influences not explained by SES and CHAOS accounted for between 16–40% of the variance. The nonshared environmental variance remained relatively stable across the waves for Stanford-Binet SAS. Both SES and CHAOS accounted for a significant portion of the total variance in Stanford-Binet SAS at both wave 1 and wave 2 (3–8%).

4.2.3. Multivariate genetic analysis—Following this univariate examination, multivariate analyses were conducted to determine how the measured shared environmental variables predict the longitudinal stability of Stanford-Binet SAS. First, a bivariate Cholesky decomposition was conducted to examine the genetic and environmental influences on the stability and instability of cognitive ability from wave 1 to wave 2 (see Fig. 2). This model parameterizes common genetic (represented as “A”), shared environmental (C) and nonshared environmental (E) variances in Stanford-Binet SAS scores across wave 1 and wave 2. Also, the model parameterizes specific genetic (a), shared environmental (c) and nonshared environmental influences on wave 2 Stanford-Binet SAS scores independent from wave 1.

Fig. 3 represents the multivariate model used in this study to determine how the measured environmental variables affect the longitudinal stability of cognitive ability (i.e., from wave 1 to wave 2). Similar to Fig. 2, the covariance between wave 1 and wave 2 Stanford-Binet SAS is influenced by common general genetic (A), shared environmental (C) and nonshared environmental (E) factors. Additionally, the independent variance in Stanford-Binet SAS at wave 2 is influenced by specific genetic (a), shared environmental (c) and nonshared environmental (e) factors. Furthermore, the identified shared environmental measures (SES, wave 1 CHAOS and wave 2 CHAOS) are also parameterized in the model. In particular, these measures influence three environmental factors (ENVFAC, CHAOSFAC, and CHAOSw2RESID), which in turn influence wave 1 Stanford-Binet SAS and wave 2 Stanford-Binet SAS. ENVFAC accounts for the common variance between all three identified environmental factors. CHAOSFAC measures the effects of the covariance between CHAOS at wave 1 and wave 2 that are independent from SES. Lastly, CHAOSw2RESID taps variance in CHAOS at wave 2 that is independent from SES or CHAOS at wave 1.

4.2.4. Variance estimates from multivariate models—Parameter estimates from the full model are presented in Tables 6 and 7. Looking at Table 6, results are presented for both the bivariate model (Fig. 2) as well as the measured environment mediation model (Fig. 3). The bivariate model suggests significant heritability, shared environment, and nonshared environment for waves 1 and 2 Stanford-Binet SAS. Heritability estimates were moderate in wave 1 and wave 2, and both are significantly different from zero (wave 1 $h^2 = .28$, wave 2 $h^2 = .44$). The estimates for the shared environmental influence are also moderate, with both waves showing estimates significantly different from zero (wave 1 $c^2 = .46$, wave 2 $c^2 = .30$). Subsequently, the estimates for nonshared environmental variance were low, but both waves were still significantly different than zero (wave 1 $e^2 = .26$, wave 2 $e^2 = .26$).

When examining the measured environment mediation model, estimates of heritability and nonshared environment are similar to the bivariate model. However, estimates of shared environment (c^2) are attenuated because a portion of this variance is now explained by identified measures of the shared environment (ENVFAC and CHAOSFAC). ENVFAC (or the shared influence of SES and wave 1 and wave 2 CHAOS) accounted for 5% of the total variance on wave 1 Stanford-Binet SAS, and 3% of the total variance in wave 2. CHAOSFAC (the covariance of wave 1 and wave 2 CHAOS independent from SES) accounted for 5% of the total variance in SAS in wave 1, and 6% in wave 2. CHAOSw2RESID (or the influence of wave 2 CHAOS separate from SES and wave 1 CHAOS) accounted for zero variance in wave

2 Stanford-Binet SAS. Total measured environment accounted for 10% of the total variance in wave 1 SAS, and 9% of the total variance in wave 2 Stanford-Binet SAS.

Moreover, ENVFAC accounted for 11% of the shared environmental variance in Stanford Binet SAS at wave 1 [11%=the proportion of variance accounted for by ENVFAC (.05) divided by the variance accounted for by ENVFAC (.05)+CHAOSFAC (.05)+ c^2 (.35)]. In wave 2, ENVFAC accounted for 10% of the shared environmental variance in Stanford Binet SAS [10%= 100* (.03/.03+.06+ .21)]. CHAOSFAC accounted for an additional 11% of the shared environmental variance in Stanford Binet SAS at wave 1 [11%=100* (.05/.05+ .05 + .35)] and 20% of the shared environmental variance at wave 2 [20%=100* (.06/.03+.06+.21)].

4.2.5. Estimated phenotypic correlations—Additionally, genetic, shared environmental and non-shared environmental contributions to the correlation between wave 1 and wave 2 Stanford-Binet SAS are presented in Table 7. Again, results are presented for the bivariate model and the mediation model. In the bivariate model, the total estimated phenotypic correlation between Stanford-Binet SAS wave 1 and wave 2 was $r=.74$. This was determined by summing the standardized genetic (A), shared environmental (C), and nonshared environmental (E) pathways from the bivariate model ($r_{\text{estimated}}=.74=.35+.36+.03$). Of this estimated phenotypic correlation, 47% ($r=.35$) of the total was due to genetic overlap, 49% ($r=.36$) due to shared environmental overlap, and 4% ($r=.03$) due to nonshared environmental overlap.

When examining the mediation model, the estimated phenotypic correlation was $r=.76$ between wave 1 and wave 2 Stanford-Binet SAS. Genetic pathways accounted for 47% ($r=.36$), the shared environmental influence, 34% ($r=.26$), and the nonshared environmental influence, 4% ($r=.03$), of this phenotypic overlap. Similar to the univariate estimates of c^2 in Table 6, the shared environmental (C pathway) overlap was attenuated in the mediation model compared to the bivariate because of the inclusion of measured shared environmental influences (SES, wave 1 CHAOS, wave 2 CHAOS). These measured environmental influences accounted together for 14% ($r=.11$) of the total estimated correlation between wave 1 and wave 2 Stanford-Binet SAS. Separately, ENVFAC accounted for 5% ($r=.04$), CHAOSFAC for 8% ($r=.06$), and CHAOSw2RESID for 1% ($r=.01$) of the estimated phenotypic correlation between wave 1 and wave 2 Stanford-Binet SAS.

Although statistical significance was determined by confidence intervals for A, C, E, and a, c, e parameters, four submodels were fit to the full model in order to test whether ENVFAC, CHAOSFAC, and CHAOSw2RESID accounted for a significant proportion of the variance in wave 1 and wave 2 Stanford-Binet SAS (see Table 8). The first submodel dropped the pathways from ENVFAC to the Stanford-Binet SAS. If this submodel resulted in a significant decline in model fit relative to the full model (determined by χ^2_{change}), then the effect of ENVFAC on Stanford-Binet SAS is assumed to be important to the fit of the model. Results for this submodel showed a significant decrease in model fit ($\chi^2_{\text{change}} = 15.44$, $\Delta df=2$, $p<.05$).

Second, we examined whether CHAOSFAC accounted for significant variance in wave 1 and wave 2 Stanford-Binet SAS (drop CHAOSFAC). This model also resulted in a significant decrease in model fit ($\chi^2_{\text{change}}=19.05$, $\Delta df=3$, $p<.05$). Third, dropping the pathway from CHAOSFAC to wave 2 Stanford-Binet SAS also resulted in a significant decrease in model fit ($\chi^2_{\text{change}} = 16.85$, $\Delta df = 1$, $p<.05$), suggesting that CHAOSFAC also accounted for the covariance between wave 1 and wave 2 Stanford-Binet SAS. Finally, the fourth submodel tested the significance of the independent variance in CHAOS at wave 2 (CHAOSw2RESID). This submodel did not result in a significant decrease in model fit ($\chi^2_{\text{change}}=0.07$, $\Delta df=1$, ns).

5. Discussion

The goals of this study were to: 1) examine whether SES and CHAOS accounted for a significant portion of the shared environmental variance in general cognitive ability; 2) examine whether SES and CHAOS accounted for a portion of the longitudinal stability in general cognitive ability; and 3) examine whether CHAOS explained a portion of shared environmental variance in cognitive ability independent from SES.

As expected from previous research, both genetic and shared environmental influences were significant within and across assessments of general cognitive ability in our early school-age sample. More interesting, SES and CHAOS mediated a significant portion of the shared environmental variance in general cognitive ability in the two waves of longitudinal assessment. It is well established that SES has a significant relationship with cognitive ability, so it is not surprising that this study showed that SES accounted for a significant proportion of shared environmental variance (Brody, 1992). However, like Petrill, Pike et al. (2004), this study also shows that chaos in the home accounts for a significant proportion of the shared environmental variance in Stanford-Binet SAS. These results suggest that growing up in a calm and orderly home is an important environmental factor in positively influencing children's cognitive functioning.

Importantly, this study also showed that CHAOS was an independent predictor of Stanford-Binet SAS separate from SES. Thus, although SES and CHAOS are correlated in our study, and other studies have shown that parents of low income families tend to have fewer years of schooling, and may therefore have lower educational expectations of their children, and concurrently have more chaotic households (Caspi, Taylor, Moffitt, & Plomin, 2000; Davis-Kean, 2005), it is not the case that the effect of CHAOS on general cognitive ability can be accounted for by SES. The mediation of both SES and CHAOS on the shared environmental variance of Stanford-Binet SAS were small (around 4% for SES and 7% for CHAOS), but significant. Together, these factors accounted for about 10% of the shared environmental variance for wave 1 and 2 Stanford-Binet SAS. Furthermore, the results of the current study suggested that SES and CHAOS account for a portion of the longitudinal stability in general cognitive ability. Related to this, it was shown that CHAOS scores at wave 2 were insignificant to the model, suggesting that there was little systematic variance left in wave 2 CHAOS, after accounting for the correlation ($r=.01$, see Table 7) between CHAOS from wave 1 to wave 2. Together SES and CHAOS accounted for 14% of the phenotypic correlation of the Stanford-Binet SAS scores between wave 1 and wave 2. These results are almost identical to those reported by Petrill, Pike et al. (2004), a notable conclusion as the current study examined tester-administered psychometric measures in a sample of school-age children whereas Petrill, Pike et al. (2004) employed parent-report measures of cognitive ability in a sample of 3 and 4 year olds.

Despite these findings, the study has some limitations. First, our identified measures of the environment accounted for a significant, but small proportion of the variance in general cognitive ability. In particular, the correlation between SES and general cognitive ability, generally shown in the literature to be around $r=.30-.40$ (Jensen, 1998), was somewhat lower in our study ($r=.24$). While this difference is likely not significant in the meta-analytic sense, the sample in the current study was nevertheless skewed in terms of SES. Moreover, the estimates of heritability in the current study appear to be somewhat lower than what is generally found in the literature. Again, confidence intervals in the current study suggest that the heritability estimates are in fact within the range of what is expected from the literature.

A third limitation is that parent education level may not completely capture SES. However, a review of the literature suggests that parental education is highly correlated with SES and is

an acceptable marker of socioeconomic conditions (see Entwisle & Astone, 1994, for review; Bradley & Corwyn, 2002). A final limitation is that we cannot be certain if the effects of chaos on the twins can be generalized to all family types, as most of our sample consisted of two-parent, intact families.

This study's conclusions are also valid to the extent that passive genotype-environment correlations are not spuriously inflating shared environmental effects. Since the entire family, including the children's parents, shares the same SES status, as well as the same home environment, it can be said that SES and CHAOS cannot be thought of as simply environmental effects, but that shared genes in the family could also be involved (Plomin & Bergeman, 1991). There is no way of knowing for sure if our measured "environmental" variables are purely environmental in nature, or if passive genotype-environment correlations are present (Turkheimer, D'Onofrio, Maes, & Eaves, 2005). However, there is little evidence that passive genotype-environmental correlation has a significant effect on cognitive ability in early childhood (Rice, Fulker, & DeFries, 1986).

Finally, while these identified measures of the environment account for a small, but significant portion of the shared environmental variance in early general cognitive skills, it is impossible to say whether these environmental measures are the actual environmental processes that lead to individual differences in cognitive ability. More likely, these measures are markers. Growing up in a more ordered home may allow a child to make better use of learning opportunities in a home, such as reading, or being able to do homework more efficiently and accurately. Also, a more chaotic home may be representative of more people being present within the home, so that a quiet home may allow a child more time with his or her parents, or be free from distractions while learning. Although this paper does show the importance of SES and CHAOS as environmental mediators of cognitive ability, it is important to remember that many other aspects of the environment not measured in the current study are likely candidates as additional environmental mediators of cognitive ability.

Despite these shortcomings, this study showed that SES and CHAOS account for a small but still significant part of the variance in Stanford-Binet SAS, and that these effects are independent and were stable longitudinally. The current results are quite similar to the Petrill, Pike et al. (2004) study, lending strength to the argument that CHAOS is a fairly robust environmental mediator of general cognitive ability. Moreover, although there is only one year between assessments, this year is very important as most children are making the transition to early elementary school. This further reinforces the conclusion that CHAOS is an important shared environmental mediator of general cognitive ability that is associated with stability across this important period of development. It will be interesting for future research to further test if the influence of CHAOS remains stable throughout cognitive development (i.e., not just over a year in early childhood or the early elementary school years), or whether the effect of CHAOS is more strongly mediated by genes as children have greater control over their own environments. Also, this and previous studies looking at environmental mediators show the importance of further study of new mediators in genetically sensitive designs. Just as there is a need to identify the genes responsible for the heritability of complex traits such as cognitive ability, it is just as important to determine the factors that go into environmental variance.

References

- Bradley RH, Caldwell BM. The relation of home environment, cognitive competence, and IQ among males and females. *Child Development* 1980;51:1140–1148.
- Bradley RH, Caldwell BM, Rock Ramey CT, Barnard KE, Gray C, et al. Home environment and cognitive development in the first 3 years of life: A collaborative study involving six sites and three ethnic groups in North America. *Developmental Psychology* 1989;25(2):217–235.

- Bradley RH, Corwyn RF. Socioeconomic status and child development. *Annual Review of Psychology* 2002;53:371–399.
- Brody, N. *Intelligence*. Vol. 2. New York: Academic Press; 1992.
- Carlson MJ, Corcoran ME. Family structure and children's behavioral and cognitive outcomes. *Journal of Marriage & the Family* 2001;63(3):779–792.
- Caspi A, Taylor A, Moffitt TE, Plomin R. Neighborhood deprivation affects children's mental health: Environmental risks identified in a genetic design. *Psychological Science* 2000;11(4):338–342. [PubMed: 11273396]
- Davis-Kean PE. The influence of parent education and family income on child achievement: The indirect role of parental expectations and the home environment. *Journal of Family Psychology* 2005;19(2):294–304. [PubMed: 15982107]
- Dumas JE, Nissley J, Nordstrom A, Smith EP, Prinz RJ, Levine DW. Home chaos: Sociodemographic, parenting, interactional, and child correlates. *Journal of Clinical Child and Adolescent Psychology* 2005;34(1):93–104. [PubMed: 15677284]
- Ellis L, Bonin SL. Genetics and occupation-related preferences. Evidence from adoptive and non-adoptive families. *Personality and Individual Differences* 2003;35:929–937.
- Entwisle DR, Astone NM. Some practical guidelines for measuring youth's race/ethnicity and socioeconomic status. *Child Development* 1994;65(6):1521–1540.
- Evans GW, Gonnella C, Marcynyszyn LG, Salpekar N. The role of chaos in poverty and children's socioemotional adjustment. *Psychological Science* 2005;16(7):560–565. [PubMed: 16008790]
- Feinstein L, Bynner J. The importance of cognitive development in middle childhood for adulthood socioeconomic status, mental health, and problem behavior. *Child Development* 2004;75(5):1329–1339. [PubMed: 15369517]
- Goldsmith HH. A zygosity questionnaire for young twins: A research note. *Behavior Genetics* 1991;21(3):257–269. [PubMed: 1863259]
- Gutman LM, Sameroff AJ, Cole R. Academic growth curve trajectories from 1st grade to 12th grade: Effects of multiple social risk factors and preschool child factors. *Developmental Psychology* 2003;39(4):777–790. [PubMed: 12859129]
- Jensen AR. Raising the IQ: The Ramey and Haskins study. *Intelligence* 1981;5:29–40.
- Jensen, AR. *The g factor*. Wesport, CT: Praeger; 1998.
- Jimerson S, Egeland B, Sroufe A, Carlson B. A prospective longitudinal study of high school dropouts: Examining multiple predictors across development. *Journal of School Psychology* 2000;38(6):525–549.
- Matheny AP, Wachs TD, Ludwig JL, Phillips K. Bring order out of chaos: Psychometric characteristics of the confusion, hubbub, and order scale. *Journal of Applied Developmental Psychology* 1995;16:429–444.
- McGue, M.; Bouchard, TJ.; Iacono, WG.; Lykken, DT. Behavioral genetics of cognitive ability: A life-span perspective. In: Plomin, R.; McClearn, GE., editors. *Nature, Nurture and Psychology*. Washington, DC: American Psychological Association; 1993.
- McLoyd VC. Socioeconomic disadvantage and child development. *American Psychologist* 1998;53:185–204. [PubMed: 9491747]
- Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. *Mx: Statistical Modeling*. Vol. 5. Box 126 MCV, Richmond, VA 23298; Department of Psychiatry; 1999.
- Neiss M, Rowe DC. Parental education and child's verbal IQ in adoptive and biological families in the National Longitudinal Study of Adolescent Health. *Behavior Genetics* 2000;30(6):487–495. [PubMed: 11523707]
- NICHD Early Child Care Research Network. Nonmaternal care and family factors in early development: An overview of the NICHD study of early child care. *Applied Developmental Psychology* 2001;22:457–492.
- Pederson NL, Plomin R, Nesselroade JR, McClearn GE. A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychological Science* 1992;3:346–353.

- Petrill SA, Deater-Deckard K, Thompson LA, DeThorne LS, Schatschneider C. Reading skills in early readers: Genetic AND shared environmental influences. *Journal of Learning Disabilities* 2006;39(1): 48–55. [PubMed: 16512082]
- Petrill SA, Lipton PA, Hewitt JK, Plomin R, Cherny SS, Corely R, et al. Genetic and environmental contributions to general cognitive ability through the first 16 years of life. *Developmental Psychology* 2004;40(5):805–812. [PubMed: 15355167]
- Petrill SA, Pike A, Price T, Plomin R. Chaos in the home and socioeconomic status are associated with cognitive development in early childhood: Environmental mediators identified in a genetic design. *Intelligence* 2004;32:445–460.
- Plomin R, Bergeman CS. The nature of nurture: Genetic influence on “environmental” measures. *Behavioral and Brain Sciences* 1991;14:373–427.
- Plomin, R.; DeFries, JC.; McClearn, GE.; McGuffin, P. *Behavioral genetic*. Vol. 3. New York: W. H. Freeman; 2001.
- Rice T, Fulker DW, DeFries JC. Multivariate path analysis of specific cognitive abilities in the Colorado Adoption Project. *Behavior Genetics*. Special Issue: Multivariate Behavioral Genetics and Development 1986;16(1):107–125.
- Rumberger R. High school dropouts: A review of issues and development. *Review of Educational Research* 1987;57(2):101–121.
- Thorndike, RL.; Hagen, EP.; Sattler, JM. *Guide for administering and scoring the fourth edition: Stanford-Binet Intelligence Scale*. Chicago, IL: Riverside; 1986.
- Thorndike, RL.; Hagen, EP.; Sattler, JM. *Technical manual*. Vol. 4. Chicago, IL: Riverside; 1986. *The Stanford-Binet Intelligence Scale*.
- Turkheimer E, D’Onofrio BM, Maes HH, Eaves LJ. Analysis and interpretation of twin studies including measures of the shared environment. *Child Development* 2005;76(6):1217–1233. [PubMed: 16274436]
- van den Oord EJCG, Rowe DC. A cousin study of associations between family demographic characteristics and childrens’ intellectual ability. *Intelligence* 1999;27(3):251–266.
- Vernon, PA. Behavioral genetic and biological approaches to intelligence. In: Nyborg, H., editor. *The scientific study of human nature: Tribute to Hans. J. Eysenck at eighty*. Amsterdam, Netherlands: Pergamon/Elsevier Science Inc; 1997. p. 240-258.

Fig. 1. Univariate mediation model estimating genetic, shared environmental, nonshared environmental and identified shared environmental variances in waves 1 and 2 Stanford-Binet SAS.

Fig. 2.
Bivariate model estimating genetic, shared environmental, nonshared environmental variance for waves 1 and 2 Stanford-Binet SAS.

Fig. 3. Multivariate mediation model estimating genetic, shared environmental, nonshared environmental and identified shared environmental variance for waves 1 and 2 Stanford-Binet SAS.

Table 1

Items from the CHAOS questionnaire

1	2	3	4	5
Definitely untrue	Somewhat untrue	Not really true or untrue	Somewhat true	Definitely true
Please circle				
1	2	3	4	5
The children have a regular bedtime routine (for example, same bed time each night, a bath before bed, reading a story, saying prayers...).				
1	2	3	4	5
You can't hear yourself think in our home.				
1	2	3	4	5
It's a real zoo in our home.				
1	2	3	4	5
We are usually able to stay on top of things.				
1	2	3	4	5
There is usually a television turned on somewhere in our home.				
1	2	3	4	5
The atmosphere in our house is calm.				

Above are some things that happen in most homes. Please read each item carefully and circle the number beside it to tell us what best describes YOUR home.

Table 2

Means and standard deviations for SES, waves 1 and 2 CHAOS and Stanford-Binet SAS

Variable	Mean	SD	<i>n</i>
SES	5.72	1.77	690
CHAOS w1	2.35	.65	534
CHAOS w2	3.33	.60	376
SAS w1	100.41	13.19	514
SAS w2	102.37	12.40	428

Note. *n*=number of individuals.

Table 3
Pearson correlations among waves 1 and 2 Stanford-Binet SAS, SES and waves 1 and 2 CHAOS

Variable	SAS w1	SAS w2	SES	CHAOS w1	CHAOS w2
SAS w1	1.00				
(n)	514				
SAS w2	0.72	1.00			
(n)	409	428			
SES	0.24	0.17	1.00		
(n)	506	422	690		
CHAOS w1	0.28	0.29	0.21	1.00	
(n)	469	398	530	534	
CHAOS w2	0.29	0.27	0.21	0.81	1.00
(n)	362	346	372	364	376

Note. Waves 1 and 2 CHAOS were reverse coded.

n=number of individuals.

All correlations were significant at two-tailed $p<.01$.

Table 4
Intraclass and cross-twin correlations for waves 1 and 2 Stanford-Binet SAS by zygosity

Zygosity	Stanford-Binet SAS w1 twin 2	Stanford-Binet SAS w2 twin 2
Monogygotic		
Stanford-Binet SAS w1 twin 1	.76	
Stanford-Binet SAS w2 twin 1	.72	.75
Dizygotic		
Stanford-Binet SAS w1 twin 1	.58	
Stanford-Binet SAS w2 twin 1	.48	.69

Note. All correlations were significant at two-tailed $p < .01$.

Table 5
Univariate model fitting results with confidence intervals

Variable	h^2 [C.I.]	c^2 [C.I.]	e^2 [C.I.]	SES ² [C.I.]	CHAOS ² [C.I.]	-2ll	df
SAS w1							
SES mediation	.29 [.26,.54]	.40 [.17,.59]	.26 [.19,.35]	.05 [.01,.11]		2060.06	770
CHAOS mediation	.27 [.04,.51]	.39 [.17,.58]	.26 [.20,.36]		.07 [.02,.14]	1973.77	745
SAS w2							
SES mediation	.51 [.23,.77]	.20 [.00,.44]	.26 [.19,.36]	.03 [.01,.09]		1892.05	690
CHAOS mediation	.49 [.21,.73]	.16 [.00,.40]	.27 [.19,.37]		.08 [.03,.15]	1797.07	665

Table 6
Heritability, shared environmental, nonshared environmental, and identified environmental influences with confidence intervals in waves 1 and 2 Stanford-Binet SAS estimated from multivariate models

Estimate	Wave 1	Wave 2
Bivariate model		
h^2 [C.I.]	.28 [.10,.50]	.44 [.21,.71]
c^2 [C.I.]	.46 [.23,.66]	.30 [.10,.57]
e^2 [C.I.]	.26 [.20,.33]	.26 [.21,.36]
Mediation model		
h^2 [C.I.]	.29 [.11,.51]	.45 [.21,.70]
c^2 [C.I.]	.35 [.12,.54]	.21 [.03,.47]
e^2 [C.I.]	.26 [.20,.33]	.26 [.21,.36]
ENVFAC ²	.05 ^a	.03 ^a
CHAOSFAC ²	.05 ^a	.06 ^a
CHAOSw2RESID ²	xx	.00
(Total measured environment) ²	(.10)	(.09)

Note: Confidence intervals tested in submodels presented in Table 8.

^a $p < .05$.

Table 7

Component of correlation between waves 1 and 2 Stanford-Binet SAS accounted for by genetic, shared environmental, nonshared environmental and identified environmental factors with confidence intervals

Estimate	Correlation	Proportion (%)
Bivariate model		
h^2 [C.I.]	.35 [.18,.55]	47
c^2 [C.I.]	.36 [.17,.57]	49
e^2 [C.I.]	.03 [.00,.08]	4
Total estimated correlation	.74	100
Mediation model		
h^2 [C.I.]	.36 [.19,.56]	47
c^2 [C.I.]	.26 [.07,.45]	34
e^2 [C.I.]	.03 [.00,.08]	4
ENVFAC	.04 ^a	5
CHAOSFAC	.06 ^a	8
CHAOSw2RESID	.01	1
(Total measured environment)	(.11)	(14)
Total estimated correlation	.76	100

Note: Confidence intervals tested in submodels presented in Table 8.

^a $p < .05$.

Model fitting results examining SES, waves 1 and 2 CHAOS mediation of the shared environment: waves 1 and 2 Stanford-Binet SAS

Model	-2ll	df	χ^2 change	Δdf	p change
Full	3936.49	1608			
Drop ENVFAC	3951.93	1610	15.44	2	*
Drop CHAOSFAC	3955.54	1611	19.05	3	*
Drop correlation between CHAOSFAC and wave 2 SAS	3953.34	1609	16.85	1	*
Drop CHAOSw2RESID	3936.56	1609	.07	1	NS

$n=119$ MZ and $n=164$ DZ pairs using raw data in Mx.

* $p<.05$.