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Title: Blunted cortisol stress reactivity in low–income children relates to lower memory function

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Highlights

- Lower income is associated with a blunted CAR and blunted stress reactivity in children
- Hyporeactivity in stress reactivity is related to lower memory among lower–income children only
- Smaller hippocampal volume in lower income was not associated with poorer memory or cortisol

Abstract

Lower socioeconomic status (SES) environments are marked by higher stress that is hypothesized to alter cortisol secretion in children, thereby damaging hippocampal volume and memory performance. However, empirical evidence demonstrating these putative links is lacking. We assessed the diurnal cortisol awakening response (CAR) on two mornings and cortisol stress reactivity (CSR) with the Trier Social Stress Test for Children in 102 healthy, socio–demographically diverse 6–to–7–year–old children (46% female). Children performed a hippocampal–dependent item–location associative memory task and 60 of these children underwent structural MRI scanning for hippocampal volume. Cortisol values were modeled with latent–change structural equation models to represent overall levels and change. We found lower income is associated with a flatter CAR, blunted reactivity and recovery to acute stress, and smaller hippocampal volume. Furthermore, hyporeactivity in CSR was related to lower memory among lower–income children, whereas there was no reliable association of CSR and memory among higher–income children (an income x cortisol interaction). We found no evidence that smaller hippocampal volume in lower income was associated with poorer memory performance. Notably, hyporeactivity in both CAR and CSR was specific to using income as the SES predictor. The income x cortisol interaction and smaller hippocampal effects, however, were replicated with education and an SES composite score.

This suggests that hyporeactivity to acute stress may function as a mediator in SES–cognition associations at the lower end of the SES spectrum, but it does not imply environmental– or genetically–mediated causation.

Keywords: cortisol awakening response; cortisol stress reactivity; memory; socioeconomic status; stress; middle childhood

1. Introduction

Families of low socioeconomic status (SES, indicated by income, education, occupation) experience higher instances of stress, such as more adverse life events (Hackman et al., 2010). Stress in lower SES is postulated to influence children's cortisol secretion and neural and cognitive development, however vigorous evidence is lacking (Lupien et al., 2009).

Studies in children have found lower SES or related chronic stressors, such as parenting stress or cumulative risk, to be associated with both higher basal cortisol levels (Blair et al., 2011; Chen et al., 2010; Lupien et al., 2001, 2000) and lower basal cortisol levels (Badanes, Watamura, & Hankin, 2011; Chen & Paterson, 2006; Kliewer, Reid–Quinones, Shields, & Foutz, 2008; Pagliaccio et al., 2014; Wagner et al., 2016) in lower SES (Badanes et al., 2011; Blair et al., 2011; Chen et al., 2010; Chen and Paterson, 2006; Kliewer et al., 2008; Lupien et al., 2000) or more stressed healthy children (Pagliaccio et al., 2014; Wagner et al., 2016). These inconclusive results may partly reflect that this literature has been characterized by unreliable cortisol measurements, age variability, and a neglect of diurnal dynamics. For example, basal cortisol indices show considerably less intra–individual stability than repeated diurnal cortisol measurements, such as the cortisol awakening response (CAR; Rotenberg et al., 2012). The CAR reflects both reactivity to awakening and circadian regulation that is influenced by state factors and stable individual differences (Stalder et al.,

2015). The few studies that have looked at diurnal cortisol secretion indicate reductions in total diurnal cortisol secretion including the CAR (Raffington et al., 2018) and flatter diurnal cortisol slopes (Martin et al., 2016; Wolf et al., 2008) in more stressed (Martin et al., 2016; Raffington et al., 2018; Wolf et al., 2008) and lower SES healthy children (Wolf et al., 2008). We know of no study exploring SES predictors on prepubescent children's CAR, although a blunted CAR has been reported in low–SES adolescent girls (McFarland and Hayward, 2014).

More so, cortisol stress reactivity (CSR) in response to the Trier Social Stress Test for Children (TSST–C; Buske–Kirschbaum et al., 1997) is rarely studied, although its validity compared to other stress challenges and health outcomes has made it the gold standard paradigm in human stress research. Both very high and blunted stress reactivity is considered maladaptive. The CSR is largely unrelated to the CAR, here collectively called 'reactivity', and differentially affected by individual differences (Bouma et al., 2009). Two developmental studies report lower CSR (Badanes et al., 2011; Kraft and Luecken, 2009) associated with low SES–related stress, although null results have also been reported (Hostinar et al., 2015; Sheridan et al., 2013) and no studies examined the effects of altered CSR on cognition. Null results may partly derive from threshold effects, since the gradient of SES–disparities in neural structure and cognitive functioning is steeper at the lower end and some associations are specific to low–SES (Hair et al., 2015). Accordingly, the weighting of causes underlying SES–cognition associations may not be the same along the entire SES spectrum (Farah, 2017). Taken together, it is not established whether lower SES is associated with cortisol reactivity dysregulation in prepubescent children's CAR and CSR.

Additionally, little is known about the impact of aberrant cortisol secretion on cognition in children. Long–term memory is of specific interest, because SES has been positively associated with incidental learning (Noble et al., 2007) and episodic memory (Akshoomoff et al., 2014). Long–term memory critically relies on the hippocampus and prefrontal cortex (PFC) (Shing et al., 2010), regions involved in regulating the stress response and rich in glucocorticoid receptors, which renders them sensitive to stress hormones (Popoli

et al., 2012). Animal models provide causal evidence that stress exposure affects glucocorticoid secretion, showing patterns of glucocorticoid hyperresponsive or hyporesponsive dysregulation, depending on the type and frequency of the stressor (e.g., repeated maternal separation, social isolation), and age of exposure (McEwen, 2000; Sánchez et al., 2001). Glucocorticoid dysregulation in turn makes the hippocampus and PFC vulnerable to adverse neuroplastic changes that reduce neural volume and impair memory (Popoli et al., 2012). Accordingly, lower SES is related to smaller hippocampal volume (Brody et al., 2017; Hair et al., 2015; Hanson et al., 2011; Jednoróg et al., 2012; Luby et al., 2013; Noble et al., 2015, 2012a, 2012b; Rao et al., 2010; Yu et al., 2017). Functionally, rodent models and adult human studies suggest that glucocorticoids and memory have an inverted U–shaped relationship, with optimal memory performance at medium levels (Ursache and Noble, 2016). Thus, animal models imply that cortisol dysregulation and hippocampal structure are important candidates in linking stress–related SES disparities to individual differences in memory.

This study aims to explore evidence of this pathway in children by exploring SES–related disparities in cortisol reactivity and hippocampal structure of 102 6–to–7–year–old children in relation to their associative memory performance. To identify the exact role of cortisol secretion at work in human memory is important to garner a mechanistic understanding of individual differences that may inform intervention research. First, we collected state–of–the–art measures of both the CAR and CSR to the TSST–C in a socio–demographically diverse sample. Second, we use latent–change structural equation models (SEMs) to separate the effects of cortisol *levels* whilst representing within–person *change* (reactivity), which are the two principal components of diurnal cortisol secretion across multiple studies (Khoury et al., 2015). SEM models, such as those used in this study, also significantly reduce the number of cortisol outcome variables and statistical comparisons, because stress reactivity and recovery can be modeled in one latent slope. Third, we follow Farah's (2017) recommendations to increase comparability across studies by reporting effects of each SES indicator. Lastly, children completed an associative memory task that required

binding of an item to a specific location, which is known to engage the hippocampus particularly (Sander et al., 2012).

We first hypothesized that lower SES would be associated with lower memory functioning, lower CARs and stress hyporeactivity. Second, we predicted that cortisol dysregulation would associate with lower memory performance. Third, we hypothesized that lower SES would be related with smaller hippocampal volume that predicts lower memory performance. Lastly, we explored SES X Cortisol and SES X Hippocampus interactions to test for gradient versus threshold effects in cortisol–memory associations, with no directional hypotheses.

2. Method

2.1. Participants.

For recruitment, 7000 general research invitation letters were sent to families with 6–to–7–year–old children in Berlin, randomly selected from three higher and three lower SES districts, of which 288 families indicated potential interest in participation. These families were telephone screened for inclusion criteria including the child attending first or second grade, no psychiatric, developmental and physical health disorders, no prolonged steroid medication use, no traumatic childhood experiences (e.g., maltreatment, severe illness), at least 37 weeks gestation, and one fluent German–speaking parent.

A total of 147 children and parents participated in the baseline measurement of an ongoing longitudinal study, of which 5 children chose to discontinue their participation during the first session (final sample n = 142). 102 children (*M* = 7.16 years, *SD* = 0.46, range = 6.08 – 7.98, 46% female) were randomly assigned to the TSST–C. The other children functioned as a control group for an experimental task (not reported here) exploring effects of acute stress. 17% of this sample was at–risk of poverty (< 1767 Euros/month), similar to the 18% of Berliners at–risk of poverty under 18 years (Amt für Berlin-Brandenburg Statistik, 2015). However, the parents were more highly educated (56% had a higher education degree compared to 36%; Abel, 2015) and more likely employed (5% had an unemployed parent

compared to 9.8%; Statista, 2016) as compared to the average Berlin population. 92% of the parents identified their children's geographical ancestry as 'European'. Five children were bilingual, 2 trilingual, and 9 were raised by single parents (for descriptive statistics, see Table 1). Sixty of these children (whom did not differ in demographics, all p's > 0.28) underwent MRI scanning for hippocampal volume. The study was approved by the 'Deutsche Gesellschaft für Psychologie' ethics committee (YLS_012015).

2.2. Procedure

Parents were instructed not to give their children large meals or caffeine for 2 hours before the session, scheduled between 2–6pm on weekdays. Parents provided informed written consent and children verbal assent. While children completed the memory followed by the TSST–C task, parents filled out a digitized questionnaire battery pertaining to SES and covariates. At the end of the approximately 2.5 hour session, parents were instructed in collecting saliva samples at home. A subsample of randomly selected children willing to participate in MRI was invited to scanning within 3 weeks as a voluntary study extension.

2.3. Measures

2.3.1. *Socioeconomic Status*

Parents self–reported their total combined monthly household income after taxes and education degree from 1 = none to 13 = PhD with 9 or more indicating higher education (see Table 1 for descriptive statistics). Occupational status was self–reported from 1 = never employed to 15 = high civil service. We chose to focus on maternal, not paternal, education and occupation to reduce the number of SES predictors as they were strongly correlated (education $r = 0.64$, occupation $r = 0.41$, p's < 0.05). SES indicators were moderately correlated with each other (income with education $r = 0.44$, and occupation $r = 0.27$; education with occupation $r = 0.58$, all p's < 0.05). An SES composite score was computed based on a single factor analysis of family income, maternal education and occupational status (see Appendix A). All predictors were standardized for analyses.

2.3.2. *Cortisol awakening response*

We applied expert consensus guidelines (Stalder et al., 2015) to assess the CAR at 0, 15, 30 min post–awakening on the morning following the test session, which was always a weekday, and on a weekend day within the next month. Mean number of days between the collection days was 11.98 (SD = 11.06, range = 1– 46). Parents were asked to wake their child. Saliva

was collected using a cotton swab held under the tongue for 2 min. It was stressed that sampling had to be postponed if the child had woken up spontaneously or fell ill. Parents were told to withhold food, drinks, and brushing teeth and fill in a protocol recording sampling times and daily events. Sampling times were verified via electronic monitoring (MEMS 6 TrackCap; Aardex Ltd., Switzerland) that recorded container opening when saliva swabs were extracted.

Saliva samples were stored in parents' home freezer and picked up by an experimenter within 3 weeks. They were stored at -80°C, then brought to room temperature, centrifuged at 3000 rpm for 15 min, and assayed using the same highly sensitive enzyme immunoassay (Salimetrics, Suffolk, UK) with a detection range from 0,012 µg/dL – 3 µg/dL. lower sensitivity limit of 0,007 µg/dL, and average intra– and inter–assay coefficients of variation < 7%. The average of duplicate assays was used for all samples.

A lenient sampling exclusion threshold was chosen in consideration of an expert consensus (Stalder et al., 2015), excluding samples if a difference ≥ 15 min was found between pre–specified and MEMS verified sampling times. This excluded 1 sample on each day. Lower sampling timing compliance was not significantly predicted by SES (all *p*'s > 0.21). In order to reduce outlier effects, individual samples ≥ 5 SD above the mean were winsorized with the 99th percentile (Hostinar et al., 2015). This affected 4 samples on day 1 and 5 samples on day 2. All raw cortisol values were log transformed to correct for significant skew and standardized to sample 1 (at awakening).

2.3.3. *Cortisol stress reactivity*

Children were kept separate from their accompanying parent before and following the TSST–C to prevent buffering effects, unless the child explicitly requested to see the parent. Excluding the 12 cases who requested did not affect results. The TSST–C consisted of a story preparation, story telling, and mental calculation part (5 min each) and was performed in front of 2 live female judges and a video camera (for details see Buske-Kirschbaum et al., 1997). Judges completed training of administering the TSST–C in the Kirschbaum lab in Dresden.

During testing, one judge rated children on stress–related (e.g., fiddling, crying) and performance–related behaviors (e.g., story fluidity) from 0 = none to 2 = strong/frequent, which was later discussed with the second judge for consistency. Children reported a significant increase in negative mood immediately post TSST–C by pointing to comic faces indicating 0 = happy, 1 = neutral, or 2 = upset feelings (pre–stress mean = 0.12 vs. post–stress mean = 0.53, t = -5.64, $p < 0.05$, CI = -0.55 – -0.27), which was back to normal 10 min later (post–stress mean = 0.11, t = 0.30, p = 0.76).

Salivary cortisol values were collected at 8 times: 10 min and immediately preceding the stress task and at 0, 10, 20, 30, 40, and 50 min after the stress task. Swabs were frozen at -80°C until they were shipped on dry ice to the laboratory of the Institute of Medical Psychology at Charité – Universitätsmedizin Berlin and stored at -80°C until assayed in the same manner as CAR. All raw cortisol values were log transformed to correct for significant skew and standardized to sample 1 (the first pre-stress sample).

2.3.4. *Memory*

Participants completed an item–association memory task, where they had to remember at what location on a computer screen they had seen a black–colored sketched item (e.g. a shoe, lemon) (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 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. 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 percentage of

correct locations from 15 trials. Memory was standardized for analyses.

2.3.5. *Hippocampus*

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

Volumetric segmentation was performed with the FreeSurfer image analysis suite described elsewhere (Fischl et al., 2002). Segmented images were manually inspected for accuracy, and excluded if motion artifacts led to inaccurate registration (6 cases, final n = 60). Left and right hippocampal volumes and total intracranial volume (ICV) were standardized for analyses. Although the association of Freesurfer segmented and manually traced hippocampal volumes in children is not strong (Schoemaker et al., 2016), follow-up analyses of hippocampal subfields extracted for the hippocampal body corroborated our results. For the latter, we followed the semi-automated procedure using a custom hippocampal subfield atlas (both the procedure and the atlas described in Bender et al., 2018) using ASHS (Automatic Segmentation of Hippocampal Subfields; Yushkevich et al., 2015).

2.4. Data Analysis

First, two latent–change SEMs using MPlus 7.4 were compiled to represent the CAR and CSR intercepts and slopes (see Appendix C for a graphical depiction). The CAR model estimated awakening baseline levels (intercept) and change in response to awakening (slope) as indicated by two observed measurements (one from each day). Since measurement error is explicitly modeled, we were able to account for shared residual variance in samples collected on the same day by constraining them to be equal. The mean CAR intercept is an indicator of the average awakening level (in this case 0 because cortisol values were standardized) and the

cortisol intercept variance represents between–person differences in levels. Similarly, the mean CAR slope is an indicator of the average rate of change in cortisol per 15 min and the CAR slope variance represents between–person differences in this change.

Next, the CSR models estimated pre–stress baseline levels (intercept) and both reactivity and recovery change (slope). Notably, a two–slope model of CSR separating reactivity and recovery provided worse fit to the data, because they were very highly correlated (standardized $\rho = 0.96$, SE = 0.01, $p < 0.05$). The mean CSR slope is an indicator of the average change in cortisol per 10 min and the CSR slope variance represents between–person differences in this change. All following steps were identical for the CAR and CSR.

Second, we tested whether cortisol reactivity was associated with SES and memory. Cortisol intercept and slope were regressed on SES to test for main effects of SES. Memory was regressed on SES, intercept, and slope to test for main effects of reactivity onto memory. Given that it has been shown that statistical mediation analyses on cross–sectional data is biased (Lindenberger et al., 2011), no such tests were performed.

Third, we investigated SES moderation effects by including an SES X Cortisol Slope interaction. Significant interactions were explored by graphing the interaction in line with recommendations for latent interactions in Mplus (Maslowsky et al., 2015) and with simple slope analysis of exported latent slope estimates. However, simple slope analysis treats the latent variable as an observed variable without including standard errors and must therefore be treated with caution. All main effects and the moderation effect were tested within one model. Age, sex, and cortisol covariates (CAR: time of year, day type, hours slept the previous night, awakening time, bedtime, positive or negative events on the preceding day, sampling time compliance; CSR: time since awakening, stress task time of day, hours slept the previous night, contact with parent during session) were included as additional regressors.

Fourth, to test whether hippocampal volume (indicated by left and right volumes) was associated with SES and memory, hippocampus was regressed on SES and memory was regressed on hippocampus. To investigate moderation effects, we added an SES X Hippocampus interaction term. Sex, age, and ICV were included as covariates. Lastly, we

explored correlations between CAR, CSR and hippocampal volume and performed a post–hoc power analysis to inform future research.

The above steps were repeated for each SES predictor (income, education, occupation, SES composite). We prioritize income in our results, because income is the most variable SES indicator that may be more closely associated with different levels of family stress including financial strain (Raver et al., 2015) and has been shown to correlate with children's stress reactivity (Badanes et al., 2011; Kraft and Luecken, 2009).

All models were fitted using full information maximum likelihood (FIML) estimation to accommodate missing at random data. 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 (Little, 2013). We report the final models including the SES X Cortisol interaction, because none of the main effects were significantly affected by excluding the interaction. Final models included bootstrapped parameter estimates (5,000 bootstrap samples) to adjust for multiple comparisons (MacKinnon et al., 2004; Westfall, 2011) and standardized parameter estimates as effect sizes. Parameter significance at $p < 0.05$ was determined by comparing the model with versus without the parameter (as a 1 df χ^2 test).

3. Results

3.1. *Cortisol awakening response*

The CAR was characterized by a significant mean rise in cortisol (slope). Significant intercept and slope variance parameters suggest CARs differed significantly between children. Furthermore, a higher intercept was negatively correlated with a lower slope. Of the covariates added individually to the model, only day type was significant and thus retained in the model. Age and sex were added as covariates, but had no effect. The final model indicated good fit ($\chi^2 = 47$, $df = 52$, $CFI = 1$, $E_a = 0$, CI = 0-0.05).

Lower income was significantly associated with a lower CAR slope (Table 2 for parameter estimates, see Figure 1) and lower memory. Next, a higher CAR intercept was

associated with better memory, but this effect did not survive bootstrapping. There was no main effect of CAR slope on memory. Testing for moderation, there was no Income X CAR slope interaction on memory.

3.2. *Cortisol stress reactivity*

The CSR was characterized by a cortisol rise (reactivity) and decline (recovery) captured by the slope. Significant intercept and slope variance parameters suggested that children's CSRs differed significantly from each other. CSR intercept and slope were not significantly correlated. Of the covariates added individually, only time since awakening and stress task time of day were significant and retained in the model. Age and sex were added as covariates and only the significant effect of sex onto CSR slope was retained in the model. Accordingly, girls had significantly higher CSR slopes than boys. The final model of CSR, income and memory indicated good fit (χ^2 = 125, *df* = 84, *CFI* = 0.967, E_a = 0.067, CI = 0.04 – 0.09).

Lower income was associated with a lower CSR slope, representing both reactivity and recovery (see Table 3 for parameter estimates and Figure 1). Next, there was no main effect of CSR slope on memory performance. Testing for moderation, there was a significant Income X CSR slope interaction predicting memory performance. The plotted interaction revealed that the relation between CSR slope and memory performance became more positive as income decreased (see Figure 2). Simple slope analysis of exported latent CSR slope estimates suggested that for low–income children, lower CSR was related to lower memory performance (simple slope at Z = -1: 0.618, t = 2.01, $p < 0.05$), whereas for children from higher income, lower CSR was related to better memory performance (simple slope at Z =1: -0.617, t =-2.63, $p < 0.05$). However, given the plotted interaction directly derived from the model, we conclude that for children from higher income CSR was unrelated to memory.

Then we assured that these CAR and CSR associations were not confounded by other variables. There was no significant Income X Sex interaction and adding this variable did not affect the significance of the Income X CSR interaction. Furthermore, income was not correlated with children's self–reported mood decreases, stress–related or performance–

related behaviors, sleeping duration or awakening time (all *p*'s > 0.20). Self–reported mood decreases or children's behaviors during the stressor were not associated with CSR (all *p*'s > 0.15). Adding geographical ancestry, bilingualism, birth weight, gestational age at birth, sleep or parental smoking as covariates did not affect CAR or CSR results.

In addition, we explored whether the CAR and CSR were related to each other. Exported latent intercepts ($r = 0.24$, $p < 0.05$) and slopes ($r = 0.28$, $p < 0.05$) were moderately positively correlated.

3.3. *Hippocampus*

Lower income was associated with smaller bilateral hippocampal volume (see Table 4 for parameter estimates). Next, there was a non–significant trend ($p = 0.07$) of hippocampal volume on memory with a negative parameter. Testing for income moderation, there was no interaction of Income X Hippocampus on memory.

To assure that these hippocampal associations were not confounded by other variables, we added geographical ancestry, bilingualism, birth weight, sleep or parental smoking as covariates. None of these significantly affected results. However, the MRI subsample of children had significantly higher memory performance (mean 0.44 versus 0.37, $t = -2.29$, $p < 0.05$). In addition, we explored whether hippocampal volume was correlated with CAR and CSR. Exported latent hippocampal volume was not significantly correlated with CAR ($r = -0.15$, $p = 0.24$) or CSR slopes ($r = -0.01$, $p = 0.95$).

A post hoc power calculation of the effect of income on CAR, CSR, and hippocampus indicated power of 0.68, 0.75 and 0.52, respectively, and 0.88 for Income X CSR on memory.

3.4. Other SES predictors

Lastly, we explored whether our results replicated with other SES indicators (see Appendix B for parameter estimates). All income results replicated with income divided by the number of household members to correct for family size. As summarized in Table 5, the main effects

onto CAR and CSR slopes, indicating hyporeactivity, were specific to income. All other effects, including the significant SES X CSR interaction and smaller hippocampal volume, replicated with education and the SES composite score, whereas occupational status was not related to any variables of interest. The significant SES X CSR interactions replicated the type of cross–over interaction described for income.

4. Discussion

This study explored associations between SES, cortisol reactivity, hippocampal volume, and associative memory in children. First, we found that lower income children have both a blunted CAR and blunted reactivity and recovery to acute stress. Interestingly, the main effect indicating hyporeactivity in CAR and CSR was specific to using income as the predictor and did not replicate with education, occupation or an SES composite. We hypothesize that income may have specific effects because it is the most variable SES indicator and may be more closely associated with different levels of family stress related to financial strain (Raver et al., 2015), even in relatively well–educated parents with socially recognized occupations. Thus, our results corroborate that SES indicators have diverging effects on child outcomes.

Our results based on reliable and valid cortisol measurements significantly contributes to the mixed literature showing higher and lower basal cortisol patterns in association with low SES (Badanes et al., 2011; Blair et al., 2011; Chen et al., 2010; Chen and Paterson, 2006; Kliewer et al., 2008; Lupien et al., 2000) or related stressors (Pagliaccio et al., 2014; Wagner et al., 2016). We believe the current evidence suggests that low–income prepubescent children's diurnal cortisol secretion could be profiled by a hyporeactive CAR, flatter diurnal slopes (Martin et al., 2016; Wolf et al., 2008), potentially higher bedtime cortisol levels (Wolf et al., 2008), and total reductions in diurnal cortisol secretion (Raffington et al., 2018). Correspondingly, in adults chronically low SES from infancy through early adulthood predicts the lowest CARs, flattest diurnal slopes, highest bedtime

cortisol levels, and lowest total cortisol levels (Desantis et al., 2015). This hypothesized diurnal profile awaits further empirical scrutiny in future studies that need to adequately sample from low SES strata, control for age and pubertal status, collect reliable diurnal cortisol from multiple days (minimum 2, but preferably 4 to explore bedtime levels; Rotenberg et al., 2012) using data quality controls (e.g., timing compliance monitoring), and statistically disentangle cortisol level and change parameters.

Furthermore, cortisol reactivity to acute stress was blunted in lower–income children, which provides profound evidence for HPA axis dysregulation and is in line with some previous research (Badanes et al., 2011; Kraft and Luecken, 2009). A recent study in young adults suggests that higher rumination is associated with blunted reactivity to the typical stressful TSST, but higher cortisol reactivity to an intermediately difficult low–stress TSST (Vrshek-Schallhorn et al., 2017). Thus, low–SES children may similarly show blunted reactivity to acute psychosocial stress, as suggested by our TSST–C study, but heightened reactivity to low–stress challenging situations. One theory of cortisol hyporeactivity suggests it reflects lower steady–state HPA axis activity at the level of the pituitary in response to chronic activation from the hypothalamus (Fries et al., 2005). Hyporeactivity may be a distinct allostatic adaptation of the HPA axis to higher chronic stress exposure to protect from excessive expenditure of metabolic resources in the face of recurrent stress and to reduce the risk of neural damage from overexposure to cortisol (Levine, 2006). Indeed, emerging research on long–term cumulative cortisol levels measured in hair (e.g. 2–3 months) suggest higher levels in lower SES children (Rippe et al., 2015; Vaghri et al., 2014), although another study found lower levels in children under 13 years followed by steeper increases with age cross-sectionally that result in higher levels in later adolescence (Tucker-Drob et al., 2017). More frequent cortisol stress reactions and higher nocturnal secretion as a continuation of higher bedtime levels could lead to higher long–term cumulative levels, despite diurnal hypocortisolism and stress hyporeactivity. Additionally, in contrast to enthusiastic interpretations of hair cortisol being a biomarker of cumulative chronic stress exposure, genetic factors account for approximately half of the variation in cortisol (Tucker-Drob et al.,

2017). Therefore, it remains to be established whether altered hair cortisol levels are associated with hyporeactivity in lower income children's CAR and CSR in middle childhood and to what degree these associations are driven by environmental or genetic effects.

Furthermore, income, as well as the other SES predictors, moderated the association of stress hyporeactivity with associative memory. Specifically, stress hyporeactivity was related to lower associative memory among lower–SES children, whereas there was no reliable association of CSR and memory among higher–SES children. We believe this provides evidence that there may be threshold effects in the mechanisms associating SES and cognition in middle childhood, such that cortisol dysregulation may only be associated with memory at the lower end of the SES spectrum. In higher SES families, lower stress exposure may partly dissociate cortisol–cognition associations even though main effects of SES on cognition are still found. This may reflect differently weighted combinations of causes operating at different levels of SES that future research can disentangle by exploring moderation effects of SES and poverty (Farah, 2017).

In contrast, hyporeactivity in low–income children's CAR was not related to memory. Since the CAR and CSR were only moderately correlated and only the CSR showed sex differences, we believe the CAR and CSR are largely distinct indices (Bouma et al., 2009) that are differentially associated with different forms of memory. Thus, cortisol–memory relationships may be specific to CSR, because CSR is a response to experiencing a lack of control and social–evaluative threat in response to a cognitively challenging task, whereas the CAR is not. Correspondingly, the medial PFC seems to be involved only in stress–induced HPA axis activity, but not in the regulation of diurnal HPA axis activity (Herman et al., 2005). Hence, associative memory functioning and CSR may show more overlapping neural correlates, whereas the CAR seems more closely associated with preparing the brain generally for upcoming tasks, including remembering what actions must be performed that day (Bäumler et al., 2014). Alternatively, the CSR may just be a more valid measure of HPA axis functioning (Hellhammer, 2011), whereas reliability of the CAR is modest even when it is measured on several days (Rotenberg et al., 2012). Consequently, our results suggest a

stress–hyporeactive mechanism of lower associative memory performance in low–SES children, which may reflect an impairment in long–term potentiation in the hippocampus and a decrease in synaptic activity in PFC circuitry (Lupien and Lepage, 2001).

Third, lower income was associated with smaller hippocampal volume, which was replicated with other SES predictors and reflects previous studies (Brody et al., 2017; Hair et al., 2015; Hanson et al., 2011; Jednoróg et al., 2012; Luby et al., 2013; Noble et al., 2015, 2012a, 2012b; Rao et al., 2010; Yu et al., 2017). However, since hippocampal volume had a trend–level *negative* association with memory (cf. Van Petten, 2004), but income had a *positive* relationship with hippocampal volume and memory, volume is less likely to mediate income disparities in memory cross–sectionally. Moreover, cortisol reactivity dysregulation was not correlated with hippocampal volume, similar to a previous study reporting a null result of this association (Sheridan et al., 2013). Yet, Freesurfer is limited in segmenting hippocampal volume in child brains (Schoemaker et al., 2016) and, more generally, null results can only be interpreted with caution. In contrast, higher early childhood basal cortisol levels was found to mediate genetic risk and early life stress effects on smaller hippocampal volumes in later childhood (Pagliaccio et al., 2014). This may indicate that HPA axis activity in early childhood is an important predictor of neural development in middle childhood. Importantly, without reliable evidence that these SES–related hippocampal differences link to underlying memory deficits in lower SES children, we are relying on reverse inference using between–person neural differences to infer participants' cognitive abilities. Thus, while some evidence suggests environmental stress and parenting may mediate SES disparities in hippocampal volume (Brody et al., 2017) it is currently not established that smaller hippocampal volume observed in low SES indicates effects of cortisol secretion on a stress–sensitive region that mediates memory disparities in children. Furthermore, structural hippocampal development continues beyond middle childhood, is non–linear in some regions, and is complexly linked to different memory functions (Keresztes et al., 2017). Therefore, the coupling of hippocampal volume and memory, and its susceptibility to cortisol dysregulation, is likely to differ along developmental time.

Emerging evidence suggests that SES may moderate functional brain–behavior relations (Farah, 2017). SES seems to have a selective influence on hippocampal–prefrontal–dependent memory, showing less hippocampal activation in associative memory in children (Sheridan et al., 2013), but spared striatal–dependent procedural memory in adolescents (Leonard et al., 2015). The notion that SES moderates the employment of brain systems within the same cognitive task is strengthened by experimental evidence advocating that stress shifts engagement of a hippocampal–based, flexible system to striatum–based, rigid habit memory system (Schwabe and Wolf, 2013). Further, stress may alter the expression of plasticity neurotransmitters, such as BDNF, which is critically involved in hippocampal plasticity for learning (Gray et al., 2013). Future work is needed to explore functional hippocampal, striatal and PFC activity that may be differentially engaged in low–SES children's associative memory performance.

Several limitations of this study warrant attention. First, our sample was biased in attracting more highly educated parents and passed stringent exclusion criteria that will underestimate the effects of low SES. Second, we believe our 15–point occupational scale lacked specificity, which may explain the lack of associations with all other variables of interest. Future studies should aim at collecting more fine-scaled assessments of occupation that are sensible within the country of data collection. Third, measuring the CAR on two days only has modest reliability (Rotenberg et al., 2012) and our study was somewhat underpowered, which may conceal SES–cortisol–memory associations. We further chose to examine a narrow age range of prepubescent children in order to subvert moderating effects of puberty, yet, this also means that results cannot be extrapolated to other age ranges. Moreover, several family–level environmental factors, such as quality of the home environment, mediate SES disparities in child cognition (Hackman et al., 2015) that were not explored here. This study focused on identifying effects of SES, rather than family–level characteristics, because they directly test a stress system mechanism of low SES. Nevertheless, several potential confounds of low SES were ruled out, such as premature birth, parental smoking, and health disorders. There was also no evidence that lower SES children

performed differently in the TSST–C or reported more negative mood in response to it. Similarly, memory encompasses encoding and retrieval processes in short–term, episodic, semantic, procedural, and prospective memory. It therefore remains to be established whether results generalize to other forms of memory and cognition.

Additionally, the cross–sectional nature does not allow causal inferences to be made between SES, cortisol reactivity, and memory. Prospective longitudinal studies on SES predicting cortisol reactivity and hippocampal volume, and those predicting cognitive performance are needed. Related to this, SES disparities in cognition are known to be under genetic influence and are likely to partly explain our correlational results (Ericsson et al., 2017). Furthermore, animal models suggest that glucocorticoid responses to early stress change the expression of genes involved in neural development leading to poorer memory (Meaney, 2010), suggesting that cortisol secretion may interact with genes in influencing memory. Behavior genetic and genome–wide association research provide elegant methods to look at the interplay of genetic, SES and cortisol dysregulation effects in cognition.

In conclusion, our study showed that lower income, but not other SES predictors, is associated with a blunted CAR and blunted reactivity and recovery to acute stress in pre–pubescent middle childhood. Hyporeactivity to stress was associated with lower memory performance among low–SES children only, which may imply threshold effects for the role of cortisol secretion in SES–memory associations. Since smaller hippocampal volume observed in low–SES children did not associate with poorer memory performance, we believe compromised hippocampal function, not structure, particularly plasticity for learning, underlies associative memory deficiency. Notably, evidence of a stress mechanism presented here does not imply environmental– or genetically–mediated causation, as the study lacks genetic and longitudinal data. Future interventions should test whether improving stress coping in lower SES children attenuates cognitive disparities.

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Conflicts of interest: None.

5. Appendix

Table A.1

Parameter estimates for SES Composite Score.

Model Fit	χ^2=0.11, *df*=3, *CFI*=1.00, E_a=0, CI=0-0
	A. Mean and Variance Parameters (Standard Error)
SES Intercept Mean	Fixed at 0
SES Intercept Variance	Fixed at 1
	B. Loading Parameters (Standard Error)
Income onto SES	0.46* (0.11)
Education onto SES	0.91* (0.15)
Occupation onto SES	0.61* (0.13)

[a] Standardized parameters. Asterisks denote significance at the α level of 0.05.

Table B.1

Parameter estimates for CAR with differing SES Predictors.

	A. Education	$\Delta\chi^2(1)$	$\varphi_c(1)$
Education onto Memory	0.21* (0.10)	4.48*	0.21
Education onto CAR Intercept	-0.14 (0.11)	1.50	0.12
Education onto CAR slope	0.11 (0.13)	0.80	0.09
CAR Intercept onto Memory [b]	0.25 (0.14)	4.47*[b]	0.21
CAR Slope onto Memory	-0.01 (0.09)	0.02	0.01
Education X CAR slope Interaction onto Memory	Fixed at 0[a]	–	
	B. Occupation	$\Delta\chi^2(1)$	
Occupation onto Memory	0.06 (0.11)	0.35	0.06
Occupation onto CAR Intercept	-0.02 (0.13)	0.01	0.01
Occupation onto CAR slope	-0.06 (0.14)	0.17	0.04
CAR Intercept onto Memory [b]	0.23 (0.12)	3.67	0.19
CAR slope onto Memory	-0.01 (0.08)	0.01	0.01
Occupation X CAR slope Interaction onto Memory	Fixed at 0[a]	–	
	C. SES Composite	$\Delta\chi^2(1)$	
SES onto Memory	0.27* (0.01)	5.28*	0.23
SES onto CAR Intercept	-0.14 (0.11)	1.65	0.13
SES onto CAR slope	0.12 (0.13)	0.98	0.10
CAR Intercept onto Memory [b]	0.25 (0.14)	4.59*[b]	0.08
CAR slope onto Memory	-0.01 (0.09)	0.02	0.01
SES X CAR slope Interaction onto Memory	Fixed at 0[a]	–	

[a] Models provided significantly better fit when this non-significant interaction was excluded.

[b] This effect did not survive bootstrapping correcting for multiple comparisons.

[c] Standardized parameters and standard error in parentheses. The $\Delta\chi^2$ values refer to likelihood ratio tests with one df resulting from model comparisons of the full model with a

model leaving out the corresponding effect. Asterisks denote significance at the α level of 0.05. φ_c = Cramer's phi as effect size estimate.

Table B.2

Parameter estimates for CSR with differing SES Predictors.

	A. Education	$\Delta\chi^2(1)$	$\varphi_c(1)$
Education onto Memory	0.43* (0.15)	7.94*	0.28
Education onto CSR Intercept	-0.15 (0.11)	1.89	0.14
Education onto CSR slope	-0.06 (0.10)	0.37	0.06
CSR Intercept onto Memory	0.07 (0.11)	0.43	0.06
CSR slope onto Memory	-0.01 (0.07)	0.02	0.01
Education X CSR slope Interaction onto Memory	-0.23* (0.11)	4.80*	0.22
	B. Occupation	$\Delta\chi^2(1)$	
Occupation onto Memory	-0.03 (0.18)	0.02	0.01
Occupation onto CSR Intercept	-0.04 (0.05)	0.53	0.07
Occupation onto CSR slope	-0.01 (0.06)	0.02	0.01
CSR Intercept onto Memory	0.22 (0.26)	0.76	0.09
CSR slope onto Memory	-0.02 (0.14)	0.02	0.01
Occupation X CSR slope Interaction onto Memory	0.06 (0.22)	0.06	0.02
	C. SES Composite	$\Delta\chi^2(1)$	
SES onto Memory	0.42* (0.14)	8.32*	0.29
SES onto CSR Intercept	-0.15 (0.10)	2.07	0.14
SES onto CSR slope	-0.03 (0.10)	0.11	0.03
CSR Intercept onto Memory	0.08 (0.10)	0.60	0.08
CSR slope onto Memory	0.01 (0.07)	0.01	0.01
SES X CSR slope Interaction onto Memory	-0.21* (0.10)	4.48*	0.21

[a] Standardized parameters and standard error in parentheses. The $\Delta\chi^2$ values refer to likelihood ratio tests with one df resulting from model comparisons of the full model with a model leaving out the corresponding effect. Asterisks denote significance at the α level of 0.05. φ_c = Cramer's phi as effect size estimate.

Table B.3

Parameter estimates for Hippocampus with differing SES Predictors.

	A. Education	$\Delta\chi^2(1)$	$\varphi_c(1)$
Education onto Hippocampus[b]	0.25* (0.11)	4.77*	0.22
Education onto Memory	0.27* (0.11)	5.71*	0.24
Hippocampus onto Memory	-0.26 (0.14)	3.47	0.18
Education *Hippocampus Interaction onto Memory	0.08 (0.13)	0.39	0.06
	B. Occupation	$\Delta\chi^2(1)$	
Occupation onto Hippocampus[b]	0.13 (0.12)	1.18	0.11
Occupation onto Memory	-0.03 (0.18)	0.62	0.08
Hippocampus onto Memory	-0.17 (0.15)	1.25	0.11
Occupation X Hippocampus Interaction onto Memory	0.13 (0.12)	1.23	0.11
	C. SES Composite	$\Delta\chi^2(1)$	
SES onto Hippocampus[b]	0.25* (0.11)	5.08*	0.22
SES onto Memory	0.27* (0.01)	6.34*	0.25
Hippocampus onto Memory	-0.23 (0.13)	2.99	0.17
SES X Hippocampus Interaction onto Memory	0.12 (0.12)	0.98	0.10

[a] Standardized parameters and standard error in parentheses. The $\Delta\chi^2$ values refer to likelihood ratio tests with one df resulting from model comparisons of the full model with a model leaving out the corresponding effect. Asterisks denote significance at the α level of 0.05. φ_c = Cramer's phi as effect size estimate.

[b] Hippocampus is a latent variable indicated by loadings fixed to 1 from left and right hippocampus.

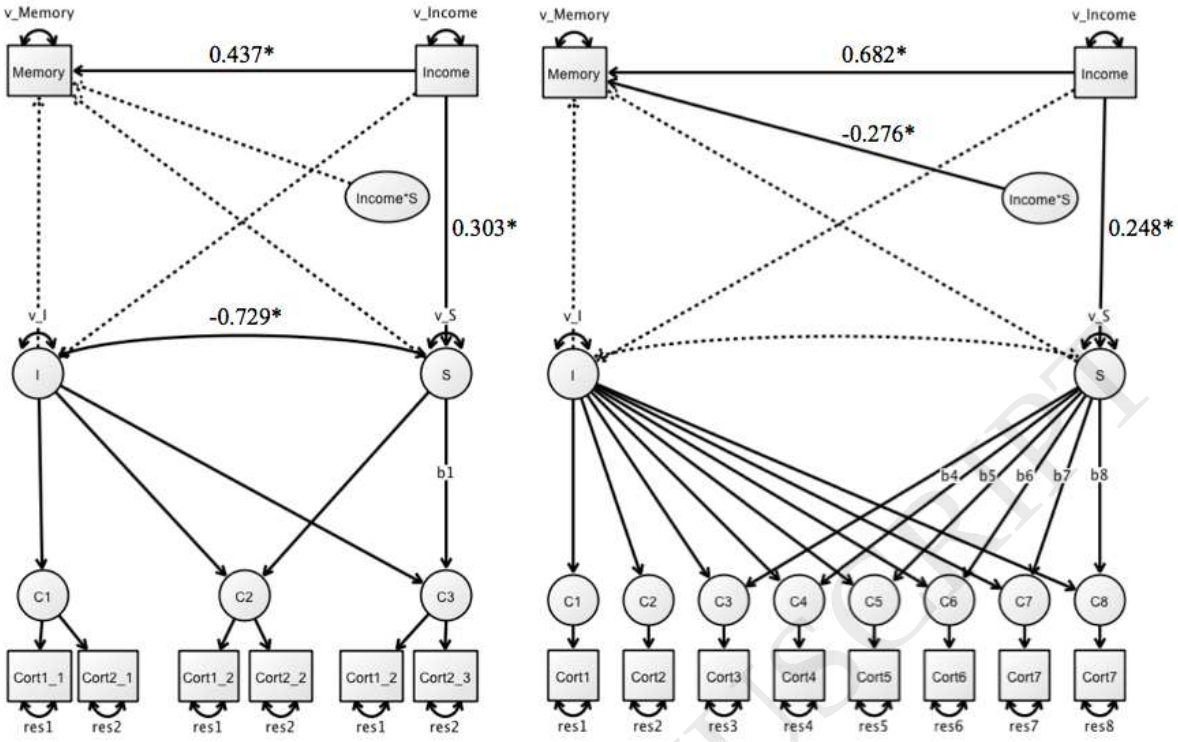

Figure C. Graphical illustration of CAR (left panel) and CSR (right panel) with income and the Income X Slope interactions predicting memory. Observed variables are depicted as squares, latent variables as circles, regressions as one–headed arrows, and variances as two–headed arrows. Significant paths are marked as solid lines and labeled with standardized parameter estimates. Paths without values were fixed at 1. Actual models included slope means, age, sex, and covariates, which were not shown for simplicity of model interpretation. Figure compiled using Onyx 1.0 (http://onyx.brandmaier.de).

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Table 1 *Descriptive Statistics.*

	Mean (SD)		
	Whole sample	-1 SD Income[a]	+1 SD Income[a]
Income (Monthly)	3707 (2060)	1049 (304)	7470 (1119)
Maternal Education [b]	8.88 (3.45)	5.71 (3.04)	10.47 (3.36)
Maternal Occupational Status [c]	7.75 (3.23)	6.62 (3.62)	8.6 (3.29)
SES Composite	0 (1)	-0.91 (0.81)	0.56 (0.87)
CAR 1_1[d]	0.39 (0.25)	0.37 (0.30)	0.47 (0.25)
CAR 1_2	0.52 (0.23)	0.50 (0.20)	0.62 (0.20)
CAR 1_3	0.54 (0.22)	0.50 (0.23)	0.65 (0.24)
CAR 2_1	0.34 (0.17)	0.37 (0.16)	0.28 (0.11)
CAR 2_2	0.43 (0.21)	0.41 (0.09)	0.43 (0.18)
CAR 2_3	0.48 (0.23)	0.47 (0.19)	0.49 (0.25)
CSR 1[e]	0.09 (0.04)	0.10 (0.04)	0.08 (0.05)
CSR 2	0.09 (0.05)	0.10 (0.05)	0.09 (0.08)
CSR 3	0.17 (0.14)	0.22 (0.18)	0.22 (0.14)
CSR 4	0.29 (0.25)	0.32 (0.33)	0.39 (0.28)
CSR 5	0.29 (0.28)	0.29 (0.32)	0.41 (0.41)
CSR 6	0.22 (0.22)	0.20 (0.18)	0.40 (0.39)
CSR 7	0.17 (0.17)	0.16 (0.13)	0.29 (0.32)
CSR 8	0.14 (0.13)	0.14 (0.09)	0.21 (0.20)
Memory Task Score	0.41 (0.15)	0.32 (0.18)	0.45 (0.15)
Hippocampal Volume	8113 (797)	7685 (992)	8523 (567)

[a]Low income = 1 SD below mean, n = 17. High income = 1 SD above mean, n = 15. Post–tax monthly household income in Euro.

[b] Maternal Education of 6 = advanced vocational training, 9 = technical college, 11 = Bachelor degree or equivalent

[c] Maternal Occupational Status of 7 = qualified employment beyond German vocational training as a "Meister", 8 = freelance employee without leadership, 9 = freelance employee with leadership

[d]CAR = Cortisol awakening response taken 3 times on 2 days, e.g. CAR 1_1 = day 1 sample 1. Raw cortisol values in µg/dL.

[e]CSR = Cortisol stress reactivity measured twice before acute stress (CSR 1–2) and 6 times post–stress (CSR 3–8).

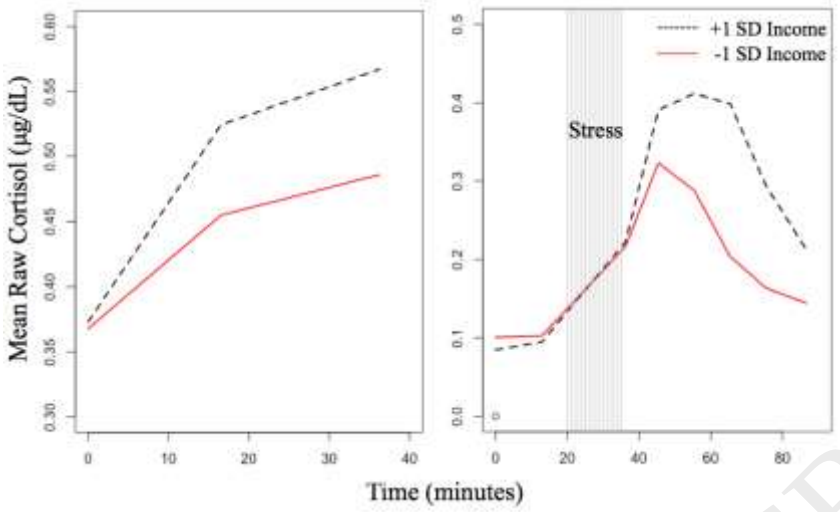

Figure 1. Cortisol concentrations of CAR (left panel) and CSR (right panel) plotted for 1 SD above mean income (dashed black line) and 1 SD below mean income (solid red line). The plot depicts the significant positive main effect of income on CAR and CSR slopes, suggesting that lower income is associated with a hyporeactive CAR and CSR.

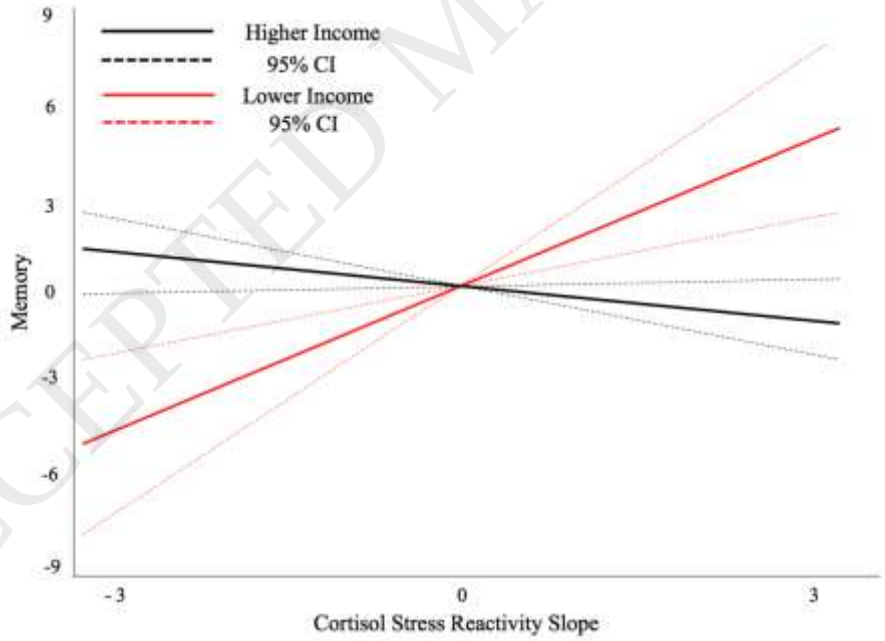

Figure 2. Graphical depiction of Income X CSR slope interaction predicting memory directly derived from the Mplus model. The red line shows the association of slope and memory for lower income with 95% confidence intervals (thin red dashed lines). The black line shows the

association of slope and memory for higher income with 95% confidence intervals (thin black dashed lines). The plot shows that the relation between CSR slope and memory becomes more positive as income decreases. This suggests low–income CSR hyporeactivity links to lower memory performance.

Table 2

Parameter estimates from best fitting CAR model.

Model Fit	χ^2=46, *df*=53, *CFI*=1, E_a=0, CI=0-0.05		
	A. Mean and Variance Parameters (Standard Error)	$\Delta\chi^2(1)$	$\varphi_c(1)$
CAR Intercept Mean	Fixed at 0	–	
CAR Intercept Variance	0.97* (0.04)	–	
CAR Slope Mean	0.75* (0.07)	–	
CAR Slope Variance	0.90* (0.08)	–	
Covariance Intercept–Slope	-0.73* (0.09)	–	
Income Mean	Fixed at 0	–	
Income Variance	0.99* (0.14)	–	
Memory Mean	Fixed at 0	–	
Memory Variance	0.87* (0.13)	–	
	B. Slope Loading Parameters (Standard Error)		
CAR 1[a] onto CAR Slope	Fixed at 0	–	
CAR 2 onto CAR Slope	Fixed at 1	–	
CAR 3 onto CAR Slope	1.30* (0.23)	–	
	C. Regression Parameters (Standard Error)		
Income onto Memory	**0.44* (0.18)**	**6.04***	**0.24**
Income onto CAR Intercept	**-0.17 (0.11)**	**2.28**	**0.15**
Income onto CAR slope	**0.30* (0.12)**	**6.12***	**0.24**
CAR Intercept onto Memory [b]	**0.27 (0.12)**	**5.20***	**0.23**
CAR slope onto Memory	**0.01 (0.08)**	**0.00**	**0**
Income X **CAR Slope Interaction onto Memory**	**-0.09 (0.09)**	**1.00**	**0.10**
	D. Covariate Parameters (Standard Error)		
Sex[d] – CAR Slope	-0.01 (0.10)	1.02	0.10
Day type – Cortisol 4-6	-0.09* (0.03)	12.84*	0.35

[a] CAR 1, 2, 3 are latent variables indicated by two samples each, one from each day.

[b] This effect did not survive bootstrapping correcting for multiple comparisons.

[c] Standardized parameters. The $\Delta\chi^2$ values refer to likelihood ratio tests with one df resulting from model comparisons of the full model with a model leaving out the corresponding effect. Asterisks denote significance at the α level of 0.05. φ_c = Cramer's phi as effect size estimate. Main paths of interest are marked in bold.

[d] Sex coded as -1 = male and 1 = female.

[e] Covariate means and variances and residual cortisol variances are not shown to aid readability.

Table 3

Parameter estimates from best fitting CSR model.

Model Fit	χ^2=124, *df*=85, *CFI*=0.967, E_a=0.067, CI=0.04-0.09		
	A. Mean and Variance Parameters (Standard Error)	$\Delta\chi^2(1)$	$\varphi_c(1)$
CSR Intercept Mean	Fixed at 0	–	
CSR Intercept Variance	0.91* (0.06)	–	
CSR Slope Mean	0.58* (0.06)	–	
CSR Slope Variance	0.77* (0.08)	–	
Covariance Intercept-Slope	-0.16 (0.11)	–	
Income Mean	Fixed at 0	–	
Income Variance	0.99* (0.14)	–	
Memory Mean	Fixed at 0	–	
Memory Variance	0.85* (0.12)	–	
	B. Slope Loading Parameters (Standard Error)		
Cortisol 1 onto CSR Slope	Fixed at 0	–	
Cortisol 2 onto CSR Slope	Fixed at 0	–	
Cortisol 3 onto CSR Slope	Fixed at 1	–	
Cortisol 4 onto CSR Slope	0.99* (0.05)	–	
Cortisol 5 onto CSR Slope	0.99* (0.05)	–	
Cortisol 6 onto CSR Slope	0.96* (0.06)	–	
Cortisol 7 onto CSR Slope	0.90* (0.06)	–	
Cortisol 8 onto CSR Slope	0.82* (0.07)	–	
	C. Regression Parameters (Standard Error)		
Income onto Memory	**0.68* (0.16)**	**16.29***	**0.40**
Income onto CSR Intercept	**-0.20 (0.10)**	**3.55**	**0.19**
Income onto CSR Slope	**0.25* (0.09)**	**7.01***	**0.26**
CSR Intercept onto Memory	**0.07 (0.10)**	**0.48**	**0.07**
CSR Slope onto Memory	**0.03 (0.07)**	**0.17**	**0.04**
Income X CSR Slope Interaction onto Memory	**-0.28* (0.09)**	**9.62***	**0.31**
	D. Covariate Parameters (Standard Error)		
Sex[b] – CSR Slope	0.27* (0.09)	7.98*	0.28
Awakening Time – CSR Intercept	0.22* (0.10)	4.60*	0.21
Awakening Time – CSR Slope	-0.23* (0.09)	5.81*	0.24
Stress Time – CSR Slope	-0.22* (0.09)	4.99*	0.22

[a] Standardized parameters. The $\Delta\chi^2$ values refer to likelihood ratio tests with one df resulting from model comparisons of the full model with a model leaving out the corresponding effect. Asterisks denote significance at the α level of 0.05. φ_c = Cramer's phi as effect size estimate. Main paths of interest are marked in bold.

[b] Sex coded as -1 = male and 1 = female.

[c] Covariate means and variances and residual cortisol variances are not shown to aid readability.

Table 4

Parameter estimates from hippocampus model.

Model Fit	χ^2=14.457, *df*=19, *CFI*=1, E_a=0, CI=0.00-0.068		
	A. Mean and Variance Parameters (Standard Error)	$\Delta\chi^2(1)$	$\varphi_c(1)$
Income Mean	Fixed at 0	–	
Income Variance	0.99* (0.15)	–	
Memory Mean	Fixed at 0	–	
Memory Variance	0.88* (0.13)	–	
Hippocampus[b] Mean	Fixed at 0	–	
Hippocampus Variance	0.48* (0.09)	–	
Hippocampus Residual Variance	0.23* (0.09)	–	
	B. Regression Parameters (Standard Error)		
Income onto Hippocampus	**0.22* (0.10)**	**4.17***	**0.26**
Income onto Memory	**0.30* (0.13)**	**8.20***	**0.37**
Hippocampus onto Memory	**-0.24 (0.13)**	**3.15**	**0.23**
Income X **Hippocampus Interaction onto Memory**	**0.25 (0.17)**	**0.52**	**0.09**
	C. Covariate Parameters (Standard Error)		
ICV – Hippocampus	0.66* (0.13)	19.67*	0.57
Sex[c] – Hippocampus	-0.03 (0.13)	0.06	0.03
Age – Hippocampus	0.01 (0.14)	0.01	0.01

[a] Standardized parameters. The $\Delta\chi^2$ values refer to likelihood ratio tests with one df resulting from model comparisons of the full model with a model leaving out the corresponding effect. Asterisks denote significance at the α level of 0.05. φ_c = Cramer's phi as effect size estimate. Main paths of interest are marked in bold.

[b] Hippocampus is a latent variable indicated by loadings fixed to 1 from left and right hippocampus.

[c] Sex coded as -1 = male and 1 = female.

[d] Covariate variances and covariances are not shown to aid readability.

Table 5. Summary table of the replicability of effects described for income in text using other SES predictors.

SES Predictor	Main effect on Memory	Main effect on CAR	Main effect on CSR	Main effect on HC	Interaction SES X CAR on Memory	Interaction SES X CSR on Memory
Income	✓	✓	✓	✓		✓
Education	✓			✓		✓
Occupation						
SES composite	✓			✓		✓

[a] HC = hippocampus