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# Early Life Development in a Multiethnic Sample and the Relation to Late Life Cognition

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***Objectives.*** Poor quality of early life conditions has been associated with poorer late life cognition and increased risk of dementia. Early life physical development can be captured using adult measures of height and head circumference. Availability of resources may be reflected by socioeconomic indicators, such as parental education and family size. We sought to determine the association between early life development and experience and late life semantic memory, episodic memory, and executive functioning abilities, as well as rate of cognitive decline.

***Method.*** This study was conducted using the UC Davis Aging Diversity cohort, an ethnically diverse sample of Caucasian, African American, and Hispanic individuals from northern California. We used latent variable modeling to measure growth and childhood socioeconomic environment (SES) and examine their associations with longitudinal cognitive outcomes using mixed effects modeling.

***Results.*** Growth was positively related to higher childhood SES. Higher childhood SES was associated with better semantic memory. Both low growth and low SES were associated with increased rate of cognitive decline.

***Discussion.*** These findings demonstrate that early life experiences influence the trajectory of cognitive aging. Early life development and experience appears to provide a distal basis upon which additional risk and protective factors interact in the development of dementia.

***Key Words:*** Cognition—Longitudinal change—Minority and diverse populations.

DEVELOPMENT is highly dependent upon quality of living conditions, particularly in childhood. Although it is difficult to retrospectively assess early life experiences and development, prior research has identified markers of early life environment that are amenable to measurement in later life. Under poor living conditions, which may include poor nutrition, medical illness, or emotional stress, available resources for physical development are directed toward trunk, head, and organ growth over limb growth (for a review, see Bogin & Varela-Silva, 2010). Following these observations, researchers have shown that morphometric measurements in adulthood, such as leg length, height, and head circumference (HC), reflect not only genetic factors that dictate growth potential but additionally carry information about quality of the early childhood environment.

Availability of resources in the environment that may foster optimal development can also be captured by information about the family structure and functioning. This includes factors such as parental education, parental occupation, the total number of children in a family, the number of siblings deceased before adulthood, and wealth. Studies conducted around the globe have found associations between childhood environment and morphometrics in adulthood, suggesting that greater availability of resources enables optimal growth (for a review, see Silventoinen, 2003). In the United States, it is estimated that approximately 20%–30% of the variation in adult height is due to environmental factors (Silventoinen, Krueger, Bouchard, Kaprio, & McGue, 2004; Stunkard, Foch, & Hrubec, 1986).

Studies of cognitive aging have examined the association between early childhood environment and late life cognition (Alwin & Hofer, 2011; Haan, Zeki Al-Hazzouri, & Aiello, 2011; Raikkonen et al., 2013; Richards & Hatch, 2011). In general, shorter height and smaller HC are predictive of poor cognition and/or Alzheimer's disease (AD) (Abbott et al., 1998; Graves et al., 1996; Guven & Lee, 2013; Kim, Lee, et al., 2008; Maurer, 2010; Mortimer, Snowdon, & Markesbery, 2003; Schofield, Logroscino, Andrews, Albert, & Stern, 1997). Poorer cognition in late adulthood is associated with childhood experiences thought to reflect lower socioeconomic status (SES), such as rural living environment, father in manual occupation, and large number of children in the household (Abbott et al., 1998; Borenstein et al., 2005; Kim, Stewart, et al., 2008).

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Morphometric and childhood SES data share independent associations with late life cognition but, when examined in a single model, growth has been more strongly predictive of late life cognition (Gale, Walton, & Martyn, 2003; Scazufca et al., 2008). It has also been observed that individuals with the apolipoprotein E (APOE) e4 allele are more likely to show adverse late life cognitive outcomes in the presence of low HC/height or childhood SES (Borenstein et al., 2005; Kim, Lee, et al., 2008; Moceri et al., 2001; Perneczky, Alexopoulos, Wagenpfeil, Bickel, & Kurz, 2012; Tate et al., 2011). Thus, research so far suggests that childhood experiences are related to late life cognition. However, much of the research to date has been cross-sectional in nature, and it remains unclear if childhood experiences affect the rate of cognitive aging.

Although these observations have been made in samples across the world, including Japanese, Korean, African-Caribbean, European, and American, the role that ethnicity might have on this association has not been fully studied. Differences in ethnic background have been proposed to modify the course of cognitive aging (Glymour & Manly, 2008; Haan et al., 2011). In a mixed sample of adults in NYC, including African American, Caucasian, and Hispanic Americans, AD diagnosis was associated with smaller HC after controlling for ethnicity (Schofield et al., 1997). Somewhat paradoxically, in the Chicago region, early life adversity was found to have a protective effect in African Americans relative to Whites (Barnes et al., 2008). Thus, ethnic group membership appears to reflect an important aspect of diversity that may affect the importance of early life experience on late life cognitive functioning.

The present investigation aimed to characterize the association between early life environment and late life cognition in a diverse sample from northern California, including near equal numbers of Caucasian, African American, and Hispanic participants. We used latent variable modeling methods to characterize two factors thought to reflect childhood environment, one using adult morphometrics and a second using multiple indicators of childhood SES. We then investigated the impact of ethnicity on these early life factors. Finally, we investigated whether these factors were associated with late life cognition (both global and domain specific) and importantly, cognitive decline. We hypothesized that the growth and SES factors would be correlated with one another in all ethnic groups and that both would be significant predictors of late life cognition and cognitive decline.

## Method

### *Participants and Recruitment*

Data were obtained from the UC Davis Aging Diversity Cohort, a longitudinal study of cognitive aging in an ethnically and demographically diverse population of older adults. Community-based outreach methods were used to optimize the racial/ethnic diversity of the cohort. These methods have been described elsewhere (Hinton, 2010). The study was carried out within the research program of the University of California at Davis Alzheimer's Disease Center (UCD ADC). Eligibility criteria required that participants be age 60 or older at baseline and be able to speak English or Spanish. Participants with an unstable major medical illness at baseline, a major primary psychiatric disorder (history of schizophrenia, bipolar disorder, or recurrent major depression), or substance abuse or dependence in the last 5 years were excluded from the study. The present study included only participants who described themselves as Caucasian, African American, or Hispanic due to the small number of participants who fell within other ethnic groups ($n$ = 16). Only participants with two or more study visits were included. All participants signed informed consent, and human participant involvement was overseen by institutional review boards at University of California at Davis, the Veterans Administration Northern California Health Care System, and San Joaquin General Hospital in Stockton, California.

Sample characteristics are presented in Table 1. There were 333 participants (Caucasian, $n$ = 104; African American, $n$ = 120; Hispanic, $n$ = 109). Hispanics were significantly younger and had less education than Caucasian and African American. The Caucasian group included more male participants than African American and Hispanic groups. Forty-seven percent of the Hispanic sample and one African American participant were Spanish monolingual. Forty-two percent of Hispanics and 3% of Caucasians were English/Spanish bilingual. Across the entire sample, 66% were found to have normal cognition, 25% were diagnosed with mild cognitive impairment (MCI), and 9% were diagnosed with dementia. Descriptive statistics for observed indicators of growth and childhood SES are presented in Table 2.

### *Cognitive Assessment*

Measures of episodic memory, semantic memory, and executive function derived from the Spanish and English Neuropsychological Assessment Scales (SENAS) were used as cognitive outcome measures. The SENAS has undergone extensive development, and psychometric methods based on item response theory were used to create psychometrically equivalent measures across language and race/ethnicity (Mungas, Reed, Crane, Haan, & González, 2004; Mungas, Reed, Haan, & González, 2005; Mungas, Reed, Marshall, & González, 2000; Mungas, Reed, Tomaszewski Farias, & DeCarli, 2005). These measures were administered at all evaluations.

### *Indicators of Childhood Environment*

Morphometric data included physical measurements of height, HC, knee height, and femur length. These

Table 1. Characteristics of the Study Sample at the Baseline Assessment by Ethnicity

| Variable | African American | Hispanic | Caucasian | *p* Value |
|---|---|---|---|---|
| Sample size | *n* = 120 | *n* = 109 | *n* = 104 | |
| Number of assessments | 4.24 (1.74) | 4.50 (1.86) | 4.05 (1.96) | .210 |
| Years in study—mean (*SD*) | 4.27 (2.13) | 4.57 (2.25) | 3.77 (2.28) | .030 |
| Episodic memory—mean (*SD*) | 0.16 (0.86) | –0.34 (0.93) | 0.17 (1.13) | .001 |
| Semantic memory—mean (*SD*) | –0.05 (0.74) | –0.61 (1.00) | 0.69 (0.83) | .001 |
| Executive function—mean (*SD*) | 0.01 (0.83) | –0.43 (0.97) | 0.43 (1.03) | .001 |
| Age—mean (*SD*) | 74.43 (7.08) | 71.47 (6.46) | 74.93 (6.76) | .001 |
| Gender—% female | 73 | 66 | 58 | .048 |
| Years of education—mean (*SD*) | 13.54 (3.03) | 8.39 (5.48) | 14.40 (3.05) | .001 |
| Apolipoprotein E ε4—% positive (*n*) | 45 (49) | 20 (20) | 35 (32) | .001 |
| Monolingual English—% (*n*) | 99 (119) | 11 (12) | 97 (101) | .001 |
| Monolingual Spanish—% (*n*) | 1 (1) | 47 (51) | 0 (0) | .001 |
| Bilingual—% (n) | 0 (0) | 42 (46) | 3 (3) | .001 |
| Normal cognition—% (*n*) | 62 (73) | 72 (75) | 64 (65) | .026 |
| Mild cognitive impairment—% (*n*) | 31 (36) | 18 (19) | 25 (26) | .10 |
| Dementia—% (*n*) | 7 (8) | 10 (10) | 11 (11) | .006 |

*Note.* Diagnosis was missing from 10 participants.

Table 2. Sample Descriptive Statistics for Indicators of Childhood SES and Growth/Physical Development

| Variable | Mean | *SD* | Range |
|---|---|---|---|
| Height (in.) | 63.76 | 3.88 | 54–74 |
| Head circumference (cm) | 55.71 | 2.32 | 49–63 |
| Femur length (cm) | 36.79 | 4.11 | 26–49 |
| Knee height (cm) | 49.98 | 3.70 | 41–62 |
| Mother's education (years) | 8.40 | 4.90 | 0–16 |
| Father's education (years) | 8.22 | 5.37 | 0–20 |
| Father's occupation complexity[a] | 3.59 | 2.17 | 1–10 |
| Number of siblings | 4.77 | 3.12 | 1–18 |
| Number of siblings who died | 0.48 | 0.85 | 0–3 |

*Note.* [a]Ten-point scale where 1 indicates low complexity and 10 indicates high complexity.

measurements are commonly used to interrogate the association between growth and cognitive aging. HC may reflect growth particularly within the first 6 years of life, whereas height and leg length markers may reflect growth up to the first two decades (see Borenstein, Copenhaver, & Mortimer, 2006, for a review). These measurements were obtained following a standardized protocol (Center for Disease Control, 1988). HC was measured by placing a measuring tape over the eyebrows and passing it around the head to fit over the most posterior protuberance of the occiput. Knee height was measured while the participant was in a seated position by placing a measuring tape at the top of the patellar bone, a flat edge was then extended out, and the distance from the top of the height of the patella to the floor was measured. Femur length was also measured while the participant was in a seated position by placing a measuring tape at the crease of the hip to the start of the patellar bone. Data regarding childhood SES were drawn from the Life Experiences and Activities Form (LEAF), an interview-based instrument used to characterize experience across the participant's life span. Our variables of interest included mother's educational attainment, father's educational attainment, complexity of the father's job (Roos & Treiman, 1980), number of siblings, and number of siblings who died before age 18.

### *Clinical Evaluations and APOE Genotyping*

The standardized clinical evaluation procedures carried out by UCD ADC have been described elsewhere (DeCarli et al., 2008; Mungas et al., 2010). The SENAS measures were not considered in diagnostic classification of participants (cognitively normal vs. MCI vs. dementia). APOE genotyping was conducted using the LightCycler ApoE mutation detection kit (Roche Diagnostics, Indianapolis, IN).

### *Data Analysis*

The primary independent variables in this study were variables related to early life SES and markers of growth. There were three stages of data analysis. In Stage 1, we developed a factor model that used the multiple indicators of growth and SES to measure these constructs. In Stage 2, we examined how SES and growth factors related to demographic variables and APOE. In Stage 3, we evaluated how the growth and SES factors related to late life cognitive trajectories.

### *Factor Analysis of Early Life SES and Growth/Physical Development*

We developed a two-factor model of growth/physical stature as a reflective factor and early life SES as a formative factor. Reflective factors are more commonly used, and conceptually, the summary factor score is considered to "cause" or explain the correlation among the indicators for that factor. In the case of physical stature, the concept is that individual indicators of physical stature increase in a correlated manner as overall physical stature increases.

In contrast, formative factors are considered to be "caused" or explained by their observed indicators. The observed indicators need not be highly correlated or unifactorial but have some relation to the construct of interest. SES is a prime example; parental education is an established determinant of an individual's early life SES, but it is harder to envision a child's SES as having a causative effect on her or his parent's educational attainment. A formative factor in effect is an optimally weighted linear combination of the individual indicators for that factor. Indicators of formative factors are selected on the basis of their construct validity, without regard to the correlations among indicators (Bollen & Lennox, 1991; Edwards & Bagozzi, 2000). In the present analyses, indicators of childhood SES obtained from the LEAF were selected based on prior research supporting the association between each indicator and early life SES. Weights associated with formative factors are comparable to beta weights in regression, in that they reflect the relative contribution of each indicator to the factor.

The factor model for growth and early life SES is presented in Figure 1. Indicators for growth were height, HC, femur length, and knee height. Indicators for childhood SES were mother's educational attainment, father's educational attainment, complexity of the father's job, number of siblings, and number of siblings who died before age 18. The growth factor was residualized within the factor model for effects of gender to account for well-established gender differences in stature (Growth-Res in Figure 1).

Model estimation was performed with Mplus version 7.0 (Muthen & Muthen, 2010) using a maximum likelihood estimator for continuous variables applied to a mean and covariance data structure. Latent variable modeling traditionally uses an overall chi-square test of model fit, often supplemented by a number of fit indices to better characterize model fit. Commonly used fit indices include the comparative fit index (CFI; Bentler, 1990), the Tucker–Lewis index (TLI; Tucker & Lewis, 1973), the root mean square error of approximation (RMSEA; Browne & Cudeck, 1993), and the standardized root mean squared residual (SRMR; Bentler, 1995).

### *Relations of Early Life SES and Growth/Physical Development with Demographic Variables and APOE*

The growth and childhood SES estimated factor scores from the two-factor model estimated in Stage 1 were entered as dependent variables into a linear regression model. Independent variables included ethnicity, age, education, gender, and APOE. Gender was not an independent variable for growth because growth was residualized for gender in the Stage 1 factor model. Growth was regressed on childhood SES in this model because this is a substantively important path for understanding these variables.

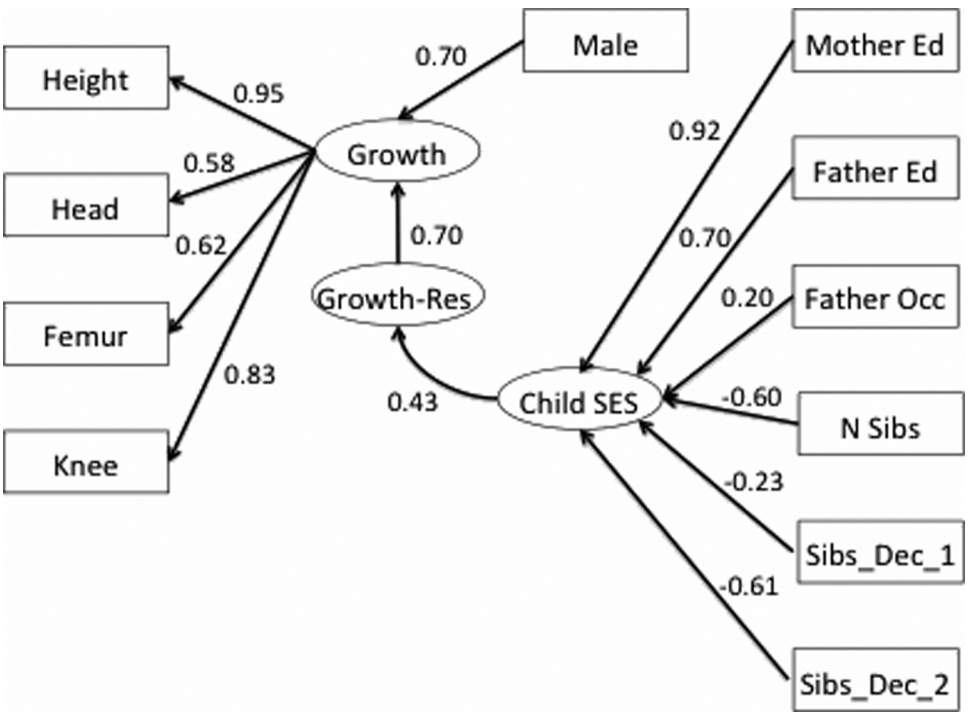


Figure 1. Confirmatory factor analysis model of childhood SES and growth/physical development. Growth is modeled as a reflective factor and childhood SES (Child SES) as a formative factor. Values presented for the growth factor are standardized loadings; values presented for childhood SES are correlations of the latent factor with the observed indicators for this factor. Growth-Res is a latent variable that captures the residual of growth not explained by gender (Male). The values for the regressions of growth on Male and Growth-Res and for Growth-Res on Child SES are standardized regression coefficients. Male and Growth-Res made equal contributions to variance in Growth.

A multiple group analysis was performed to test whether relations of growth and childhood SES with one another and with demographic variables and APOE were invariant across African American, Hispanic, and Caucasian groups. A base model constrained the regression coefficients relating these variables to be the same in the three groups. Model modification indices were examined to identify noninvariant regression coefficients. A threshold of 6.63 was used as the standard for significant improvement, which corresponds to $p = .01$ for a chi-square variate with one degree of freedom.

### *Effects of Early Life SES and Growth/Physical Development on Late Life Cognitive Trajectories*

Estimated factor scores from the two-factor model estimated in Stage 1 were used as the primary independent variables to explain baseline cognitive scores and rate of change over time. SENAS measures of episodic memory, semantic memory, and executive function, standardized using means and standard deviations (*SD*s) from the baseline evaluation of the full sample, were used as longitudinal outcomes. Mixed effects longitudinal analyses were performed using MPlus version 7.0 multilevel modeling (Muthen & Muthen, 2010). Mixed effects models for longitudinal data provide estimates of the baseline value and rate of change in the outcomes of interest. They also estimate how differences in the baseline level and rate of change relate to variables of interest (fixed effects) that differ between subjects (e.g., demographic variables). These models allow for heterogeneity in the number of assessment time points and in the lags between assessments across persons. Complete data were not available on all variables, and so the missing data analysis option of Mplus was used. Mplus uses full information maximum likelihood estimation, which provides unbiased parameter estimates in the context of missing at random (Newman, 2003). Missing data were primarily missing by design, which meets requirements for missing at random (Bollen & Curran, 2006). Analyses took advantage of the complete sample to estimate baseline and change random effects for the three cognitive outcomes and to estimate how basic demographic variables and APOE influenced cognitive baseline and change.

Model building proceeded in steps. Briefly, we first developed a base model to estimate intercept and slope random effects for all three outcomes. This model included within-subjects terms to account for practice and form effects. Specifically, the episodic memory measure included in SENAS consisted of three forms that were alternated across measurement occasions to control for practice effects. A time-varying covariate coded for the episodic memory form administered at each evaluation. For each of the three cognitive outcome measures, a variable coding for previous exposure versus no exposure was created and included as a time-varying fixed effect. The initial model allowed the six random effects latent variables (intercept and slope random effects for each of the three outcomes) to freely correlate, but we then estimated second-order latent variables that explained the correlations among the random effects. We compared fit of models with 0, 1, and 2 second-order factors using the Sample Size Adjusted Bayesian Information Criterion (SA-BIC). The SA-BIC weights model parsimony and model fit and has been shown in simulation studies to be useful for comparing model fit (Enders & Tofighi, 2008; Tofighi & Enders, 2007). We identified a best fitting base model that had separate intercepts for the three outcomes but a single, second-order slope factor representing global cognitive change. We then added growth and SES variables as the primary independent variables to explain cognition baseline and change. We added APOE genotype and basic demographic variables age (years, centered at 70), gender, education (years, centered at 12), language, and ethnicity as fixed-effect independent variables to explain cognition baseline and change. We examined interaction effects involving ethnicity and other covariates and retained significant interaction effects in subsequent models.

Previous studies have suggested that physical stature does not have linear effects on cognitive outcomes, and a number of studies have recoded continuous values into grouped variables based on quintiles (Abbott et al., 1998; Graves et al., 1996; Kim, Lee, et al., 2008; Kim, Stewart, et al., 2008; Lee, Eom, Cheong, Oh, & Hong, 2010; Maurer, 2010; Mortimer et al., 2003; Schofield et al., 1997). To address this possibility, we performed analyses using continuous growth and using categorical variables in which groups corresponded to quintiles of the distributions of the continuous variables. We also evaluated whether childhood SES was better conceptualized as a continuous variable or as a categorical variable.

Mixed model regression analyses are sensitive to assumptions of linearity, normality, and constant variance. These assumptions were examined using graphical and statistical diagnostics. Residuals and random effects were examined to assure that they were normally distributed, and plots of residuals against predicted values and effects were examined to verify that nonlinear trends in the data or nonconstant variances were not present. Additional diagnostics included evaluation of variance components related to random effects and within-subject error variance to address adequacy of statistical estimation procedures associated with the random effects modeling.

### *Development of the Measurement Model for Childhood SES and Growth/Physical*

Development and incorporation of these variables in subsequent structural models occurred in separate stages. There are clear advantages of estimating all measurement and structural parameters within the same model, but there were practical issues that made this problematic in this

study. First, with respect to analyses in which the childhood SES variable was a dependent variable, the weights for the a priori indicators of this factor would be influenced by the weights of the other independent variables in a single step analysis, and the construct being measured would be somewhat different. Consequently, two steps would be required to measure the desired factor: the first step would estimate the parameters for the a priori indicators and the second would fix these parameters at the estimated values and regress the resulting latent variable on the independent variables. Second, recoding continuous factor scores into ordinal, quintile-defined variables within a single analysis would be difficult. Third, the multilevel modeling framework presents conceptual and computational challenges for using a between-subject measurement model derived from cross-sectional baseline evaluation data to explain between-subject differences in within-subject intercept and slope parameters.

## Results

### *Factor Analysis of Growth and Childhood SES*

The hypothesized two-factor model accounting for markers of growth and childhood SES fit well ($\chi^2[24] = 37.1$, CFI = 0.988, TLI = 0.983, RMSEA = 0.035 (0.007–0.056), SRMR = 0.021). Table 3 shows standardized loadings for the growth factor and standardized regression coefficients for childhood SES. The loadings for growth can be interpreted as the correlations of the observed indicators with the underlying latent factor. The growth factor was very strongly related to height ($\lambda = 0.95$) and knee height (0.84) with weaker but still strong loadings for femur length (0.62) and HC (0.58). The strength of these loadings supports a latent factor that explains correlations among these observed variables. The values presented in Table 3 for childhood SES represent independent contributions of the childhood SES markers to the latent, formative factor. Mother's educational attainment was the strongest determinant of the latent factor (standardized $\beta = .80$). Having two or more siblings die during childhood made a significant independent contribution ($\beta = -.36$). The other indicators did not make significant contributions independent of these two variables. Figure 1 presents results from a somewhat different perspective. The numeric values presented for the growth factor are the same standardized loadings that are in Table 2, but the values for childhood SES are correlations of the latent factor with the individual indicators. These results show that father's education ($r = .70$) and number of siblings (–.60) also were strongly related to this factor, with weaker relationships for complexity of father's occupation (.20) and having one sibling die during childhood (–.23). Although these results would suggest that this factor can be adequately characterized by two variables, we chose to retain all six indicators for subsequent analyses because this adds to measurement precision. Factor scores from this analysis were estimated and entered as variables in subsequent analyses.

### *Relations of Demographic Variables and APOE with Growth and Childhood SES*

Factor scores estimated in Stage 1 were entered as dependent variables in a regression analysis in which ethnicity, age, education, gender (for childhood SES), and APOE were independent variables. Standardized regression coefficients from this analysis are presented in Table 4. A standardized regression coefficient can be interpreted as the correlation of the independent variable with the dependent variable independent of the other independent variables in the model. Growth was significantly lower in Hispanics (–0.47 *SD*). Older age was associated with lower growth (standardized regression coefficient [$\beta$] = –.15), and higher childhood SES was associated with higher growth

Table 3. Standardized Loading and Regression Coefficients for Two-Factor Model of Growth/Physical Development and Childhood SES

| Factor | Indicator | Coefficient | *SE* | *p* Value |
|---|---|---|---|---|
| Growth | Head circumference | 0.579 | 0.034 | .001 |
| | Height | 0.946 | 0.013 | .001 |
| | Femur length | 0.615 | 0.032 | .001 |
| | Knee height | 0.832 | 0.018 | .001 |
| Childhood SES | Mother's education | 0.802 | 0.256 | .002 |
| | Father's education | –0.020 | 0.313 | .948 |
| | Father's occupation complexity | –0.148 | 0.178 | .406 |
| | *N* siblings | –0.138 | 0.171 | .417 |
| | 1 sibling died | –0.027 | 0.155 | .864 |
| | 2+ siblings died | –0.360 | 0.159 | .023 |

*Notes.* Growth is modeled as a reflective factor and tabled coefficients are standardized regression coefficients that can be interpreted as the correlation of the observed indicators with the latent factor. Childhood SES was modeled as a formative factor, and coefficients are standardized regression coefficients that indicate the contribution of the observed indicator to the latent factor independent of the other variables contributing to that factor.

Table 4. Effects of Demographic Variables and Apolipoprotein E (APOE) on Growth/Physical Development and Childhood SES

| Dependent variable | Independent variable | Coefficient | *SE* | *p* Value |
|---|---|---|---|---|
| Growth | African American | 0.023 | 0.060 | .700 |
| | Hispanic | **–0.468** | **0.075** | **.001** |
| | Age | **–0.148** | **0.054** | **.006** |
| | Education | 0.010 | 0.065 | .873 |
| | APOE ε4 | –0.033 | 0.054 | .542 |
| | Childhood SES | **0.208** | **0.079** | **.008** |
| Childhood SES | African American | **–0.143** | **0.070** | **.041** |
| | Hispanic | **–0.477** | **0.075** | **.001** |
| | Male | –0.028 | 0.060 | .645 |
| | Age | 0.004 | 0.065 | .946 |
| | Education | **0.300** | **0.072** | **.001** |
| | APOE ε4 | 0.075 | 0.063 | .235 |

*Note.* Tabled values are standardized regression coefficients. Statistically significant effects ($p \leq .05$) are shown in bold.

(β = .21). Older age independently accounted for about 2% of the variance in the growth factor and childhood SES for about 4%. Childhood SES was lower in Hispanics (−0.48 *SD*) and African Americans (−0.14 *SD*) and was positively related to educational attainment (β = .30, 9% of variance).

A multiple group analysis evaluated invariance of regression coefficients for age, education, gender, and APOE in African Americans, Hispanics, and Caucasians. Model fit significantly improved when the regression coefficient of growth on childhood SES was allowed to differ in Hispanics, but the model with this single source of noninvariance fit well ($\chi^2$[23] = 25.0, CFI = 0.939, TLI = 0.928, RMSEA = 0.028 (0.000–0.085), SRMR = 0.067) and freeing equality constraints on other regression coefficients did not result in better fit ($\chi^2$[17] = 24.4, $p$ = .11). Childhood SES was not related to growth in Hispanics (β = .01, *SE* = 0.14, $p$ = .94) but was related to growth in African Americans (β = .39, *SE* = 0.09, $p$ = .001; 15% of variance) and Caucasians (β = .30, *SE* = 0.08, $p$ = .001; 9% of variance).

### *Effects of Growth and Childhood SES on Cognitive Trajectories*

In Stage 3, effects of growth and childhood SES on cognitive baseline and rate of change were evaluated using multilevel models. We compared results for continuous factor scores and groups corresponding to quintiles of the distributions of these continuous variables. The categorical variables were used to evaluate nonlinearity in relations with cognition. These analyses included either growth or childhood SES, continuous or categorical, as the primary independent variable. Covariates included ethnicity, gender, education, language of test administration, APOE, and an ethnicity by education interaction for executive function baseline that was identified for the base model.

Table 5 presents effects of continuous and categorical childhood SES on cognitive outcomes (global cognitive change, episodic memory baseline, semantic memory baseline, executive function baseline). For the categorical SES variable, the lowest four quintiles all had significantly greater average cognitive decline than the highest quintile. Differences in comparison with quintile 5 in rate of decline ranged from −0.059 to −0.079 *SD* per year. This translates to average 10-year differences in amount of decline of about 0.6–0.8 *SD*. Continuous childhood SES was associated with higher semantic memory at baseline, and the first and third quintiles for the categorical variable had lower average scores in comparison with the fifth quintile. The third and fourth quintile groups had lower average baseline executive function, and the other two quintile groups also were lower, but the differences were not significant.

Results showing effects of growth on cognitive outcomes are similarly presented in Table 6. Continuous growth was not significantly related to cognitive change or estimated baseline scores. Categorical growth was related to global cognitive change; rate of decline was significantly greater in the two lowest quintiles in comparison with the highest and differences approached significance for quintiles 3 and 4 ($p$s < .06). There was a trend for the lowest quintile group for growth to have the fastest rate of decline. These results correspond to the lowest quintile declining by about 1.0 *SD* more than the highest over 10 years, and the second quintile declining about 0.7 *SD* more.

Categorical variables appeared to better explain the relations of both childhood SES and growth with cognitive trajectories. Results for the categorical variables are summarized in Figure 2 for global cognitive change and in Figure 3 for semantic memory baseline.

A subsequent model included categorical childhood SES and categorical growth as joint independent variables along with covariates. Childhood SES was not related to global cognitive change, but effects of childhood SES on baseline cognition were substantially the same (results not shown). Growth effects were not significant in this model, and specifically, effects of growth on cognitive change were no longer significant.

To summarize, categorical variables derived from childhood SES and growth factors appeared to better explain relations with baseline cognition and cognitive change. High childhood SES was associated with better baseline scores. Low SES and low growth were associated with faster cognitive decline. These effects were independent of covariates that included ethnicity, education, gender, language, and

Table 5. Effects of Childhood SES Factor on Domain-Specific Cognitive Baseline Scores and Global Cognitive Change

| Independent variable | Global slope | Episodic memory baseline | Semantic memory baseline | Executive function baseline |
|---|---|---|---|---|
| Childhood SES—continuous | 0.017 (0.012) | 0.029 (0.069) | **0.168 (0.065)** | 0.034 (0.077) |
| Childhood SES—first quintile[a] | **−0.079 (0.032)** | −0.280 (0.169)[†] | **−0.583 (0.185)** | −0.265 (0.164) |
| Childhood SES—second quintile[a] | **−0.059 (0.030)** | −0.322 (0.183)[†] | −0.121 (0.195) | −0.293 (0.180) |
| Childhood SES—third quintile[a] | **−0.062 (0.024)** | −0.211 (0.181) | **−0.358 (0.173)** | **−0.336 (0.167)** |
| Childhood SES—fourth quintile[a] | **−0.077 (0.029)** | −0.316 (0.183)[†] | −0.120 (0.166) | **−0.398 (0.167)** |

*Notes.* Results are presented for continuous childhood SES and categorical recoding of continuous childhood SES (first to fourth quintiles). Tabled values are regression coefficients (standard errors in parentheses) from a parallel process latent growth model that estimated random effects for intercepts and slopes for all three cognitive outcomes but included a second-order latent variable that explained correlations among slopes (Global Slope). Ethnicity, education, gender, language of test administration, and apolipoprotein E were covariates (results not shown). Statistically significant ($p \le .05$) results are shown in bold.

[a]In comparison with fifth quintile as reference group.

[†]$p < .10$.

Table 6. Effects of Growth Factor on Domain-Specific Cognitive Baseline Scores and Global Cognitive Change

| Independent variable | Global slope | Episodic memory baseline | Semantic memory baseline | Executive function baseline |
|---|---|---|---|---|
| Growth—continuous | 0.021 (0.014) | 0.054 (0.069) | 0.094 (0.069) | 0.099 (0.073) |
| Growth—first quintile[a] | **–0.106 (0.040)** | –0.179 (0.172) | –0.246 (0.173) | –0.261 (0.186) |
| Growth—second quintile[a] | **–0.069 (0.035)** | 0.000 (0.167) | –0.268 (0.161)[†] | –0.143 (0.173) |
| Growth—third quintile[a] | –0.064 (0.033)[†] | –0.168 (0.160) | –0.163 (0.152) | –0.263 (0.175) |
| Growth—fourth quintile[a] | –0.053 (0.028)[†] | –0.097 (0.163) | –0.158 (0.145) | –0.036 (0.177) |

*Notes.* Results are presented for continuous growth and categorical recoding of continuous growth (first to fourth quintiles). Tabled values are regression coefficients (standard errors in parentheses) from a parallel process latent growth model that estimated random effects for intercepts and slopes for all three cognitive outcomes but included a second-order latent variable that explained correlations among slopes (Global Slope). Ethnicity, education, gender, language of test administration, and apolipoprotein E were covariates (results not shown). Statistically significant ($p \leq .05$) results are shown in bold.

[a]In comparison with fifth quintile as reference group.

[†]$p < .10$.

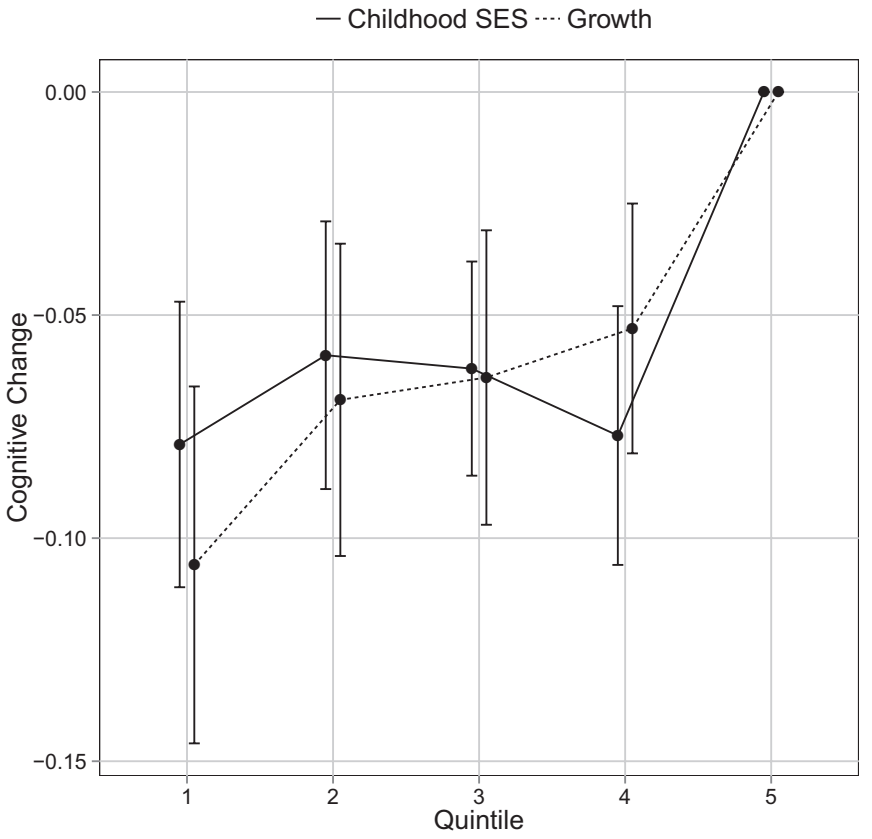


Figure 2. Cognitive change by categorical childhood SES and growth. Results show how groups defined by the first to fourth quintiles of the childhood SES and growth factor distributions differ in rate of global cognitive change from groups representing the fifth quintiles. Rate of cognitive change is a second-order latent variable that explained correlations among slopes estimated in a parallel process latent growth model. Separate models estimated results for childhood SES and growth. Demographic and apolipoprotein E covariates were included in both models.

APOE status. Growth effects were substantially attenuated by effects of childhood SES. Childhood SES effects on cognitive change also were attenuated when growth was added to the model, but childhood SES effects on baseline cognition were independent of growth.

## Discussion

We explored the association between markers of childhood development and late life cognition in a sample of Caucasian, African American, and Hispanic participants from northern California. We found good fit of a two-factor model reflecting growth and childhood SES. Growth was lower in the Hispanic group than the others and shared an inverse association with age. Childhood SES was significantly higher in Caucasians relative to the African American and Hispanic groups and was positively related to education. Growth was related to SES within the whole sample and in African Americans and Caucasians, but not in Hispanics. Using models that accounted for demographic variables and APOE status, we observed that lower childhood SES was associated with poorer cross-sectional

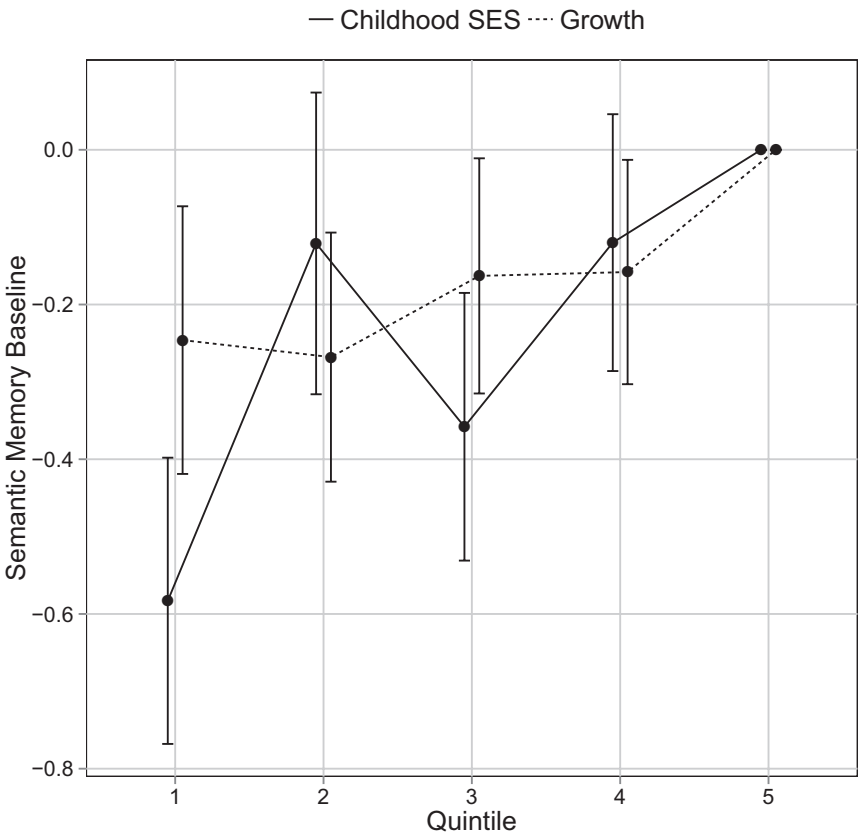


Figure 3. Semantic memory baseline by categorical childhood SES and growth. Results show how groups defined by the first to fourth quintiles of the childhood SES and growth factor distributions differ in baseline semantic memory from groups representing the fifth quintiles. Baseline semantic memory is a random effect estimated in a parallel process latent growth model. Demographic and apolipoprotein E covariates were included in both models.

measurement of semantic memory. Importantly, we also examined the impact of these factors on cognitive decline. We report that lower childhood SES is predictive of greater rate of cognitive decline and confirm previous studies suggesting that low growth is related to faster cognitive decline in late life. These findings are consistent with a model of cognitive aging in which early childhood development continues to exert an influence on cognition throughout life.

It has been proposed that the etiology of dementia may be considered from a life-span framework, whereby genetic and environmental factors interact throughout the lifetime in the development of dementia (Whalley, Dick, & McNeill, 2006). When proposing this model in 2006, Whalley and colleagues suggested that conceptually, low childhood SES would increase the risk of AD. However, available data at the time were underwhelming. Both the Chicago Health and Aging study, a study in community-dwelling Black and non-Black individuals residing in the Chicago area (Everson-Rose, Mendes de Leon, Bienias, Wilson, & Evans, 2003), and the Religious Order Study (Wilson, Scherr, Hoganson, et al., 2005) failed to detect an association between childhood SES and global cognitive decline. Null findings between childhood SES and global cognitive decline have since been replicated in the Chicago Health and Aging study (Wilson, Scherr, Bienias, et al., 2005). Providing support for the original theory, cross-sectional research has suggested that indicators of lower childhood SES share associations with late life cognition (Abbott et al., 1998; Zhang et al., 2009) and dementia risk (Borenstein et al., 2005; Fors, Lennartsson, & Lundberg, 2009; Kim, Stewart, et al., 2008; Moceri, Kukull, Emanuel, van Belle, & Larson, 2000; Ogunniyi et al., 2006; Rogers et al., 2009). In contrast, in a sample of French older adults, individuals at the highest SES bracket showed paradoxically more rapid decline in verbal fluency, but null findings were reported for nonverbal memory, executive, and global cognition (Glymour, Tzourio, & Dufouil, 2012). Consistent with the life-span model, we report here that lower childhood SES is indeed associated with increased rate of global cognitive decline in late life. In our sample, those with the lowest SES declined at a rate between 0.6 and 0.8 *SD*s faster compared with those in the top quintile over a 10-year period. This is notable given how distal early childhood events are to late life cognition. In addition, we controlled for education in all analyses, and education may partially mediate the association between early life experiences and adult cognition (Richards & Sacker, 2003). Our findings suggest that the social and family circumstances in which one develops may provide the foundation upon which additional risk and protective factors interact in the course of cognitive decline.

The reasons for the discrepancy between our finding that lower childhood SES predicts rate of cognitive decline and past research are not entirely clear. In the UC Davis Aging and Diversity cohort, participants reflect a large spectrum of Caucasian, African American, and Hispanic individuals. It has been proposed that for ethnic minorities in the United States, cognitive aging occurs in a context of local environment, migration patterns, SES and financial resources, education and occupation opportunities, and discrimination (Glymour & Manly, 2008). The Hispanic population in northern California reflects a mix of both native and foreign born individuals, with various levels of acculturation. In the current study, 47% of the Hispanic sample was Spanish monolingual and 42% was Spanish/English bilingual. The average years of education were 8 for the Hispanic group and 14 for the African American and Caucasian groups. Cognitive abilities were also varied, with 34% of the sample rated as having MCI or dementia. It appears that the participants in the UC Davis Aging Diversity Cohort reflect one of the more diverse cohorts in the United States. This greater diversity also yields greater variability, which may explain our ability to detect the association between adult cognition and distal factors.

Consistent with previous work, we observed that low growth was associated with a faster rate of cognitive decline. In our sample, those in the lowest quintile declined by a rate equivalent to 1.0 *SD* more than the highest quintile over a 10-year span. Our growth factor reflected height, leg length markers, and HC. Our work supports past research. Faster rate of decline was associated with smaller HC in a British cohort (Gale et al., 2003). Smaller HC was also associated with increased rate of decline in Korean samples after controlling for height (Lee, Cheong, et al., 2010; Lee, Eom, et al., 2010). Null findings between leg length and rate of cognitive decline were reported in an African-Caribbean sample (Mak, Kim, & Stewart, 2006). Lower HC was predictive of incident AD diagnosis in some studies (Borenstein et al., 2006; Borenstein Graves et al., 2001; Mortimer, Snowdon, & Markesbery, 2008) but not others (Espinosa et al., 2006). Although the specific measurements used to reflect growth vary across studies, the prevailing research is consistent with a life-span model. Growth, conceptualized to reflect genetics, childhood nutrition, and childhood medical illness, appears to provide a backdrop upon which cognitive aging occurs.

In addition to examining rate of global cognitive decline, we explored the association between growth and childhood SES and specific neuropsychological abilities. Growth was not significantly associated with episodic memory, semantic memory, or executive functioning. This is discrepant from a previous study using the present cohort that observed that intracranial cavity was associated with semantic, executive, and visuospatial abilities (Farias et al., 2012). That study additionally included participants recruited from the UC Davis Memory Clinic and thus had more participants with cognitive impairment than the current study. These differences in methodology may in part explain the discrepancy. Our findings are also in contrast to a study in the United Kingdom that found that larger HC was predictive of higher reasoning scores (Gale et al., 2003).

We observed that childhood SES was directly related to semantic memory and somewhat associated with executive functioning. This is consistent with findings from the Religious Order Study (Wilson, Scherr, Hoganson, et al., 2005), Rush Memory and Aging Project (Jefferson et al., 2011), and Kuopio Ischemic Heart Disease Risk Factor Study of Finland (Kaplan et al., 2001; Turrell et al., 2002) that higher childhood SES is associated with multiple neuropsychological domains. Semantic memory is a measure of knowledge of the world, and so it is not surprising that it tracks most closely with SES conditions (Farah et al., 2006; Noble, Norman, & Farah, 2005). Our finding with executive functioning was more complicated: the third and fourth quintiles had significantly lower performance than the fifth (highest SES) quintile, but no differences were observed between the fifth and bottom two quintiles. The frontal lobes continue to mature into the early 20s (Sowell, Thompson, Holmes, Jernigan, & Toga, 1999), and it may be that experiences in adolescence and early adulthood complicate this association. Future studies should clarify this finding.

Regarding our analytic approach, when growth was modeled as a continuous variable, we did not observe an association with any of the cognitive outcomes or with rate of global cognitive decline. However, based on previous research (e.g., Abbott et al., 1998; Graves et al., 1996; Kim, Lee, et al., 2008; Kim, Stewart, et al., 2008; Lee, Eom, et al., 2010; Maurer, 2010; Mortimer et al., 2003; Schofield et al., 1997), we divided the group into categories and observed that those in the smallest two growth quintiles showed the greatest rate of cognitive decline, with a trend toward faster decline in the middle two quintiles. Those with the lowest growth scores were presumably those individuals with the greatest environmental deprivation. It may be that as environmental conditions improve, there is a point in which full potential is realized (Bogin & Varela-Silva, 2010). Although one can continue to grow, further growth past this point no longer carries any information about the quality of the environment. Similarly, we found that childhood SES was best modeled using a categorical approach, suggesting that optimal development is associated with the top tier of SES as measured in this study.

Previous studies have shown that APOE e4 interacts with early childhood experience in predicting AD (Borenstein Graves et al., 2001; Kim, Lee, et al., 2008; Mortimer et al., 2008; Perneczky et al., 2012). APOE was not associated with growth or childhood SES in the present study. We controlled for APOE in all analyses, suggesting that early childhood development does indeed exert an effect on late life cognition over and above the risk conferred by APOE.

There are many strengths to the present investigation. We utilized psychometrically robust measures of cognition (Mungas et al., 2004; Mungas, Reed, Haan, et al., 2005; Mungas et al., 2000; Mungas, Reed, Tomaszewski Farias, et al., 2005). Study participants were recruited using targeted community-based methods (Hinton, 2010) and resulted in a diverse cross-section of the northern California population. We also utilized latent variable models that likely increased our measurement precision. Limitations include a relatively small sample size for evaluating effects of distal life experiences on late life cognition. Our findings suggest that early social and family circumstances influence trajectories of cognitive change extending into late life, but they do not address mechanism. Additionally, the Hispanic subgroup in our study may not be representative of the full population of U.S. Hispanics. Hispanics represent a heterogeneous group, with different countries of origin and races (U.S. Census Bureau, 2010). Hispanics in the present study were mostly of Mexican descent. Hispanics of Mexican origin/descent account for 63% of all Hispanics in the United States and represent the largest subgroup within this segment of the population (U.S. Census Bureau, 2010). Yet, other subgroups of Hispanics, such as those of Caribbean descent, may be different in terms of race and thus associated physical characteristics such as height. Future studies including other subgroups of Hispanics might help determine the generalizability of our findings to the broader population of Hispanics in the United States.

In sum, we observed that both low growth and childhood SES are significant predictors of rate of cognitive decline in old age. From a life-span perspective, it appears that cognitive aging acts on a foundation set fairly early in life. The social environment, which may reflect—among other factors—family structure, family/cultural values, and access to resources, provides a context in which physical development can occur. Physical development affected by genetics, nutrition, medical illness, and stress interacts with this local environment. These early life experiences provide the foundation upon which later risk and protective factors for cognitive decline develop. The life-span model predicts that although early life development is indeed an important part of cognitive aging, reduced quality of midlife experiences may be more important in predicting cognitive decline. This suggests that disparities in early life experiences may be countered by interventions provided throughout adulthood.

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Conflict of Interest

None of the authors have any conflicts of interest to disclose.

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