Stable longitudinal associations of family income with children's hippocampal volume and memory persist after controlling for polygenic scores of educational attainment

Laurel Raffington, Max Planck Institute for Human Development
Darina Czamara, Max Planck Institute of Psychiatry
Johannes Julius Mohn, Max Planck Institute for Human Development
Johannes Falck, Max Planck Institute for Human Development
Vanessa Schmoll, Max Planck Institute of Psychiatry
Christine Heim, Emory University
Elisabeth Binder, Emory University
Yee Lee Shing, Max Planck Institute for Human Development

Journal Title: DEVELOPMENTAL COGNITIVE NEUROSCIENCE
Volume: Volume 40
Publisher: ELSEVIER SCI LTD | 2019-12-01, Pages 100720-100720
Type of Work: Article | Final Publisher PDF
Publisher DOI: 10.1016/j.dcn.2019.100720
Permanent URL: https://pid.emory.edu/ark:/25593/vhfny

Final published version: http://dx.doi.org/10.1016/j.dcn.2019.100720

Copyright information:

© 2019

This is an Open Access work distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License (https://creativecommons.org/licenses/by-nc-nd/4.0/).

Accessed October 17, 2023 12:05 AM EDT
Stable longitudinal associations of family income with children’s hippocampal volume and memory persist after controlling for polygenic scores of educational attainment

Laurel Raffington a,b, Darina Czamara c, Johannes Julius Mohn a,d, Johannes Falck a, Vanessa Schmoll e, Christine Heim d,e,f, Elisabeth B. Binder c,h, Yee Lee Shing a,b,s

a Center for Lifespan Psychology, Max Planck Institute for Human Development, Berlin, Germany
b Department of Psychology, University of Texas at Austin, TX, USA
c Max Planck Institute of Psychiatry, Department of Translational Research in Psychiatry, Munich, Germany
d Berlin School of Mind and Brain, Humboldt-Universität zu Berlin, Berlin, Germany
e Charité - Universitätsmedizin Berlin, Institute of Medical Psychology, Berlin, Germany
f Penn State College of Medicine, Department of Biobehavioral Health, University Park, PA, USA
g Institute of Psychology, Goethe University Frankfurt, Frankfurt am Main, Germany
h Berlin School of Mind and Brain, Humboldt-Universität zu Berlin, Berlin, Germany
i Dept. of Psychiatry and Behavioral Sciences, Emory University School of Medicine, Atlanta, GA, USA

A R T I C L E   I N F O

Keywords:
Socioeconomic status
Memory
Hippocampus
Childhood
Longitudinal
Genetics

A B S T R A C T

Despite common notion that the correlation of socioeconomic status with child cognitive performance may be driven by both environmentally- and genetically-mediated transactional pathways, there is a lack of longitudinal and genetically informed research that examines these postulated associations. The present study addresses whether family income predicts associative memory growth and hippocampal development in middle childhood and tests whether these associations persist when controlling for DNA-based polygenic scores of educational attainment. Participants were 142 6–to–7-year-old children, of which 127 returned when they were 8–to–9 years old. Longitudinal analyses indicated that the association of family income with children’s memory performance and hippocampal volume remained stable over this age range and did not predict change. On average, children from economically disadvantaged background showed lower memory performance and had a smaller hippocampal volume. There was no evidence to suggest that differences in memory performance were mediated by differences in hippocampal volume. Further exploratory results suggested that the relationship of income with hippocampal volume and memory in middle childhood is not primarily driven by genetic variance captured by polygenic scores of educational attainment, despite the fact that polygenic scores significantly predicted family income.

1. Introduction

Longitudinal research investigating the relationship of socioeconomic status (SES; income, education, occupation) and children’s cognitive development is recently amassing. Children from socioeconomically disadvantaged background tend to have lower levels and slopes in general cognitive ability as well as multiple cognitive and achievement domains (Hackman et al., 2015; Lawson and Farah, 2017; von Stumm and Plomin, 2015; Wang et al., 2017). For instance, children growing up at-risk of poverty in the US perform nearly 1 SD below children not at-risk of poverty on achievement measures of verbal comprehension and math ability throughout middle childhood and early adolescence (Raffington et al., 2018a). Achievement disparities are rooted in differences in psychological characteristics, including self-control and motivation (Belsky et al., 2018; Malanchini et al., 2017), as well as more basic cognitive processes such as executive functions (Lawson et al., 2017), and episodic memory (Akshoomoff et al., 2014; Noble et al., 2007), which show moderately sized linear association with SES indicators in middle childhood.

A few studies have examined the repeated, time-lagged relationship of income and cognition with structural equation models to strengthen inferences on bivariate relationships (Hamaker et al., 2015). Indeed,
longitudinal changes in income predict child cognition in early childhood (Dearing et al., 2001) as well as in later childhood and early adolescence (Raffington et al., 2018a), but only for children growing up at-risk of poverty. This could suggest that income losses are especially detrimental to child cognitive development at the lower end of the income spectrum.

An increasing number of neuroscientific studies further suggest that SES indicators are positively correlated with children’s hippocampal volume (Brody et al., 2017; Ellwood-Lowe et al., 2018; Hair et al., 2015; Hanson et al., 2011; Jednorog et al., 2012; Luby et al., 2013; Merz et al., 2019; Noble et al., 2015, 2012; Raffington et al., 2018b; Yu et al., 2017), with the association between SES and hippocampal volume growing from ages 5–to–25–years (McDermott et al., 2019). Based on work in the animal literature, it is commonly theorized that smaller hippocampal volume in socioeconomically disadvantaged individuals may partially reflect differences in stimulating experiences and exposure to stress (Luby et al., 2013; Lupien et al., 2009). It is also known that hippocampal volume is partially heritable, thus SES–hippocampus volume associations could derive from passive gene–environment correlations (Sullivan et al., 2001). In either case, it is plausible to assume that SES disparities in hippocampal volume mediate SES disparities in memory performance, since learning and memory critically rely on the hippocampus and connected regions (Shing et al., 2010). However, evidence to suggest that hippocampal volume mediates SES–memory correlations is currently lacking.

More generally, the developmental relationship of hippocampal volume and memory functioning is not well–understood. For instance, a meta–analysis suggests that hippocampal volume has a negative association with memory in children and adults (Van Petten, 2004). Recent evidence suggests that structural hippocampal development continues beyond middle childhood, is non–linear in some subfield regions, and is complexly linked to different memory functions (Daugherty et al., 2016; Keresztes et al., 2017; J. K. Lee et al., 2014). However, the overwhelmingly cross–sectional nature of these studies has been shown to obscure true longitudinal developmental patterns (Kievit et al., 2013). Therefore, the coupling of hippocampal volume and memory is likely to differ along developmental time and remains largely obscure. Middle childhood is of particular interest given marked improvements in episodic memory performance (Ghetti and Bunge, 2012; Shing et al., 2010).

Individual differences in cognitive development that are commonly found to correlate with SES are known to be both environmentally and genetically transmitted (Belsky et al., 2018; Plomin and von Stumm, 2018). For instance, a higher income score at wave 1 predicts a larger gain from wave–1–to–wave–2 in associative memory and hippocampal volume. We further expected hippocampal volume to mediate the income–memory association. In addition, we performed non–preregistered exploratory analyses that add a PGS of educational attainment as a control variable and hypothesized that this would attenuate the income–memory and income–hippocampus associations, by having the PGS accounting for some of the variance that underlies these associations.

2. Method

2.1. Participants

142 children (66 girls) and their parents from 136 unique families (1 non–twin sibling pair, 4 dizygotic twin pairs, 1 monozygotic twin pair) participated in wave 1 of this longitudinal study (see Raffington et al. (2018b) for more details on sample). The children were identified by their parents as being of European (88%), European–African (4%), or European–Asian (6%) geographical ancestry (2% missing). 15% of this sample (13% at wave 2) were at-risk of poverty (monthly family net income at or below the Berlin state poverty line of that year, adjusted for family size and composition; Statistische Ämter des Bundes und der Länder, 2018). This is slightly less than the 19.2% of Berliners who were at-risk of poverty in 2017 (Statistische Ämter des Bundes und der Länder, 2018).

127 children (59 girls) from 121 unique families returned approximatley two years later for wave 2 (see Table 1 for descriptive statistics). Inclusion criteria at wave 1 included the child attending first or second grade, no psychiatric, developmental and physical health disorders, no prolonged steroid medication use, no parent–reported maltreatment or severe illness, at least 37 weeks gestation, and at least one fluent German–speaking parent. There were no exclusion criteria for wave 2. Nine children had a definite or probable medical diagnosis at wave 2 (e.g., ADHD, autism spectrum disorder). Excluding these children did not affect the results, thus they were retained. At wave 1, a subsample (n = 90) of randomly selected children balanced by gender and willing to participate in MRI was invited to scanning. At wave 2, all children were invited to scanning and 104 accepted. At wave 1, all participants were invited to participate in genome–wide DNA extraction for polygenic scoring and 118 contributed data. The study was approved by the ‘Deutsche Gesellschaft für Psychologie’ ethics committee (YLS_012015).

2.2. Procedure

At both waves, parents provided informed written consent and children verbal assent. While children completed the memory and other cognitive tasks not reported here, parents filled out a digitized questionnaire battery pertaining to SES and covariates. Children willing to participate in MRI were invited to scanning within 3 weeks.
2.3. Measures

2.3.1. Household income

Parents self-reported their total combined monthly household income after taxes (see Table 1 for descriptive statistics). There were no outliers over 5 SDs above or below the mean.

2.3.2. Memory

Participants completed exactly the same item–association memory task at both waves. They had to remember what location on a computer screen they had seen a black-colored object item (e.g., a shoe, lemon; adapted from Kessels et al., 2007). The targets were randomly selected from the stimuli pool and targets versus new items were screened to not be categorically or semantically closely related. For encoding, they were instructed to name the item and memorize at what location in a grid of 36 gray boxes they saw it. All children saw the same 15 pictures shown consecutively for 3 s at the respective same location with an interstimulus interval of 1 s. The experimenter then distracted the child for 60 s by asking them to name their favorite animals, foods, or toys. During retrieval, the child saw 30 items consecutively, of which 15 had been previously seen. They verbally responded whether they had seen the picture or not, and if yes, they pointed to the corresponding location. Prior to the task, participants completed a practice version with 3 items, which was repeated until they correctly located 2 of 3 items. A correct item–location matching was scored as 1 and an incorrect one as 0. The outcome variable was the proportion of correct locations from 15 trials. There were no outliers over 5 SDs above or below the mean at either wave.

2.3.3. Hippocampal volume

Structural MRI images were acquired on a Siemens Magnetom Trio Tim syngo 3 T scanner with a 12-channel head coil (Siemens Medical AG, Erlangen, Germany) using a 3D T1-weighted MPRAGE sequence (192 slices; field of view = 256 mm; voxel size = 1 mm³; TR = 2500 ms; TE = 3.69 ms; flip angle = 7°; TI = 1100 ms).

Volumetric segmentation was performed with the Freesurfer 6.0.0 image analysis suite (http://surfer.nmr.mgh.harvard.edu/) described elsewhere (Fischl, 2012). Previous studies suggest that software tools based on adult brain templates provide inaccurate segmentation for pediatric samples, which can be improved through the use of study–specific template brains (Phan, Smeets, Talcott, & Vandermoten, 2017; Schoemaker et al., 2016). We created two study-specific template brains (one for each wave) using Freesurfer’s “make_average_subject” command (https://surfer.nmr.mgh.harvard.edu/fswiki/make_average_subject). This pipeline utilizes the default adult template brain registrations of the “recon-all” command to average surfaces, curvatures, and volumes from all subjects into a study–specific template brain. All subjects were then re-registered to this study–specific template brain to improve segmentation accuracy. Segmented images were inspected for accuracy and 8 cases at wave 1 and 5 cases at wave 2 were excluded for inaccurate or failed registration due to excessive motion. The use of study–specific template brains was not preregistered. There were no outliers over 5 SDs above or below the mean.

2.3.4. Polygenic score for educational attainment

Genotyping was performed using Illumina GSA chips following the manufacturer’s protocol. After genotyping, we performed a stringent quality control using PLINK (https://www.cog-genomics.org/plink2, Chang et al., 2015) and removed any SNPs presenting with a call rate < 98%, a minor allele frequency below 1%, or a p-value for Hardy-Weinberg-Equilibrium below $1 \times 10^{-6}$. We calculated the identical-by-descent matrix (with a fraction of shared genotypes of at least 12.5%) and excluded the sibling sample with a lower call-rate from each sibling pair. We performed a MDS-analysis on the pruned genotypes (using the PLINK parameters –indep-pairwise 200 100 0.2) and removed any samples and the respective sib-pair identified as outliers (defined as presenting with a position on any of the first ten MDS-components, which deviated with at least 4 SDs from the respective mean of this component). Furthermore, we removed samples which presented with a heterozygosity rate deviating by at least 4 SDs from the mean heterozygosity over all samples.

Imputation was performed using shapeit2 (https://mathgen.stats.ox.ac.uk/genetics_software/shapeit/shapeit.html, O’Connell et al., 2014) and impute2 (https://mathgen.stats.ox.ac.uk/impute/impute_v2.html, Howie et al., 2009). After imputation, we only kept SNPs presenting with an info score metric of at least 0.6. This resulted in a dataset containing 9,629,396 imputed SNP genotypes and 118 samples.

PGS were calculated using PLINK and were based on the summary statistics of a GWAS of educational attainment by Lee et al. (J. J. Lee et al., 2018).

At first, we used LD-clumping on the best-guessed SNP genotypes based on these summary statistics and derived 464,967 independent SNPs. Afterwards, imputed genotype probabilities of these SNPs were extracted and PGS calculations were performed on these probabilities with the p-value threshold for inclusion of SNP being p = 1 and using the effect-estimates reported by Lee et al. as weights.

2.3.5. Missingness

At wave 1, logistic regression analyses showed that the final MRI subsample (n = 82) did not differ from the full sample in income or memory (p's > 0.14), but they were slightly older (mean difference 85 days, t = -3.12, p < 0.05). Those providing income data at wave 1 did not differ from families that did not in terms of their children’s wave 1 memory performance, hippocampal volume, or PGS (p’s > 0.79). Missingness in PGS was not predicted by age, sex, income, memory performance, or hippocampal volume at wave 1 (p’s > 0.26).

Longitudinally, missingness in income at wave 2 was not predicted by age, sex, income, memory, hippocampus, or PGS at wave 1 (p’s > 0.33). Similarly, missingness in memory at wave 2 was not predicted by age, sex, income, memory, hippocampus, or PGS at wave 1 (p’s > 0.33). Lastly, missingness in hippocampus at wave 2 was not predicted by age, sex, income, memory, hippocampus, or PGS (p’s > 0.14).

2.4. Data analysis

2.4.1. Confirmatory analyses

First, univariate latent difference score (LDS) structural equation
models (SEM) of income, memory and hippocampus were compiled (Ferrer and McArdle, 2010; Kievit et al., 2018). Aside from being a useful tool for longitudinal analyses, these SEMs also allowed the estimation of measurement error in hippocampal volume by building a latent hippocampal volume factor indicated by right and left volume. Unmarked paths were fixed at 1. Figure compiled using Onyx 1.0 (http://onyx.brandmaier.de).

![Figure 1](http://onyx.brandmaier.de)

**Fig. 1.** Graphical illustration of bivariate income–memory latent difference score model. Observed variables are depicted as squares, regressions as one-headed arrows, and (co-)variances (●) as two-headed arrows. Unmarked paths were fixed at 1. Figure compiled using Onyx 1.0 (http://onyx.brandmaier.de).

Third, the preregistered trivariate mediation model, including an indirect path of income onto hippocampus onto memory, was not tested, because such links were not indicated in the bivariate models.

All models were implemented in Mplus 8.2 and fitted using full information maximum likelihood (FIML) estimation to accommodate missing at random data. Models corrected standard errors for nesting of individuals within families (using the TYPE = COMPLEX feature in Mplus). Model fit was evaluated using the comparative fit index (CFI), root mean square error of approximation (RMSEA), and Chi-Squared ($\chi^2$) likelihood ratio test, where CFI values $> .95$ and RMSEA $< .08$ generally constitute good fit. Univariate and bivariate models showed good fit to the data (see Results Tables). Given that statistical tests were preregistered, no adjustments for multiple comparisons were made. We report standardized parameter estimates as effect size estimates.

### 2.4.2. Exploratory analyses

PGS of educational attainment was included as a predictor of intercepts and latent change in each domain in the bivariate models of income–memory and income–hippocampus. To test whether the covariance of income–memory was attenuated while controlling for PGS, a model fit comparison evaluated whether fit was significantly affected when the covariance parameter was free versus fixed to the parameter estimate from a model where the polygenic score paths were fixed to 0 (a df $\chi^2$–square test). PGS of educational attainment were regressed on sex, age, parent-reported geographical ancestry (European, European-African or European-Asian), and the first 10 MDS-components of the principal components analysis to control for population stratification.

### 3. Confirmatory results

#### 3.1. Univariate models

Income, memory performance and hippocampal volume showed average increasing trajectories over time (see Table 2 for fit indices and parameter estimates). Nevertheless, there were some decreasing individual trajectories in all domains (income: 22%; memory: 35%; hippocampus: 10%). Correspondingly, there was significant variability in intercepts and change in all domains.

#### Table 2

| Parameter Estimates from Three Separate Univariate Models |
|----------------------------------------------------------|
| **Income** | **Memory** | **Hippocampus** |
| Model Fit | $R^2$ ($df$) | 0 (1) | 2.17 (3) | 13.18 (15) |
| CFI | 1 | 1 | 1 |
| RMSEA (CI) | 0 (0-0) | 0 (0.13) | 0 (0-0.07) |
| SRMR | 0 | 0.04 | 0.07 |
| Mean change $\mu_m$ | 0.19$^*$ (0.06) | 0.57$^*$ (0.09) | 0.36$^*$ (0.05) |
| Intercept variance $\sigma^2_m$ | 1$^*$ (0.14) | 1$^*$ (0.11) | 0.33$^*$ (0.05) |
| Change variance $\sigma^2_c$ | 0.40$^*$ (0.13) | 1.28$^*$ (0.16) | 0.09$^*$ (0.03) |
| Correlated intercept-change $\rho_{mc}$ | -0.33$^*$ (0.09) | -0.59$^*$ (0.06) | -0.18 (0.14) |
| Age onto Intercept | 0.10 (0.07) | 0.19$^*$ (0.09) |
| Girl* onto Intercept | -0.02 | -0.16 (0.22) |
| ICV onto Intercept | - | - | 0.67$^*$ (0.11) |

Standardized regression estimates and bivariate correlations, unstandardized variance estimates. Standard errors in parentheses.

* Asterisks denote significance at the $a$ level of 0.05.

* Gender dummy coded as 1 = girls.

* Residual variances and residual correlations of left and right hippocampus as well as ICV variance and ICV correlations with gender are not shown.
3.2. Bivariate income and memory

Lower family income intercepts were associated with lower memory performance intercepts (see Table 3 for fit indices and parameter estimates). Accordingly, a 1 SD increase in family income (i.e. 2087 Euros/month; mean = 3634, range = 500–10000) was associated with 0.23 SD better performance in memory. Contrary to our first hypothesis, income score at wave 1 did not predict changes in memory or vice versa. Thus, while the association of income and memory remained stable over time, there was no dynamic longitudinal association between the two (see Fig. 2a).

3.3. Bivariate income and hippocampus

Lower family income intercepts were associated with smaller hippocampal volume intercepts (see Table 4 for fit indices and parameter estimates). Accordingly, a 1 SD increase in family income (i.e. 2087 Euros/month) was associated with 0.29 SD larger hippocampal volume, or 205 mm³. Contrary to our second hypothesis, income score at wave 1 did not predict changes in hippocampus or vice versa. Thus, the association of income and hippocampal volume remained stable over time (see Figure 2a).

3.4. Bivariate hippocampus and memory

There were no bivariate relationships between hippocampal volume and memory performance, therefore hippocampal volume did not mediate memory differences (see Table 5 for fit indices and parameter estimates).

4. Exploratory results

Contrary to our fourth hypothesis, adding children’s PGS of educational attainment did not attenuate the income–memory association (correlation with PGS control = 0.25 (0.09), p < 0.05 versus correlation without PGS control = 0.23 (0.09), p < 0.05, difference in correlation Chi-square (1) = 0.06, ns). In addition, PGS significantly predicted family income intercepts, but not income change, memory intercepts or memory change (see Table 6 for parameter estimates). Accordingly, a 1 SD increase in the children’s genetic predisposition to higher educational attainment was associated with living in a family that earned 536 Euros (0.26 SD’s) more per month. The association of family income intercepts and PGS persisted when constrained to participants of European descent (0.23 (0.09), p < 0.05).

Contrary to our fifth hypothesis, adding children’s PGS of educational attainment did not attenuate the income–hippocampus association (correlation with PGS control = 0.29 (0.10), p < 0.05 versus correlation without PGS control = 0.29 (0.00), p < 0.05, difference in correlation Chi-square (1) = 0.11, ns). In addition, PGS did not significantly predict hippocampus intercepts or change (see Table 7 for parameter estimates).

5. Discussion

Despite evidence that the association of SES and child cognitive performance is driven by both environmentally- and genetically-mediated transactional pathways, there has been little longitudinal and genetically informed research on this topic. Motivated by a lack of longitudinal research examining change and recent advances in using PGS derived from large GWAS, we applied longitudinal models to estimate dynamic associations of family income with children’s memory performance and hippocampal volume whilst controlling for genetic predispositions for educational attainment.

Our results indicate that the association of family income with children’s memory performance and hippocampal volume remains stable from ages 6 to 8 years. Contrary to hypotheses, change in hippocampal volume and associative memory was not predicted by family income. This null result could be due to sample composition, given the limited number of families living in poverty. On the other hand, widening socioeconomic differences over age across different cognitive and academic domains are often (Harden et al., 2019; Tucker-Drob, 2013), but not always found in middle childhood and adolescence (Hackman et al., 2015; Hair et al., 2015; Lawson and Farah, 2017; Raffington et al., 2018a; von Stumm and Plomin, 2015; Wang et al., 2017).

Yet, it should be noted that in studies with larger samples that allow an exploration of poverty moderation and more waves of data collection, it has been reported that changes in income predict child cognition in early childhood (Dearing et al., 2001) and later childhood and early adolescence (Raffington et al., 2018a), but only for children growing up at-risk of poverty. This could suggest threshold effects of the income–cognition association, such that being at-risk of poverty is a moderator in the longitudinal association of family income and children’s cognitive development. In the present study, we were not able to explore coupling effects of income changes onto cognitive development, for which at least three waves of data are necessary. We recommend future studies to consider a minimum of three data collection waves. Of note, intervention research has shown that positive outcomes in noncognitive domains (e.g., motivation, school achievement) may be present despite a lack of cognitive effects (Heckman, 2006). Hence, family income could have effects on change in noncognitive domains, even in children not at-risk of poverty.

Why do children from socioeconomically disadvantaged background show stably lower memory performance and a smaller hippocampal volume in middle childhood? One potential explanation is that shared genes predisposing the parents to make more earnings and the children to have a larger hippocampal volume and perform better on cognitive tasks account for their association, a phenomenon called gene–environment correlation (Plomin et al., 1977). Contrary to expectations, we found no evidence that genetic variance captured by PGS of educational attainment account for the correlation of income with children’s associative memory and hippocampal volume in middle childhood. This null result is surprising, given that those genetic differences did predict family income, which previous studies suggest is partially, but not fully, mediated by parental education (Belsky et al., 2016). Put simply, children with a higher genetic predisposition to attain more education tend to have parents with a higher genetic predisposition to make more earnings and the children genes predisposing the parents to make more earnings and the children have effects on change in noncognitive domains, even in children not at-risk of poverty.

Table 3

| Parameter | Income | Memory |
|-----------|--------|--------|
| Model Fit | R² = 2.96, df = 10, CFI = 1, ΔCFI = 0, ΔCI = 0.00, SRMR = 0.03 |       |
| Mean change µ_i | 0.19 (0.06) | 0.57 (0.09) |
| Intercept variance σ | 1.0 (0.14) | 1.0 (0.11) |
| Change variance σ_δ | 0.39 (0.12) | 1.26 (0.16) |
| Correlated intercept–change δ | -0.29 (0.09) | -0.57 (0.06) |
| Age onto Intercept | -0.10 (0.07) |       |
| Girl onto Intercept | -0.01 (0.11) |       |
| Bivariate Couplings |       | -0.23 (0.09) |
| Intercept correlation PGS | -0.12 (0.08) |       |
| Income onto memory change PGS | -0.17 (0.10) |       |
| Change-change correlation PGS | -0.01 (0.06) |       |

Standardized regression estimates and bivariate correlations, unstandardized variance estimates. Standard errors in parentheses.

* Asterisks denote significance at the α level of 0.05.

a Gender dummy coded as 1 = girls.
We believe two other mechanisms are likely to be involved in the relationship of family income with children’s hippocampal volume and memory performance in middle childhood: First, genetic variance not captured by PGS of educational attainment, such as genetic variance of hippocampal structure, confer a gene–environment correlation and, second, socioeconomic disadvantage occurring earlier in development offsets a lower trajectory that results in a fairly stable difference in later childhood. For instance, socioeconomic–related stress in prenatal and early childhood development may initiate long–lasting maturational neural processes along a different course to maximize functioning in those environments, potentially at the cost of certain cognitive functions preferred in cognitive testing and academic contexts. Correspondingly, intervention efforts have a substantially larger impact on cognitive and school achievement when they target children in early compared to later childhood (Duncan et al., 1994, 1998; Heckman, 2006). Indeed, both genetically and environmentally–mediated effects transferred through family and neighborhood environments influence children’s cognitive development and academic attainment (Belsky et al., 2018; Engelhardt et al., 2019; Harden et al., 2019; Koellinger and Harden, 2018). Thus, PGS of educational attainment combine genetic effects mediated via the home environment and transactional gene–environment correlations (Cheesman et al., 2019). Interestingly, these transactional mechanisms may differ across the socioeconomic spectrum, for instance by school quality (Harden et al., 2019). Future research should investigate intervention or quasi–experimental effects in combination with PGS as a powerful way to explore the ways in which socioeconomic disadvantage and genetic predispositions contribute to individual differences in cognitive development.

Furthermore, there was no evidence to suggest that differences in memory performance were mediated by differences in hippocampal volume, since both intercepts and change over time were unrelated to each other. Another study reports null associations of changes in episodic memory allowed to correlate with changes in the gray matter volume of frontal and parietal cortex areas in 8–to–38–year–olds (Breukelaar et al., 2017). The lack of mediation may arise from partially non–linear linkages between hippocampal subfield structure and memory performance (Keresztes et al., 2017, 2018), or brain and

---

**Fig. 2.** Individual raw monthly post–tax income in Euros (a), memory performance in proportion correct (b), and bilateral hippocampal volume in mm$^3$(c) plotted over time. Average trajectories are plotted for families earning +1 SD above mean income (blue line) and -1 SD below mean income (red line), where income was averaged over wave 1 and 2 (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).
cognition more generally (Wenger et al., 2017). Therefore, it is possible that longitudinal trajectories of subregions of the hippocampus are related to specific memory functions not captured in our memory task. Alternatively, hippocampal function may be more closely related to associative memory than its structure, and its functional engagement may be moderated by SES (Farah, 2017; Leonard et al., 2015; Sheridan et al., 2013).

We acknowledge further limitations of this study. First, our sample was somewhat biased in attracting parents that were more highly educated than the population average, and only included children that passed stringent exclusion criteria (see Raffington et al., 2018b). Second, the moderate sample size did not allow us to explore threshold effects of growing up in poverty. These limitations are likely to underestimate effects of SES and poverty on child development. Similarly, a lack of power could be a potential factor contributing to null results, since all tested factors only explain a low amount of the total variance. Third, our analyses were restricted to income–hippocampus–memory associations and may not generalize to parental education (Duncan and Magnuson, 2012) or other cognitive functions that the hippocampus is known to be involved in, such as emotion regulation (Lupien et al., 2009). Lastly, our analysis was restricted by only having two waves of data, which limits the reliability of change and understanding of longitudinal dynamics (Willett, 1989). Nevertheless, representing longitudinal assessments of change in each variable as an outcome of the other variable’s prior score or vice versa informs our understanding of bivariate relationships far beyond cross-sectional or longitudinal correlations.

Table 4: Bivariate income–hippocampus parameter estimates.

| Model Fit | Income | Hippocampus |
|-----------|--------|-------------|
| $\chi^2 = 14.48$, $df = 27$, CFI = 1, $E_\alpha = 0$, CI = 0.00, SRMR = 0.04 | | |
| Mean change $\mu_A$ | 0.20 * (0.05) | 0.37 * (0.05) |
| Intercept variance $\sigma$ | 1.0 (0.14) | 0.33 * (0.05) |
| Change variance $\sigma_A$ | 0.39 * (0.13) | 0.09 * (0.03) |
| Correlated intercept–change $\delta$ | -0.35 * (0.08) | -0.13 (0.14) |
| Age onto Intercept | -0.17 (0.09) | - |
| Girl onto Intercept | -0.16 (0.14) | - |
| ICV onto Intercept | 0.66 * (0.08) | - |
| Bivariate Couplings | | |
| Intercept correlation $p_{\text{inc}}$ | 0.29 * (0.09) | - |
| Income onto hippocampus change $\gamma_{\text{inc}}$ | -0.15 (0.15) | - |
| Hippocampus onto income change $\gamma_{\text{inc}}$ | 0.12 (0.09) | - |
| Change–change correlation $\rho_{\text{inc}}$ | -0.02 (0.15) | - |

Table 5: Bivariate hippocampus–memory parameter estimates.

| Model Fit | Income | Memory |
|-----------|--------|--------|
| $\chi^2 = 22.34$, $df = 24$, CFI = 1, $E_\alpha = 0$, CI = 0.00, SRMR = 0.07 | | |
| Mean change $\mu_A$ | 0.37 * (0.05) | 0.57 * (0.09) |
| Intercept variance $\sigma$ | 0.33 * (0.06) | 0.99 * (0.11) |
| Change variance $\sigma_A$ | 0.08 * (0.03) | 1.25 * (0.16) |
| Correlated intercept–change $\delta$ | -0.19 (0.17) | 0.59 * (0.06) |
| Age onto Intercept | 0.18 * (0.09) | 0.09 (0.08) |
| Girl onto Intercept | -0.15 (0.23) | 0.02 (0.11) |
| ICV onto Intercept | 0.67 * (0.11) | - |
| Bivariate Couplings | | |
| Intercept correlation $p_{\text{inc}}$ | 0.11 (0.11) | - |
| Hippocampus onto memory change $\gamma_{\text{inc}}$ | 0.11 (0.09) | - |
| Memory onto hippocampus change $\gamma_{\text{inc}}$ | -0.31 (0.17) | - |
| Change–change correlation $\rho_{\text{inc}}$ | 0.01 (0.09) | - |

Table 6: Income–memory parameter estimates for whole sample with polygenic scores for educational attainment.

| Model Fit | Income | Memory |
|-----------|--------|--------|
| $\chi^2 = 84$, $df = 131$, CFI = 1, $E_\alpha = 0$, CI = 0.00, SRMR = 0.07 | | |
| Mean change $\mu_A$ | 0.19 * (0.05) | 0.57 * (0.09) |
| Intercept variance $\sigma$ | 0.94 * (0.14) | 1.1 (0.11) |
| Change variance $\sigma_A$ | 0.38 * (0.12) | 1.24 * (0.17) |
| Correlated intercept–change $\delta$ | -0.27 * (0.09) | -0.56 * (0.06) |
| Age onto Intercept | -0.11 (0.07) | -0.02 (0.11) |
| Bivariate Couplings | | |
| Intercept correlation $p_{\text{inc}}$ | 0.25 * (0.09) | - |
| Income onto memory change $\gamma_{\text{inc}}$ | -0.14 (0.08) | - |
| Memory onto income change $\gamma_{\text{inc}}$ | -0.17 (0.09) | - |
| Change–change correlation $\rho_{\text{inc}}$ | -0.01 (0.06) | - |
| Polygenic Scores | | |
| Polygenic scores on income intercept | 0.23 * (0.08) | - |
| Polygenic scores on income change | -0.14 (0.07) | - |
| Polygenic scores on memory intercept | -0.06 (0.09) | - |
| Polygenic scores on memory change | 0.10 (0.10) | - |
| Girl on polygenic scores | 0.10 (0.20) | - |
| Age on polygenic scores | 0.13 (0.08) | - |
| Geographical ancestry on polygenic scores | 0.15 (0.15) | - |

Table 7: Income–hippocampus parameter estimates for whole sample with polygenic scores for educational attainment.

| Model Fit | Income | Hippocampus |
|-----------|--------|-------------|
| $\chi^2 = 143$, $df = 184$, CFI = 1, $E_\alpha = 0$, CI = 0.00, SRMR = 0.09 | | |
| Mean change $\mu_A$ | 0.20 * (0.05) | 0.37 * (0.05) |
| Intercept variance $\sigma$ | 0.94 * (0.13) | 0.32 * (0.05) |
| Change variance $\sigma_A$ | 0.38 * (0.13) | 0.08 * (0.03) |
| Correlated intercept–change $\delta$ | -0.33 * (0.09) | -0.13 (0.13) |
| Age onto Intercept | -0.18 (0.09) | -0.16 (0.13) |
| Girl onto Intercept | -0.16 (0.13) | -0.66 * (0.08) |
| ICV onto Intercept | | |
| Bivariate Couplings | | |
| Intercept correlation $p_{\text{inc}}$ | 0.29 * (0.10) | - |
| Income onto hippocampus change $\gamma_{\text{inc}}$ | -0.13 (0.15) | - |
| Hippocampus onto income change $\gamma_{\text{inc}}$ | 0.13 (0.09) | - |
| Change–change correlation $\rho_{\text{inc}}$ | -0.03 (0.16) | - |
| Polygenic Scores | | |
| Polygenic scores on income intercept | 0.23 * (0.08) | - |
| Polygenic scores on income change | -0.14 (0.08) | - |
| Polygenic scores on hippocampus intercept | 0 (0.09) | - |
| Polygenic scores on hippocampus change | -0.14 (0.16) | - |
| Girl on polygenic scores | 0.08 (0.20) | - |
| Age on polygenic scores | 0.13 (0.08) | - |
| Geographical ancestry on polygenic scores | 0.16 (0.15) | - |

Standardized regression estimates and correlations, unstandardized variance estimates. Standard errors in parentheses.

*Residual variances and residual correlations of left and right hippocampal volume as well as ICV variance and ICV correlation with gender are not shown.

Asterisks denote significance at the $\alpha$ level of 0.05.

Gender dummy coded as 1 = girls.

Principal components correcting for population stratification onto polygenic scores are not shown for brevity.

Standardized regression estimates and correlations, unstandardized variance estimates. Standard errors in parentheses.

Asterisks denote significance at the $\alpha$ level of 0.05.

Gender dummy coded as 1 = girls.

Principal components correcting for population stratification onto polygenic scores are not shown for brevity.
In conclusion, we found the association of family income with children’s memory performance and hippocampal volume to be stable from ages 6–to–8 years without bivariate effects on change. Accordingly, children from economically disadvantaged background on average showed lower memory performance and had smaller hippocampal volumes. There was no evidence to suggest that differences in memory performance were mediated by differences in hippocampal volume. The relationship of income with hippocampal volume and memory in middle childhood was not driven by genetic variance captured by PGS of educational attainment, despite the fact that PGS significantly predicted family income. Furthermore, change in hippocampal volume and memory performance observed in middle childhood seems largely independent of family income, at least in samples of moderate SES variation. Their stable association may derive from socioeconomic disadvantage occurring in earlier childhood and genetic variance not captured by PGS of educational attainment. This study also highlights the utility of including DNA-based PGS as control variables to zero-in on explanatory mechanisms involved in the study of childhood adversity and development to promote positive development.

Declaration of Competing Interest

None.

Acknowledgments

Funding: This work was supported by the Jacobs Foundation [grant 2014–1151 to YLS and CH] and conducted at the Center for Lifespan Psychology, Max Planck Institute for Human Development. LR is supported by the German Research Foundation (DFG, RA 3208-1/1). We thank all members of the Jacobs study team for their vital contribution, and all participants and family members for taking part in the study.

References

Aksuwo, N., Newman, E., Thompson, W.K., McCabe, C., Blox, C.S., Chang, L., et al., 2014. The NIH Toolbox Cognition Battery: Results from a large normative developmental sample (PING). Neuropsychology 28 (1), 1–10. https://doi.org/10.1037/neu.0000001.
Belsky, D.W., Domingue, B.W., Wedow, R., Arsenault, L., Boardman, J.D., Caspi, A., et al., 2018. Genetic analysis of social-class mobility in five longitudinal studies. Proc. Natl. Acad. Sci. 115 (31), 6275–6278. https://doi.org/10.1073/pnas.1801238115.
Breukelaar, I.A., Antees, C., Grieve, S.M., Foster, S.L., Gomes, L., Williams, L.M., 2017. Accounting for child poverty as control variables to zero-in on explanatory influences on developmental trajectories in adolescent girls. Dev. Cogn. Neurosci. 30 (December 2017), 41–50. https://doi.org/10.1016/j.dcn.2017.12.005.
Engelhardt, L.E., Church, J.A., Paige Harden, K., Tucker-Drob, E.M., 2019. Accounting for the shared environment in cognitive abilities and academic achievement with measured sociocultural contexts. Dev. Sci. 22 (1), e12699 https://doi.org/10.1111/1467-9287.12699.
Fan, X., 2003. Power of latent growth modeling for detecting group differences in linear growth trajectory parameters. Struct. Equ. Model. A Multidiscip. J. 10 (3), 380–400. https://doi.org/10.1080/10731911.2003.1011702.
Farrer, J., 2017. The neuroscience of socioeconomic status: correlates, causes, and consequences. Neuron 96 (1), 56–71. https://doi.org/10.1016/j.neuron.2017.08.034.
Ferrer, E., McArrell, J.J., 2004. An experimental analysis of dynamic hypotheses about cognitive abilities and achievement from childhood to early adulthood. Dev. Psychol. 40 (6), 925–932. https://doi.org/10.1037/1464-6477.40.6.935.
Ferrer, E., McArrell, J.J., 2010. Longitudinal modeling of developmental changes in psychological research. Curr. Dir. Psychol. Sci. 19 (3), 149–154. https://doi.org/10.1177/0963721410370300.
Fischl, B., 2012. Neuroimage 62 (2), 774–781. https://doi.org/10.1016/j.neuroimage.2012.01.021.
Ghetti, S., Bunge, S.A., 2012. Neural Changes Underlying Episodic Memory During Middle Childhood 2, 381–395.
Hackman, D.A., Gallop, R., Evans, G.W., Farah, M.J., 2015. Socioeconomic status and executive function: developmental trajectories and mediation. Dev. Sci. 18 (5), 686–702. https://doi.org/10.1111/desc.12246.
Hair, N.E., Hanson, J.L., Wolfe, B.L., Pollak, S.D., 2015. Association of child poverty, brain development, and academic achievement. JAMA Pediatr. 169 (9), 822–829. https://doi.org/10.1001/jamapediatrics.2015.1475.
Hamaker, E.L., Kuirip, R.M., Grauman, R.P.P.F., 2015. A critique of the cross-lagged panel model. Psychol. Methods 20 (1), 102–116. https://doi.org/10.1037/a0038889.
Hanson, J.L., Chandra, A., Wolfe, B.L., Pollak, S.D., 2011. Association between income and the hippocampus. PLoS One 6 (5), e18712. https://doi.org/10.1371/journal.pone.0018712.
Harden, K.P., Domingue, B.W., Belsky, D.W., Boardman, J.D., Crosnoe, R., Malanchini, M., et al., 2019. Genetic associations with mathematics tracking and persistence in secondary school. BioRxiv, 598532. https://doi.org/10.1101/598532.
Heckman, J.J., 2006. Skill formation and the economics of investing in disadvantaged children. Science 312 (5782), 1900–1902. https://doi.org/10.1126/science.1128898.
Hill, W.D., Hagenan, S.P., Marioni, R.E., Harris, S.E., Liewald, D.C.M., Davies, C., et al., 2016. Molecular genetic contributions to social deprivation and household income in UK Biobank. Curr. Biol. 26 (22), 3083–3089. https://doi.org/10.1016/j.cub.2016.09.035.
Howie, B.N., Donnelly, P., Marchini, J., 2009. A flexible and accurate genotype imputation method for the next generation of genome-wide association studies. PLoS Genet. 5 (6), e1000529. https://doi.org/10.1371/journal.pgen.1000529.
Jednoróg, K., Altarelli, I., Monzalvo, K., Flux, J., Dubois, J., Billard, C., et al., 2012. The influence of socioeconomic status on children’s brain structure. PLoS One 7 (8), e42486. https://doi.org/10.1371/journal.pone.0042486.
Keretzes, A., Bender, A.R., Bodammer, N.C., Lindeberger, U., Shing, Y.L., Werkle-Bergner, M., 2017. Hippocampal maturity promotes memory distinctiveness in childhood and adolescence. Proc. Natl. Acad. Sci. 114 (34), 9212–9217. https://doi.org/10.1073/pnas.1716541144.
Keretzes, A., Ngo, C.T., Lindeberger, U., Werkle-Bergner, M., Newcombe, N.S., 2018. Hippocampal maturation driven memory from generalization to specificity. Trends Cogn. Sci. (Regul. Ed.) 22 (8), 676–686. https://doi.org/10.1016/j.tics.2018.05.004.
Kessel, R.P., Hobbel, D., Postma, A., 2007. Aging, context memory and binding: a review of cognitive memory in young and older adults. Int. J. Neurosci. 117 (6), 795–810. https://doi.org/10.1080/00207650600910218.
Kievit, R.A., Brandmaier, A.M., Ziegler, G., van Harmelen, A.L., de Mook, S.M.M., Moutoussis, M., et al., 2018. Developmental cognitive neuroscience using latent change score models: a tutorial and applications. Dev. Cogn. Neurosci. 33 (2017), 99–117. https://doi.org/10.1016/j.dcn.2017.10.001.
Kievit, R.A., Frankenhuis, W.E., Waldorp, L.J., Borsboom, D., 2013. Simpson’s paradox in psychological science: a practical guide. Front. Psychol. 4 (August), 1–14. https://doi.org/10.3389/fpsyg.2013.00515.
Koeninger, P.D., Harden, K.P., 2018. Using nature to understand nurture. Science 359 (6374), 386–387. https://doi.org/10.1126/science.aat4297.
Lawrence, M.G., Farah, M.J., 2017. Executive function as a mediator between SES and academic achievement throughout childhood. Int. J. Behav. Dev. 41 (1), 94–104. https://doi.org/10.1177/0165025416650349.
Lawrence, M.G., Hook, C.J., Farah, M.J., 2017. A meta-analysis of the relationship between socioeconomic status and executive function performance among children. Developmental Science, Advance on e12529. https://doi.org/10.1111/desc.12529.
Lee, J.J., Wedow, R., Okbay, A., Kong, E., Maghian, O., Zacher, M., et al., 2018. Gene discovery and polygenic prediction from a genome-wide association study of
educational attainment in 1.1 million individuals. Nat. Genet. 50 (8), 1112–1121. https://doi.org/10.1038/nrg2688-018-01473-3.

Lee, J.K., Ekstrom, A.D., Gatti, S., 2014. Volume of hippocampal subfields and episodic memory in childhood and adolescence. NeuroImage 94, 162–171. https://doi.org/10.1016/j.neuroimage.2014.03.019.

Leonard, J.A., Mackey, A.P., Finn, A.S., Gabrieli, J.D.E., 2015. Differential effects of socioeconomic status on working and procedural memory systems. Front. Hum. Neurosci. 9, 554. https://doi.org/10.3389/fnhum.2015.00554.

Luby, J.L., Belden, A., Botteron, K., Marrus, N., Harm, M.P., Babb, C., et al., 2013. The effects of poverty on childhood brain development: the mediating effect of caregiving and stressful life events. JAMA Pediatr. 167 (12), 1135–1142. https://doi.org/10.1001/jamapediatrics.2013.3139.

Lupien, S.J., McEwen, B.S., Gunnar, M.R., Heim, C.M., 2009. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat. Rev. Neurosci. 10 (6), 434–445. https://doi.org/10.1038/nrn2639.

Malanchini, M., Wang, Z., Voronin, I., Schenker, V.J., Plomin, R., Petrill, S.A., Kovas, Y., 2017. Reading self-perceived ability, enjoyment and achievement: a genetically informative study of their reciprocal links over time. Dev. Psychol. 53 (4), 698–712. https://doi.org/10.1037/dev0000209.

McClelland, G.H., Judd, C.M., 1993. Statistical difficulties of detecting interactions and moderator effects. Psychol. Bull. 114 (2), 376–390. https://doi.org/10.1037/0033-2909.114.2.376.

McDermott, C.L., Seidlitz, J., Nadig, A., Liu, S., Clasen, L.S., Blumenthal, J.D., et al., 2019. Longitudinally mapping childhood socioeconomic status associations with cortical and subcortical morphology. J. Neurosci. 39 (8), 1365–1373. https://doi.org/10.1523/JNEUROSCI.1808-18.2019.

Merz, E.C., Desai, P.M., Maskus, E.A., Melvin, S.A., Rehman, R., Torres, S.D., et al., 2019. Socioeconomic disparities in chronic physiologic stress are associated with brain structure in children. Biol. Psychiatry. https://doi.org/10.1016/j.biopsych.2019.05.024.

Noble, K.G., Houston, S.M., Brito, N.H., Bartsch, H., Kan, E., Kuperman, J.M., et al., 2015. Family income, parental education and brain structure in children and adolescents. Nat. Neurosci. 18 (5), 773–778. https://doi.org/10.1038/nn.3983.

Noble, K.G., Houston, S.M., Kan, E., Sowell, E.R., 2012. Neural correlates of socioeconomic status in the developing human brain. Dev. Sci. 15 (4), 516–527. https://doi.org/10.1111/j.1467-6864.2012.01147.x.

Noble, K.G., McCannells, B.D., Farah, M.J., 2007. Socioeconomic gradients predict individual differences in neurocognitive abilities. Dev. Sci. 10 (4), 464–480. https://doi.org/10.1111/j.1467-6864.2007.00600.x.

O’Connell, J., Gurdasani, D., Delanoe, A., Pirastu, N., Ulivi, S., Cucca, M., et al., 2014. A general approach for haplotype phasing across the full spectrum of relatedness. PLoS Genet. 10 (4), e1004234 https://doi.org/10.1371/journal.pgen.1004234.

Plomin, R., DeFries, J.C., Loehlin, J.C., 1977. Genotype-environment interaction and correlation in the analysis of human behavior. Psychol. Bull. 84 (2), 309.

Plomin, R., von Stumm, S., 2018. The new genetics of intelligence. Nat. Rev. Genet. 19 (3), 148–159. https://doi.org/10.1038/nrg.2017.104.

Raffington, L., Prindle, J.J., Shing, Y.L., 2018. Income gains predict cognitive functioning longitudinally throughout later childhood in poor children. Developmental Psychology, Advance online. https://doi.org/10.1037/dev0000529.

Raffington, L., Prindle, J., Keresztes, A., Binder, J., Heim, C.M., Shing, Y.L., 2018. Blunted cortisol stress reactivity in low-income children relates to lower memory function. Psychoneuroendocrinology 90, 110–121. https://doi.org/10.1016/j.psyneuen.2018.02.002.

Rinderman, H., Ceci, S.J., 2018. Parents’ education is more important than their wealth in shaping their children’s intelligence: results of 19 samples in seven countries at different developmental levels. J. Educ. Gift. 41 (4), 298–326. https://doi.org/10.1177/0162353217799481.

Sheridan, M.A., How, J., Arausjo, M., Schamber, M.A., Nelson, C.A., 2013. What are the links between maternal social status, hippocampal function, and HPA axis function in children? Dev. Sci. 16 (5), 665–675. https://doi.org/10.1111/desc.12087.

Shing, Y.L., Werkle-Bergner, M., Brehm, Y., Muller, Y., Li, S.C., Lindenberger, U., 2010. Episodic memory across the lifespan: the contributions of associative and strategic components. Neurosci. Biobehav. Rev. 34 (7), 1080–1091. https://doi.org/10.1016/j.neubiorev.2009.11.002.

Small, B.J., Dixon, R.A., McArdle, J.J., Grimm, K.J., 2013. Do changes in lifestyle engagement moderate cognitive decline in normal aging? Evidence from the Victoria longitudinal Study. Neuropsychology 26 (2), 144–155. https://doi.org/10.1037/a0026579.

Statistische Amt der Bundes und der Länder, 2018. Sozialberichterstattung Der Amtlichen Statistik 2017. Retrieved from http://www.Amtliche-sozialberichterstattung.ung.de/A2armutsgefaehrdungsschwellen.html.

Sullivan, E.V., Pfeiferbaum, A., Swan, G.E., Carmelli, D., 2001. Heritability of hippocampal size in elderly twin men: equivalent influence from genes and environment. Hippocampus 11 (6), 754–762. https://doi.org/10.1002/hipo.1091.

Tucker-Drob, E.M., 2013. How many pathways underlie socioeconomic differences in the development of cognition and achievement? Learn. Individ. Differ. 25 (5), 12–20. https://doi.org/10.1016/j.lindif.2013.01.015.

Tucker-Drob, E.M., Briley, D.A., 2014. Continuity of genetic and environmental influences on cognition across the life span: a meta-analysis of longitudinal twin and adoption studies. Psychol. Bull. 140 (4), 949–979. https://doi.org/10.1037/a0035893.

Van Petten, C., 2004. Relationship between hippocampal volume and memory ability in healthy individuals across the lifespan: review and meta-analysis. Neuropsychologia 42 (10), 1394–1413. https://doi.org/10.1016/j.neuropsychologia.2004.04.006.

Wang, Z., Soden, B., Deater-Deckard, K., Lukowski, S.L., Schenker, V.J., Willcutt, E.G., et al., 2017. Development in reading and math in children from different SES backgrounds: the moderating role of child temperament. Dev. Sci. 20 (3), e12380 https://doi.org/10.1111/desc.12380.

Wenger, E., Brozolli, C., Lindenberger, U., Lisdén, M., 2017. Expansion and renormalization of human brain structure during skill acquisition. Trends Cogn. Sci. (Regul. Ed.) 21 (12), 930–939. https://doi.org/10.1016/j.tics.2017.09.008.

Willett, J.B., 1989. Some results on reliability for the longitudinal measurement of change: implications for the design of studies of individual growth. Educ. Psychol. Meas. 49 (3), 587–602. https://doi.org/10.1177/001316448904900309.

Yu, Q., Daugherty, A.M., Anderson, D.M., Nishimura, M., Brush, D., Hardwick, A., et al., 2017. Socioeconomic status and hippocampal volume in children and young adults. Dev. Sci. e12561. https://doi.org/10.1111/desc.12561.