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

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Abstract

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.

Keywords: socioeconomic status; memory; hippocampus; childhood; longitudinal; genetics

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, Gallop, Evans, & Farah, 2015; Lawson & Farah, 2017; von Stumm & 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, Prindle, & Shing, 2018). 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, Hook, & Farah, 2017), and episodic memory (Akshoomoff et al., 2014; Noble, McCandliss, & Farah, 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, Kuiper, & Grasman, 2015). Indeed, longitudinal *changes* in income predict child cognition in early childhood (Dearing, McCartney, & Taylor, 2001) as well as in later childhood and early adolescence (Raffington, Prindle, & Shing, 2018), 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, Hanson, Wolfe, & Pollak, 2015; Hanson, Chandra, Wolfe, & Pollak, 2011; Jednoróg et al., 2012; Luby et al., 2013; Merz et al., 2019; Noble et al., 2015; Noble, Houston, Kan, & Sowell, 2012; Raffington, Prindle, Keresztes, et al., 2018; 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, McEwen, Gunnar, & Heim, 2009). It is also known that hippocampal volume is partially heritable, thus SES–hippocampus volume associations could derive from passive gene–environment correlations (Sullivan, Pfefferbaum, Swan, & Carmelli, 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 relies 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, Bender, Raz, & Ofen, 2016; Keresztes et al., 2017; J. K. Lee, Ekstrom, & Ghetti, 2014). However, the overwhelmingly cross–sectional nature of these studies has been shown to obscure true longitudinal developmental patterns (Kievit, Frankenhuis, Waldorp, & Borsboom, 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 & 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 & von Stumm, 2018; Tucker-Drob & Briley, 2014). Recently, the use of genome–wide polygenic scores (PGS) has been validated as a way to account for a small proportion of the variance in cognitive functioning that is due to genetic factors (Belsky et al.,

2018; Plomin & von Stumm, 2018). PGS aggregate the effects of thousands of genetic variants based on weights calculated from very large genome-wide association studies (GWAS) and can be applied in samples several orders smaller than necessary for GWAS (i.e., of 100 participants; Plomin & von Stumm, 2018). For example, PGS of educational attainment have been found to predict educational attainment, but also cognitive, psychological, and socioeconomic characteristics over the life course (Belsky et al., 2018). Indeed, the genetic factors associated with income and education may be very similar (Davies, Hemani, Timpson, Windmeijer, & Davey Smith, 2015; Hill et al., 2016). It remains unexplored whether PGS of educational attainment accounts for some of the covariance between SES and brain structure in children.

The present study consists of (1) a preregistered confirmatory section that addresses whether family income predicts memory growth and hippocampal development in childhood to elucidate longitudinal dynamics and, (2) an exploratory section that explores whether these associations persist when adding children's PGS of educational attainment as a control variable to examine whether the association is driven by a gene-environment correlation. Although parental education is a better predictor of children's general cognitive ability than income and wealth (Rindermann & Ceci, 2018), income is the most volatile SES indicator over time (Duncan, Ziol-Guest, & Kalil, 2010) and change in income may be an important predictor of cognitive development (Raffington, Prindle, & Shing, 2018). Therefore, we focus on family income, due to its dynamic nature over time.

In the first section, we examine our preregistered hypotheses (<https://osf.io/7f42h>) with one major deviation: Upon further consideration of statistical power necessary for interactions (Fan, 2003; McClelland & Judd, 1993), the present sample size, and expected effect sizes no larger than a standardized parameter estimate of 0.20 (Raffington, Prindle, & Shing, 2018), we did not test for the initially hypothesized moderating effects of poverty grouping. Rather, we followed a continuous approach and hypothesized that 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, Prindle, et al. (2018) 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 approximately two years later (mean, range) 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.

Table 1

Descriptive statistics and correlations across time (wave 1 and 2) of measures of interest.

| | | Mean (SD) n | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|--|----------------|-------|-------|-------|-------|-------|-------|-------|-------|
| 1 | Income 1 ^a (Euros) n = 137 | 3634 (2087) | 0.81* | 0.24* | 0.07 | 0.21 | 0.14 | 0.02 | -0.06 | 0.24* |
| 2 | Income 2 (Euros) n = 124 | 4089 (2128) | – | 0.10 | 0.06 | 0.24* | 0.21* | -0.02 | -0.03 | 0.14 |
| 3 | Memory 1 (proportion correct) n = 142 | 0.41 (0.15) | | – | 0.34* | -0.08 | -0.15 | 0.04 | -0.03 | -0.04 |
| 4 | Memory 2 (proportion correct) n = 127 | 0.5 (0.15) | | | – | 0.02 | 0.07 | 0.17 | 0.15 | 0.02 |
| 5 | Hippo 1 (mm ³) n = 82 | 7888 (709) | | | | – | 0.90* | 0.34* | 0.28* | 0.12 |
| 6 | Hippo 2 (mm ³) n = 99 | 8130 (8130) | | | | | – | 0.28* | 0.19 | 0.08 |
| 7 | Age 1 (years) n = 142 | 7.19 (0.46) | | | | | | – | 0.93* | 0.13 |
| 8 | Age 2 (years) n = 127 | 9.25 (0.45) | | | | | | | – | 0.08 |
| 9 | Polygenic Score n = 118 | 1.94 (2.86) | | | | | | | | – |

* Pearson’s correlation $p < 0.05$.

2.3.2. Memory

Participants completed exactly the same item–association memory task at both waves. They had to remember at what location on a computer screen they had seen a black–colored sketched item (e.g., a shoe, lemon; adapted from Kessels, Hobbel, & Postma, 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 sec at the respective same location with an interstimulus interval of 1 sec. The experimenter then distracted the child for 60 sec 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 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 TrioTim syngo 3 Tesla 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 et al., 2002). 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, & Vandermosten, 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-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 1×10^{-5} . 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, Donnelly, & Marchini, 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 & 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. Individual growth is described by wave 1 (i.e., the intercept exemplified by Income[1] in Figure 1) and a person's change over time, which is directly modeled as the unobserved difference between the initial observation and subsequent observations (Δ_i). The random intercept is modeled with variance (σ_i) to indicate between-person differences in intercepts. Similarly, the random latent change component is modeled with variance (σ_{Δ_i}) to indicate between-person differences in change. The overall effect of change may be positive or negative depending on parameter estimates and the previously observed score. A group average trajectory is estimated by the mean latent change parameter (μ_{Δ_i}). Models included gender and age as covariates of memory and hippocampus intercepts. All variables were standardized to the measure of the first wave, hence the mean intercept is 0 and wave 2 measures represent the deviation from wave 1 (Raffington, Prindle, & Shing, 2018; Small, Dixon, McArdle, & Grimm, 2013).

Second, bivariate models of income–memory (see Figure 1 for graphical depiction) and income–hippocampus were compiled. We also assembled a bivariate hippocampus–memory model (this step was not explicitly preregistered, but is a necessary step to explore mediation). To test whether intercepts covaried, their covariance was estimated (ρ_{mi}). To examine whether income score at wave 1 predicts memory change from wave 1 to wave 2, change in memory was regressed on income at wave 1 ($\gamma_{i\Delta m}$). In reverse, to examine whether memory score at wave 1 predicts income change from wave 1 to wave 2, change in income was regressed on memory at wave 1 ($\eta_{m\Delta i}$). Thus, evaluation of score–onto–change coupling allow for inferences to be made between income at wave 1 being a leading indicator in time of wave–1–to–wave–2 changes in memory and vice versa (Ferrer & McArdle, 2004). These models also allowed for residual covariation between income and memory latent change variables ($\rho_{\Delta m \Delta i}$). The income–hippocampus and hippocampus–memory models were assembled in the same manner.

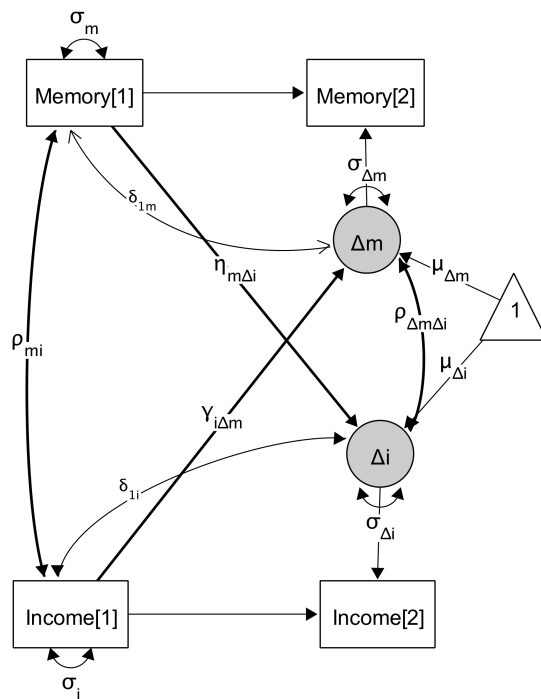


Figure 1. Graphical illustration of bivariate income–memory latent difference score model. Observed variables are depicted as squares, regressions as one-headed arrows, and (co-)variances (σ) as two-headed arrows. Unmarked paths were fixed at 1. Figure compiled using Onyx 1.0 (<http://onyx.brandmaier.de>).

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 (E_a), and Chi-Squared (χ^2) likelihood ratio test, where CFI values $> .95$ and $E_a < 0.08$ 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 1 *df* Chi–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 ^c |
|--------------------------------------|---------------|------------------|---------------|--------------------------|
| Model Fit | χ^2 (df) | 0 (1) | 2.17 (3) | 13.18 (15) |
| | CFI | 1 | 1 | 1 |
| | E(CI) | 0 (0-0) | 0 (0.13) | 0 (0-0.07) |
| | SRMR | 0 | 0.04 | 0.07 |
| Mean change μ_{Δ} | | 0.19* (0.06) | 0.57* (0.09) | 0.36* (0.05) |
| Intercept variance σ^b | | 1* (0.14) | 1* (0.11) | 0.33* (0.05) |
| Change variance σ_{Δ}^b | | 0.40* (0.13) | 1.28* (0.16) | 0.09* (0.03) |
| Correlated intercept-change δ | | -0.33* (0.09) | -0.59* (0.06) | -0.18 (0.14) |
| Age onto Intercept | | – | 0.10 (0.07) | 0.19* (0.09) |
| Girl ^a onto Intercept | | – | -0.02 (0.11) | -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 α level of 0.05.

^a Gender dummy coded as 1 = girls.

^c Residual variances and residual correlations of left and right hippocampus as well as ICV variance and ICV correlations with gender are not shown.

3.2.1 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 Figure 2a).

Table 3. Bivariate income–memory parameter estimates

| | Income | Memory |
|--|---|---------------|
| Model Fit | $\chi^2 = 2.96, df = 10, CFI = 1, E = 0, CI = 0-0, SRMR = 0.03$ | |
| Mean change μ_{Δ} | 0.19* (0.06) | 0.57* (0.09) |
| Intercept variance σ | 1* (0.14) | 1* (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 ^a onto Intercept | – | -0.01 (0.11) |
| Bivariate Couplings | | |
| Intercept correlation ρ_{mi} | 0.23* (0.09) | – |
| Income onto memory change $\gamma_{i\Delta m}$ | -0.12 (0.08) | – |
| Memory onto income change $\eta_{m\Delta i}$ | -0.17 (0.10) | – |
| Change–change correlation $\rho_{\Delta m \Delta i}$ | -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.

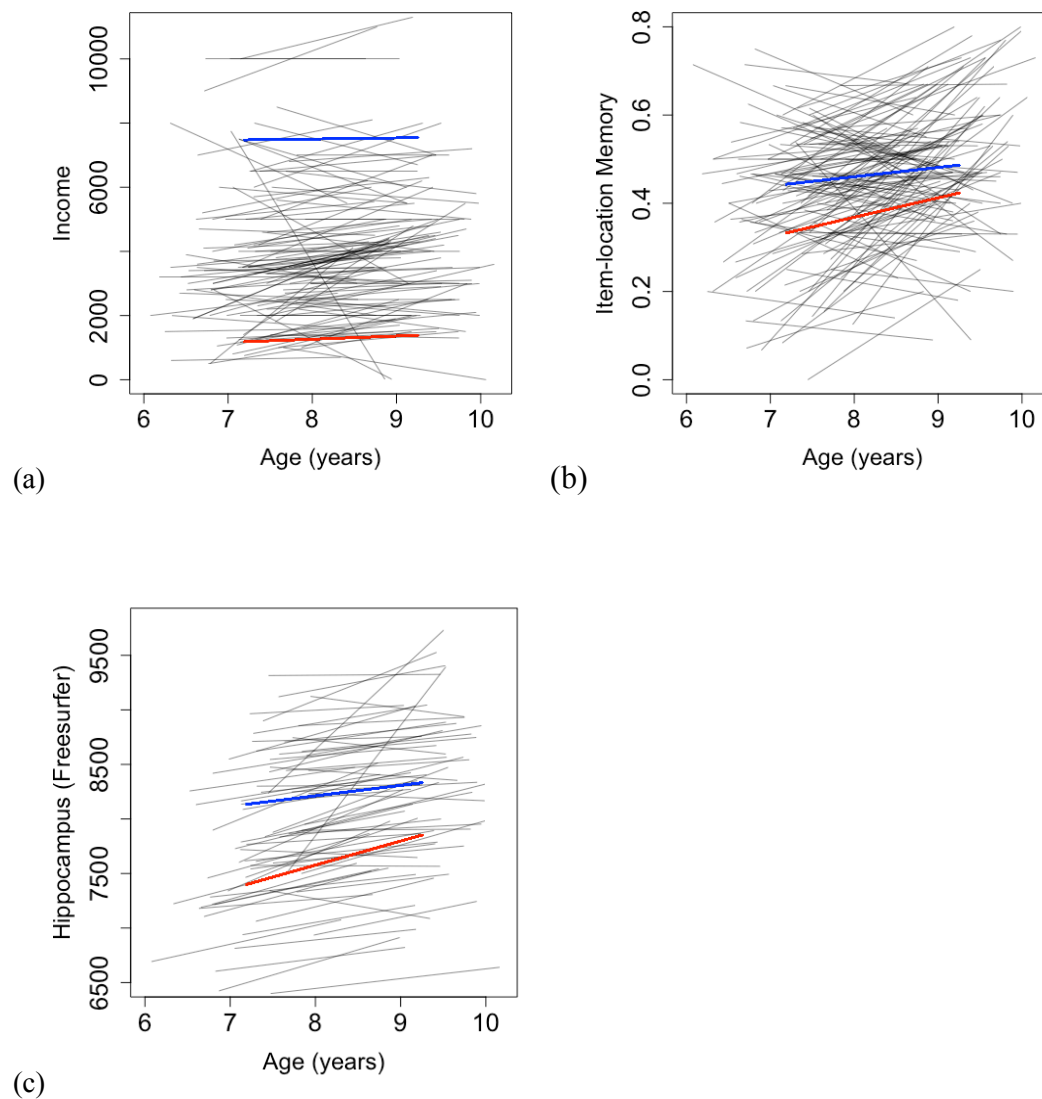


Figure 2. Individual raw monthly post-tax income in Euros (a), memory performance in proportion correct (b), and bilateral hippocampal volume in mm^3 (c) plotted over time. Average trajectories are plotted for families earning +1 SD above mean income (blue line) and -1 SD below mean income (red line), where income was averaged over wave 1 and 2.

3.2.2. 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 mm^3 . 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 4a).

Table 4. Bivariate income–hippocampal volume parameter estimates

| | Income | Hippocampus |
|--|--|--------------|
| Model Fit | $\chi^2 = 14.48, df = 27, CFI = 1, E = 0, CI = 0-0, SRMR = 0.04$ | |
| Mean change μ_{Δ} | 0.20* (0.05) | 0.37* (0.05) |
| Intercept variance σ | 1* (0.14) | 0.33* (0.05) |
| Change variance σ_{Δ} | 0.39* (0.13) | 0.09* (0.03) |
| Correlated intercept–change δ | -0.35* (0.08) | -0.13 (0.14) |
| Age onto Intercept | – | 0.17 (0.09) |
| Girl ^a onto Intercept | – | -0.16 (0.14) |
| ICV onto Intercept | – | 0.66* (0.08) |
| Bivariate Couplings | | |
| Intercept correlation ρ_{mi} | 0.29* (0.09) | – |
| Income onto hippocampus change $\gamma_{i\Delta m}$ | -0.15 (0.15) | – |
| Hippocampus onto income change $\eta_{m\Delta i}$ | 0.12 (0.09) | – |
| Change–change correlation $\rho_{\Delta m \Delta i}$ | -0.02 (0.15) | – |

Standardized regression estimates and 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.

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

3.2.3. 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).

Table 5. Bivariate hippocampus–memory parameter estimates

| | Hippocampus | Memory |
|--|---|---------------|
| Model Fit | $\chi^2 = 22.34, df = 24, CFI = 1, E = 0, CI = 0-0.06, SRMR = 0.07$ | |
| Mean change μ_{Δ} | 0.37* (0.05) | 0.57* (0.09) |
| Intercept variance σ | 0.33* (0.06) | 0.99* (0.11) |
| Change variance σ_{Δ} | 0.08* (0.03) | 1.25* (0.16) |
| Correlated intercept–change δ | -0.19 (0.17) | -0.59* (0.06) |
| Age onto Intercept | 0.18* (0.09) | 0.09 (0.08) |
| Girl ^a onto Intercept | -0.15 (0.23) | 0.02 (0.11) |
| ICV onto Intercept | 0.67* (0.11) | – |
| Bivariate Couplings | | |
| Intercept correlation ρ_{mi} | 0 (0.11) | – |
| Hippocampus onto memory change $\gamma_{i\Delta m}$ | 0.11 (0.09) | – |
| Memory onto hippocampus change $\eta_{m\Delta i}$ | -0.31 (0.17) | – |
| Change–change correlation $\rho_{\Delta m \Delta i}$ | 0.01 (0.09) | – |

Standardized regression estimates and 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.

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

1. 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), $p < 0.05$).

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

| | Income | Memory |
|--|---|---------------|
| Model Fit | $\chi^2 = 84$, $df = 131$, $CFI = 1$, $E_s = 0$, $CI = 0-0$, $SRMR = 0.07$ | |
| Mean change μ_{Δ} | 0.19* (0.05) | 0.57* (0.09) |
| Intercept variance σ | 0.94* (0.14) | 1* (0.11) |
| Change variance σ_{Δ} | 0.38* (0.12) | 1.24* (0.17) |
| Correlated intercept–change δ | -0.27* (0.09) | -0.56* (0.06) |
| Age onto Intercept | – | 0.11 (0.07) |
| Girl ^a onto Intercept | – | -0.02 (0.11) |
| Bivariate Couplings | | |
| Intercept correlation ρ_{mi} | 0.25* (0.09) | – |
| Income onto memory change $\gamma_{i\Delta m}$ | -0.14 (0.08) | – |
| Memory onto income change $\eta_{m\Delta i}$ | -0.17 (0.09) | – |
| Change–change correlation $\rho_{\Delta m \Delta i}$ | -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 ^b | 0.15 (0.15) | – |

Standardized regression estimates and 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.

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

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).

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

| | Income | Hippocampus |
|--|---|--------------|
| Model Fit | $\chi^2 = 143, df = 184, CFI = 1, E_s = 0, CI = 0-0, SRMR = 0.09$ | |
| Mean change μ_{Δ} | 0.20* (0.05) | 0.37* (0.05) |
| Intercept variance σ | 0.94* (0.13) | 0.32* (0.05) |
| Change variance σ_{Δ} | 0.38* (0.13) | 0.08* (0.03) |
| Correlated intercept–change δ | -0.33* (0.09) | -0.13 (0.13) |
| Age onto Intercept | – | 0.18* (0.09) |
| Girl ^a onto Intercept | – | -0.16 (0.13) |
| ICV onto Intercept | – | 0.66* (0.08) |
| Bivariate Couplings | | |
| Intercept correlation ρ_{mi} | 0.29* (0.10) | – |
| Income onto hippocampus change $\gamma_{i\Delta m}$ | -0.13 (0.15) | – |
| Hippocampus onto income change $\eta_{m\Delta i}$ | 0.13 (0.09) | – |
| Change–change correlation $\rho_{\Delta m \Delta i}$ | -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 ^d | 0.16 (0.15) | – |

Standardized regression estimates and 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.

^c Residual variances and residual correlations of left and right hippocampus as well as ICV variance and ICV correlation with gender are not shown.

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

2. 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 & Farah, 2017; Raffington, Prindle, & Shing, 2018; von Stumm & 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, Prindle, & Shing, 2018), 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, DeFries, & Loehlin, 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 same 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 more education, and these parents are more highly educated, which results in higher family income (Belsky et al., 2018). Thus, our results provide no evidence for the notion that the correlation of family income with memory performance or hippocampal volume is driven by a gene-environment correlation captured by PGS of educational attainment.

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, Brooks-Gunn, & Klebanov, 1994; Duncan, Yeung, Brooks-Gunn, & Smith, 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, Church, Paige Harden, & Tucker-Drob, 2019; Harden et al., 2019; Koellinger & 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; Keresztes, Ngo, Lindenberger, Werkle-Bergner, & Newcombe, 2018), or brain and cognition more generally (Wenger, Brozzoli, Lindenberger, & Lövdén, 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, Mackey, Finn, & Gabrieli, 2015; Sheridan, How, Araujo, Schamber, & Nelson, 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, Prindle, Keresztes, et al., 2018). 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 & 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.

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.

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5. References

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