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Examining inflammation, health, stress and lifestyle variables linking low socioeconomic status with poorer cognitive functioning during adolescence

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ABSTRACT

Higher C-reactive protein (CRP) is associated with cognitive difficulties. The nature of this association remains unclear given that multiple other variables are linked with both CRP and cognitive difficulties, which may confound the association. The goal of the current study is to determine whether low socioeconomic status (SES) is associated with worse cognitive functioning via higher CRP and whether this association is independent of known associations with other health, stress and lifestyle factors (e.g., depression, physical activity, body mass). Assessments in a longitudinal study of 1,029 Dutch adolescents were based on a combination of self-report and parent-report questionnaires, diagnostic assessment, behavioral testing, and blood assay. We estimated latent variables for cognitive functioning (executive functioning, verbal fluency, episodic memory) and used structural equation analysis to test whether SES (wave 1: 11.08 years (*SD*=0.55); 55% female] was associated with worse cognitive outcomes (wave 4: aged 18.97 years; *SD*=0.55) via increased CRP, depression, stress, body mass, substance use or physical inactivity (wave 3: aged 16.17 years; *SD*=0.61). Low SES was associated with worse cognitive functioning via increased CRP. Additionally, low SES was associated with (i) worse executive functioning via higher body mass, higher levels of sedentary behavior, and higher stress, (ii) worse verbal fluency via higher levels of sedentary behavior and (iii) worse episodic memory via sedentary behaviors, body mass, and substance use. These results confirm the link between SES, CRP and cognitive functioning and additionally identify four modifiable lifestyle factors that may be implicated in the link between low SES and worse performance on tests of cognitive functioning.

1. Introduction

A recent publication in *Brain, Behavior, and Immunity* showed that higher C reactive protein (CRP) was associated with worse executive functioning in a population-representative sample of Dutch adolescents (Mac Giollabhui et al., 2021). This finding aligns with accumulating evidence that dysregulated inflammatory physiology is associated with worse cognitive functioning in both clinical (Goldsmith et al., 2016) and population-representative samples (Mac Giollabhui et al., 2020; Mac Giollabhui et al., 2021) that are largely free of psychiatric symptoms. It is particularly concerning that an association between CRP and worse cognitive performance can be observed so early in life, given that adolescence is a period where optimal cognitive functioning is closely linked with academic attainment. Dysregulated inflammatory physiology is strongly associated with low socioeconomic status (SES) as well as multiple health, stress and lifestyle variables which are also associated with cognitive functioning, raising the question of how to interpret the association between inflammatory biomarkers and cognitive functioning and the source of dysregulated immune function in youth. The goal of this short communication is to extend these prior analyses to determine whether low SES explains the previously observed association between higher CRP and worse cognitive functioning and, in addition, whether this association is independent of potential confounds (e.g., physical activity, adiposity, substance use). Although relevant variables (e.g., CRP, adiposity, physical inactivity, depression) that link low SES and cognitive variables likely overlap, at least partly, they are to some extent modifiable and existing interventions that target these variables differ. Therefore, a clearer picture of which associations

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remain when adjusted for one another may aid in establishing the knowledge base needed for identifying targets for prevention and treatment.

SES is a well-established predictor of individual performance on tests of cognitive functioning. This is already apparent early in life. For example, intelligence, as assessed in a representative sample of the US population on the Wechsler Intelligence Scale for Children - Fifth Edition, is on average 20 points (1.3 *SD*) lower in children from low SES backgrounds when compared with high SES peers (Wechsler, 2014). Disparities in performance are particularly evident on tests of executive functioning, memory, and language (Hackman et al., 2015; Duncan and Magnuson, 2012). Given that neuropsychological differences in cognitive ability and academic attainment translate into the persistence of intergenerational poverty through lowered earnings (Restuccia and Urrutia, 2004), understanding the mechanisms linking low SES and cognitive functioning is critical.

Low SES is consistently associated with elevated inflammatory biomarkers (e.g., CRP/Interleukin-6) across the lifespan (Muscatell et al., 2018) and, as already noted, inflammation is associated with impaired cognition (Mac Giollabhui et al., 2021; Goldsmith et al., 2016; Mac Giollabhui et al., 2021), both concurrently and prospectively in patient as well as healthy adolescent and adult samples (Mac Giollabhui et al., 2021; Mac Giollabhui et al., 2020; Crişan et al., 2014; Paine et al., 2015). Inflammation likely affects cognitive function by disrupting neuronal processes (e.g., long-term potentiation, synaptic plasticity, neurotransmission and neurogenesis) affecting brain regions and their respective cognitive correlates (e.g., hippocampus: episodic memory; anterior cingulate cortex: executive function) (McAfoose and Baune, 2009). Thus, it is plausible that low SES contributes to worse cognitive functioning via elevated inflammatory biomarkers.

Not only is lower SES associated with cognitive dysfunction and higher inflammatory biomarkers, but it additionally is associated with multiple other risk factors, including depression (Freeman et al., 2016), adiposity (Mac Giollabhui et al., 2020), substance use (Hiscock et al., 2012), sedentary behaviors (Fairclough et al., 2009) and stress (Lupien et al., 2000). These risk factors are similarly associated with both worse cognitive functioning and higher CRP (Mac Giollabhui, 2021). The current study will investigate whether low SES is associated with worse cognitive functioning via higher CRP and (additionally) via five alternative pathways (depression, substance use, sedentary behavior, adiposity, and stress) in a large, longitudinal, population-representative sample of Dutch adolescents where SES was measured at age 11.08 *(SD* = 0.56), health/stress/lifestyle variables were measured at age 16.19 *(SD* = 0.65), and cognitive functioning at age 18.99 *(SD* = 0.56).

2. Methods

2.1. Participants

Participants were recruited as part of a prospective cohort study (TRAILS Study) examining psychosocial development and mental health during childhood, adolescence, and early adulthood. TRAILS recruited children aged 11 years and invited them for follow-up assessments every 2–3 years. From 2,230 recruited at baseline, an analytic sample of 1,076 who had complete data for measures of central interest (i.e., complete data for CRP and neuropsychological measures) was selected. Selection criteria from prior referenced manuscript (Mac Giollabhui et al., 2021) was re-used to increase comparability of results, namely: those who met criteria for a major depression or dysthymia diagnosis at Wave 4 were excluded ($n = 29$) and participants who appeared to be experiencing an acute inflammatory response ($n = 18$) were excluded (i.e., CRP > 10 mg/L *and* reporting either experiencing illness, injury, or a doctor visit/hospitalization during the prior week) in line with published guidelines (Mac Giollabhui et al., 2020). Complete missing data analyses are reported as supplementary material and indicate that boys and individuals from lower SES were more likely to be lost to follow-up.

2.2. Measures

2.2.1. Cognitive functioning

Reliable and valid measures of cognitive functioning were administered at Wave 4 and have been reported previously (Mac Giollabhui et al., 2021). Neuropsychological tests estimated verbal working memory (Digit Span Backwards), visual working memory (Self-Ordered Pointing Task), visual organization (Rey-Osterrieth Complex Figure Test), visuo-constructional ability (Block Design), immediate and delayed verbal memory (Rey Auditory Verbal Learning Test), and phonological and semantic verbal fluency (modified version of the short test of semantic and phonological fluency).

2.2.2. Depression

Depressive disorders were measured using a valid, reliable, and widely used structured diagnostic interview: the Composite International Diagnostic Interview, version 3.0 (CIDI) (Kessler and Üstün, 2004). The CIDI was administered at Wave 4 and was used to identify individuals meeting criteria for a major or dysthymic depressive diagnosis based on the Diagnostic and Statistical Manual of Mental Disorders (fourth edition) during the 30 days prior to interview. Depressive symptoms were measured at Wave 3 using the Withdrawn/Depressed scale of the Youth Self-Report (YSR) (Achenbach, 1991); internal consistency was estimated ($\alpha = 0.74$).

2.2.3. C-reactive protein

CRP is a liver-based protein that is commonly used to index systemic inflammation. At Wave 3, 39.5 ml of blood was drawn from fasting participants (90.1% fasting) and breakfast was provided following the blood draw. CRP was assayed using an immunonephelometric method, BN2 of Siemens Medical Solutions USA (Malvern, PA, USA) with a lower detection limit of 0.175 mgl^{-1}. Intra-assay coefficients of variance ranged from 2.1 to 4.4 mgl^{-1} and inter-assay coefficients of variance ranged from 1.1 to 4.0 mgl^{-1}.

2.2.4. Sedentary behavior

Adolescent sedentary behavior was estimated at Wave 3 by calculating the mean number of hours: sitting at a computer (Monday - Friday); sitting at a computer (Saturday and Sunday); watching television or video (Monday - Friday); and watching television or video (Saturday and Sunday).

2.2.5. Substance use

Substance use was estimated at Wave 3 as: the mean of the number of cigarettes smoked in the last week (0='I don't smoke'; 1='I haven't smoked in the last week'; 2='< 1 cigarette a day; 3='1–5 cigarettes a day'; 4='6–10 cigarettes a day; 5='11–20 cigarettes a day'; 6='>20 cigarettes a day') and the mean number of days the participant drank alcohol during the last week.

2.2.6. Body mass index

Height and weight were measured at Wave 3 and used to calculate body mass index (kg/m^2), which was used as an index of adiposity.

2.2.7. Stress

Twelve common and chronic stressors experienced by the child participant over the past two years was assessed at Wave 3 using a parent-report questionnaire; stressors included: chronic illnesses or handicaps, high work pressure at school, housing problems, neighborhood problems, financial problems, lack of friends, bullying, and conflicts or other relationship problems.

2.2.8. Socioeconomic status

SES was estimated based on the z-standardized sum of five commonly used indicators of SES measured at T1: family income, maternal educational level, paternal educational level, maternal

occupational level and paternal occupational level using the International Standard Classification of Occupations. Analyses used T1 SES only because of the very strong association of SES at Wave 1 and Wave 4 (r = 0.86, p <.001).

2.2.9. Demographic variables

Participant's gender was reported at Wave 1 (male was coded as '1′) and age was reported at all assessments.

2.3. Data analysis

Previous published work has identified a correlated factor model with three latent factors [executive functioning (verbal/visual working memory and visual organization/visuo-constructional abilities), verbal fluency (phonological/semantic fluency), and episodic memory (immediate/delayed recall)] as the best fit for the eight cognitive tasks (Mac Giollabhui et al., 2021). Structural equation modelling was used to test whether SES was associated with these three latent factors of cognitive functioning via CRP, substance use, sedentary behavior, adiposity, depression, and/or stress. We tested if these indirect paths from SES to cognitive functioning substantially differed from zero in 5,000 bootstrapped samples using bias corrected bootstrap confidence intervals (as per convention, a substantial indirect effect was defined as not including the value zero within the 95% confidence interval of the 5,000 bootstrapped samples).

Analyses were conducted in Mplus (Version 7.4) and standard model fit indices from structural equation modelling (SEM) were reported, including the Comparative Fit Index (CFI) and the Root Mean Square Error of Approximation (RMSEA). The Chi-Square test of model fit was reported according to convention, but was not interpreted because it has limited utility in large samples (i.e., N > 200) (Chen, 2007). Good fit for the CFI consisted of values > 0.90 and excellent fit consisted of values > 0.95. A RMSEA statistic between 0.05 and 0.10 was indicative of good fit and a value < 0.05 was indicative of excellent fit (Schermelleh-Engel et al., 2003). A conceptual model of the analysis is provided in Fig. 1 (note that not all statistical associations are presented in Fig. 1 – see Table 1 for complete list of associations).

3. Results

Bivariate correlations and descriptive statistics for variables of central interest are presented in Supplementary Table 1 based on the analytic sample of 1,029 youth. Fit statistics indicate excellent model fit:χ^2(52 = 121.67, CFI = 0.97, RMSEA = 0.036 (95%CI = 0.028, 0.045) and the standardized coefficients are provided in Table 1. Notably, SES was significantly associated with every variable in the model. Among our five predictor variables, each additional hour spent routinely engaged in sedentary behavior was associated with worse performance on our latent factors of executive functioning (β = −0.19, p <.001), verbal fluency (β = −0.14, p <.001), and episodic memory (β = −0.19, p <.001). Indirect pathways tested in 5,000 bootstrapped samples indicate that lower SES was associated with (i) worse executive functioning via CRP (β = 0.10, 95%CI = 0.007, 0.252), BMI (β = 0.13, 95%CI = 0.002, 0.309), sedentary behaviors (β = 0.35, 95%CI = 0.184, 0.595), and stressful life events (β = 0.11; 95%CI = 0.010, 0.288) (ii) worse verbal fluency via sedentary behaviors (β = 0.021; 95%CI = 0.009, 0.039), and (iii) worse episodic memory via sedentary behaviors (β = 0.149; 95%CI = 0.030, 0.303), BMI (β = 0.13; 95%CI = 0.015, 0.298), and substance use (β = 0.053; 95%CI = 0.004, 0.159).

4. Discussion

The purpose of this study was to determine whether low SES explains the previously observed association between higher CRP and worse cognitive functioning and, in addition, whether this association is independent of alternative associations with other health, stress and lifestyle factors known to be associated with both SES and cognitive functioning (e.g., physical activity, adiposity, substance use). Lower SES was indeed indirectly associated with worse executive functioning via CRP and, moreover, these associations were statistically independent of multiple overlapping processes. More broadly, lower SES was directly associated with worse executive functioning, verbal fluency, and episodic memory and a notably consistent relationship linking lower SES with executive functioning, verbal fluency, and episodic memory via higher levels of sedentary behaviors was observed.

There is a burgeoning literature examining the link between SES and cognitive health [e.g., hippocampus, amygdala, and prefrontal cortex volumes (Barch et al., 2021); however, a recent review noted that relatively little is known about the nature of the association linking SES with physical, behavioral, and mental health outcomes and, consequently, a key contribution of the current study is that it considers the association of SES and cognition in the context of multiple, potentially modifiable health, stress and lifestyle factors (Dufford et al., 2020). Prior work has identified CRP, stress, and cigarette smoking as factors potentially mediating a link between low SES and the structural integrity of white matter tracts (Gianaros et al., 2013) as well as executive functioning (Mac Giollabhui et al., 2021). The findings from this study support a role for CRP, stress, and sedentary behaviors linking SES and executive function. Although these variables are being measured separately, it is important to note that, not only do risk factors, such as inflammatory physiology, stress, and sedentary behavior overlap considerably with one another, but they also likely exert dynamic,

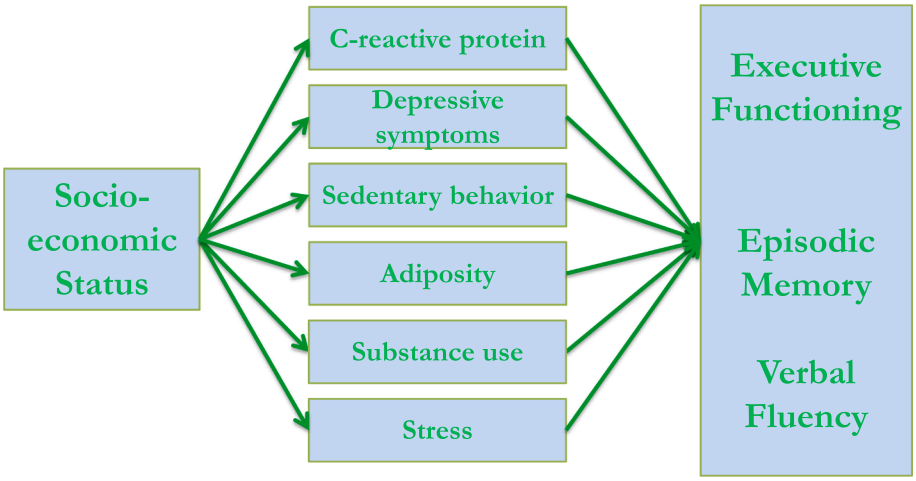

Fig. 1. Conceptual Model of Associations of Low Socioeconomic Status and Cognitive Functioning via Health, Stress and Lifestyle factors.

Table 1
Standardized Coefficients for Structural Equation Model Predicting Associations of Socioeconomic Status and Latent Cognitive Factors of Executive Functioning, Verbal Fluency, and Episodic Memory (n = 1,029) via health, stress and lifestyle factors.[a]

Associations	Coefficients
Predicting executive functioning	
- Depressive Symptoms	0.02
- C reactive protein	−0.08
- Sedentary behavior	**−0.19*****
- Body mass index	−0.08
- Substance use	−0.06
- Stress	**−0.09***
- Socioeconomic Status	**0.35*****
Predicting verbal fluency	
- Depressive Symptoms	−0.02
- C reactive protein	−0.03
- Sedentary behavior	**−0.14*****
- Body mass index	0.03
- Substance use	−0.05
- Stress	0.05
- Socioeconomic Status	**0.34*****
Predicting episodic memory	
- Depressive Symptoms	0.05
- C reactive protein	0.02
- Sedentary behavior	**−0.08***
- Body mass index	**−0.08***
- Substance use	**−0.07***
- Stress	0.02
- Socioeconomic Status	**0.14*****
Predicting Wave 3 Depressive Symptoms	
- Socioeconomic Status	**−0.10****
Predicting C reactive protein	
- Socioeconomic Status	**−0.12*****
Predicting sedentary behaviors	
- Socioeconomic Status	**−0.18*****
Predicting body mass index	
- Socioeconomic Status	**−0.16*****
Predicting substance use	
- Socioeconomic Status	**−0.08***
Predicting Stress	
- Socioeconomic Status	**−0.11*****
Association with C reactive protein and	
- Depressive Symptoms	0.00
- Sedentary behaviors	−0.04
- Body mass index	**0.30*****
- Substance use	**0.11****
- Stress	0.00
Association with Depressive Symptoms and	
- Sedentary behaviors	**0.09****
- Body mass index	0.06
- Substance use	−0.03
- Stress	**0.22*****
Association with Sedentary behaviors and	
- Body mass index	**0.09***
- Substance use	0.07
- Stress	0.01
Association with Body mass index and	
- Substance use	**0.08****
- Stress	**0.08***
Association with Substance use and	
- Stress	0.00

* = p <.05; ** = p <.01; *** = p <.001
[a] = parameter estimates for indirect assocaitions are presented in results section.

bidirectional associations (Mac Giollabhui, 2021). For instance, chronic stress exposure may lead to up-regulation of inflammatory physiology which, in turn, may lead to alterations in neural reward and threat circuitry, in turn leading to greater risk for substance use, poor diet, depression and other behaviors and disorders associated with poorer cognitive functioning (Nusslock and Miller, 2016). Alternatively, poor diet may lead to increased adiposity, which in turn leads to increases in peripheral inflammatory biomarkers and depression as well as worse cognitive functioning (Mac Giollabhui et al., 2020). Thus, interpretation should be considered within the limitations of the research design.

A strength of the study was a longitudinal design following participants over 8 years; however, results are limited by the lack of repeated measures, which precludes characterizing the directionality of the observed relationships. Similarly, although a structural equation modeling approach reduced the number of statistical tests performed – a frequent limitation of many studies examining cognitive dysfunction (Hasselbalch et al., 2011) – each of the latent factors would ideally be based on more than two indicators of a given construct in order to better evaluate model fit and to produce more reliable parameter estimates. In addition, important dimensions of executive functioning were missing (e.g., inhibition) (Miyake and Friedman, 2012). Missing data analyses suggest that individuals of low SES were particularly likely to attrite from the sample, which may limit the generalizability of these results. Finally, it is important to note that only some of the many potential factors connecting SES and cognitive functioning were modelled and the absence of familial and neighborhood factors is limiting. Moreover, as noted previously, it is likely that these factors, although measured and modelled as separate factors, overlap both at a biological (e.g., CRP and body mass) and behavior (e.g., depressive symptoms and sedentary behaviors) level in addition to exerting dynamic, bidirectional effects over time.

5. Conclusion

Cognitive dysfunction can impact academic attainment, college admission, adult income, and ultimately the transmission of intergenerational poverty. Extending previous work, we showed that low SES was associated with worse cognitive functioning via increased CRP. Low SES is linked with a wide range of physical and mental health outcomes and so it is not surprising that it may affect underlying biological processes. Future work would benefit by better characterizing specific inflammatory pathways that link low SES and cognitive dysfunction. Strong theory is needed that, first, identifies specific SES-related risk factors and the mechanisms by which they lead to dysregulated inflammatory physiology; for instance, the role played by environmental risk factors, such as exposure to environmental toxins/pollution, are largely unknown and represent one potential target for intervention. Second, although there is a growing understanding of how inflammatory physiology can disrupt cognitive processes in disease states, continued focus on understanding how more subtle alterations in immune function in the periphery interacts with the central nervous system to influence cognitive functioning is needed.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbi.2022.04.020.

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