



## Processes linking socioeconomic disadvantage and neural correlates of cognitive control in adolescence

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### ABSTRACT

Socioeconomic status (SES) is broadly associated with self-regulatory abilities across childhood and adolescence. However, there is limited understanding of the mechanisms underlying this association, especially during adolescence when individuals are particularly sensitive to environmental influences. The current study tested perceived stress, household chaos, parent cognitive control, and parent-adolescent relationship quality as potential proximal mediators of the association between family SES and neural correlates of cognitive control. A sample of 167 adolescents and their primary caregivers participated in a longitudinal study across four years. SES was indexed by caregivers' education and income-to-needs ratio at Time 1. At Time 2, adolescents reported on their perceived stress, household chaos, and relationship with parents, and parents completed a cognitive control task. Two years later, adolescents completed the same cognitive control task while blood-oxygenation-level-dependent (BOLD) response was monitored with functional magnetic resonance imaging (fMRI). A parallel mediation model indicated that parent cognitive control, but not other proximal factors, explained the relation between SES and adolescents' activation in the middle frontal gyrus during a cognitive control task. The results suggest potential targets for intervention and prevention efforts that may positively alter neurocognitive outcomes related to socioeconomic disadvantage.

### 1. Introduction

Socioeconomic status (SES) represents a set of social and economic factors that reflect one's relative position in society (Farah, 2017; McLoyd, 1998) and is one contextual factor that has the potential to shape cognitive development in children and adolescents. Indeed, SES has small-to-moderate effects on executive functioning (EF) performance from early childhood to late adolescence (Lawson et al., 2017). Advances in neuroimaging methodologies have allowed for exploration of how SES may affect neural correlates of higher-order cognitive processes similar to EF, such as cognitive control. However, family income and education, in and of itself, may not directly impact neurodevelopment; rather, there may be proximal factors related to SES which in turn contribute to child and adolescent cognitive control.

Cognitive control is the flexible regulation of behavior through overriding prepotent responses (Casey et al., 2001) and involves both working memory and attention shifting. The development of cognitive

control is not purely a function of biological maturation; both biological processes and the environmental context of the child together confer individual differences in cognitive control (Rutter and Sroufe, 2000). Family SES, including factors such as parent income and education, has broadly been linked to cognitive development across childhood and adolescence. For example, poverty is associated with worse cognitive control performance in adolescence (Lambert et al., 2017). Accumulating evidence from functional magnetic resonance imaging (fMRI) research suggests that individual differences in SES may also affect related neurobiological function, evidenced by distinct patterns of activation in the prefrontal cortex (PFC) during cognitive tasks. For example, Sheridan et al. (2012) found that children from lower-SES families demonstrated higher activation in the right middle frontal gyrus during rule-learning relative to children from high-SES families. Differences in neural function during working memory have also been observed in relation to SES in youths (Rosen et al., 2018).

The effects of SES on cognitive control are of particular interest

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during the developmental period of adolescence. Adolescents may be especially vulnerable to socioeconomic influences given the heightened sensitivity to socio-environmental contexts during this developmental period (Blakemore, 2008). Indeed, initial work in this age group demonstrates that SES can affect functional activation during cognitive control and related cognitive processes. For example, Spielberg et al. (2015) found that lower SES was associated with longitudinal increases in the anterior cingulate cortex (ACC) during an inhibitory control task (albeit only for females), suggesting that female adolescents with lower SES showed less efficient inhibitory processing (i.e., required greater compensatory recruitment of the ACC). Several studies using working memory tasks have also observed distinct patterns of functional activation among adolescents from different socioeconomic backgrounds (e.g., Finn et al., 2017). In particular, Sheridan et al. (2017) found that lower parental education was associated with higher activation in the superior parietal cortex during high working memory load among adolescents. This pattern of activation was correlated with worse task performance, suggesting less efficient patterns of neural recruitment among adolescents from lower SES families. In light of these emerging findings, further empirical evidence is needed to clarify why and how SES may contribute to neural correlates of cognitive functioning during the sensitive period of adolescence.

### 1.1. Processes linking SES and neural correlates of cognitive control

SES creates a set of conditions at more proximal levels that in turn may be related to brain development. In this way, the effects of SES may cascade into disruptions in other domains, which cumulatively contribute to development over time (Masten and Cicchetti, 2010). Specifically, SES may shape neurocognitive development through cognitive enrichment (Amso et al., 2019) and stress (Conger et al., 2010) pathways. While both of these pathways contribute to adolescent development, here, we focus on intraindividual, familial, and environmental levels of influence that may be particularly shaped by financial stress. These three levels have been identified as sources of resilience across development (Masten and Garmezy, 1985; Werner and Smith, 1982) and may link family SES with adolescent neurodevelopment. Accordingly, we considered potential mediators at each of these levels including adolescents' perceived stress (intraindividual), parent cognitive control and the parent-adolescent relationship (familial), and household chaos (environmental).

#### 1.1.1. Perceived stress

The effects of SES on family stress are well-established (Conger et al., 2010). Children from lower income families experience more stressors and elevated physiological stress relative to higher income families (Evans and English, 2002). In turn, stress affects brain structure and function, the effects of which may manifest differently across different developmental periods (Lupien et al., 2009). For example, in adolescence, the most profound effects of stress are expected in the frontal cortex because of its ongoing and protracted development, relative to other regions that mature earlier in development (e.g., hippocampus). While there is clear evidence for the associations between SES, stress, and brain function, few studies have tested these factors simultaneously to examine the mediating processes of stress during adolescence. However, there is compelling evidence for stress as a mediating factor from other developmental periods. Luby et al. (2013) found that income-to-needs ratio in preschool was negatively associated with stressful life events in childhood, which in turn predicted hippocampal volume in middle childhood/preadolescence. Additional research has shown that these patterns of effects may extend even into young adulthood, with chronic stress at ages 9–17 mediating the effect of childhood income at age 9 on brain activation (ventrolateral PFC and dorsolateral PFC) during emotion regulation at age 24 (Kim et al., 2013). Results from the same cohort found a significant association between self-reported chronic stress during childhood and brain activation

(medial PFC but not ventrolateral PFC or dorsolateral PFC) during emotion regulation in adulthood (Javanbakht et al., 2015).

#### 1.1.2. Household chaos

The physical home environment is shaped in part by family SES. In particular, household chaos is correlated with SES and reflects noise, crowding, and lack of structure or routine in the home (Wachs and Evans, 2010). Behavioral research has demonstrated that household chaos is predictive of longitudinal trajectories of self-control across childhood (Holmes et al., 2018). In adolescence, household chaos has been shown to exacerbate risk for lower EF abilities (Briant et al., 2017). These findings have not yet been extended to neuroimaging work, and it remains unclear whether household chaos may impinge upon functional development of the brain. However, given that SES often underlies chaotic home conditions which can disrupt self-regulation development, it is reasonable to believe that chaos may serve as a process linking SES and neural correlates of cognitive control.

#### 1.1.3. Parent cognitive control

A psychobiological model of the intergenerational transmission of self-regulation posits that parents transmit their self-regulatory abilities to their children through both biological and environmental factors (Deater-Deckard, 2014). That is, in addition to shared genes, parents' self-regulatory abilities are associated with caregiving behaviors and socialization practices which can promote or compromise self-regulatory development in their children (Cuevas et al., 2014). These parental regulatory processes may be challenged in the face of socioeconomic stress, with downstream consequences for children's own self-regulation. Indeed, financial or poverty-related concerns take up mental resources, thereby reducing capacity for other cognitive demands (Mani et al., 2013; Mullainathan and Shafir, 2013). Thus, if parents' own self-regulatory abilities are influenced by SES, this could ultimately impact neural and behavioral indicators of self-regulation in their children. While these associations have yet to be tested empirically in adolescents, initial evidence demonstrates that lower parent EF was associated with lower adolescent EF among families with high household chaos (Briant et al., 2017). Further research is needed to determine whether this transmission manifests on a neurobiological level, and may be influenced by family SES.

#### 1.1.4. Parent-adolescent relationship

The nature of a parent's relationship with their child can shape brain structure and function (see Belsky and De Haan, 2011 for a review). During adolescence, the parent-child relationship undergoes restructuring as children begin to seek autonomy and spend more time with same-age peers (Branje, 2018). However, parents remain an important social agent throughout adolescence, and this relationship may continue to affect development of the frontal cortex. Indeed, heightened negativity in the parent-adolescent relationship (e.g., aggression) has been associated with maladaptive structural maturation in the frontal cortex (Schwartz et al., 2017). Functionally, negative family relationship quality in adolescence was associated with longitudinal increases in brain activation (ventrolateral PFC) during a cognitive control task, indicating that relationships characterized by low cohesion and high conflict compromise functional maturation in regions involved in cognitive control (McCormick et al., 2016).

### 1.2. Present study

The goal of the current study was to examine multiple proximal factors through which SES may affect prefrontal functioning related to cognitive control, thereby identifying factors that can be targeted in intervention and prevention efforts to positively alter maladaptive trajectories related to socioeconomic disadvantage. Thus, we considered how socioeconomic disadvantage (indexed by income-to-needs ratio and parent education) in early adolescence may affect more proximal

factors one year later, and whether these proximal factors would cascade into differences in cognitive control in late adolescence. Specifically, we hypothesized that lower SES at Time 1 would be associated with higher perceived stress, higher household chaos, lower parent cognitive control, and a more negative parent-adolescent relationship at Time 2. We further hypothesized that each of these factors would in turn predict greater adolescent prefrontal activation (reflecting less efficient neural recruitment) during a cognitive control task two years later.

## 2. Method

### 2.1. Participants

Participants included 167 adolescents (53 % male) and their primary caregivers (82 % biological mothers, 13 % biological fathers, 2% grandmothers, 1% foster, 2% other) who participated in an ongoing longitudinal study across four years. Adolescents were 13–14 years of age at Time 1 ( $M = 14.07$ ,  $SD = 0.54$ ), 14–15 years of age at Time 2 ( $M = 15.05$ ,  $SD = 0.54$ ), and 16–17 years of age at Time 4 ( $M = 17.01$ ,  $SD = 0.55$ ). Adolescents primarily identified as White (78 %), 14 % as Black or African-American, 6% as more than one race, 1% as American Indian or Alaska Native, and 1% Asian. Median annual household income fell between \$35,000-\$50,000 (consistent with the median for the region; [United States Census Bureau, 2010](#)), ranging from less than \$1000 to greater than \$200,000 per year.

At Time 1, 157 families participated. At Time 2, 10 families were added for a final sample of 167 parent-adolescent dyads. However, 24 families did not participate at all possible time points for reasons including: ineligibility for tasks ( $n = 2$ ), declined participation ( $n = 17$ ), and lost contact ( $n = 5$ ) during the follow-up assessments. Rate of participation (indexed by proportion of years participated to years invited to participate) was not significantly predicted by demographic or study variables at Time 1 ( $p > .05$ ).

### 2.2. Procedures

Participants were recruited from the community via flyers, recruitment letters, and e-mail in the Southeastern United States, including small cities and rural towns and counties in Appalachia. Data collection occurred at university offices where adolescents agreed to participate via written assent, while parents provided written consent, and were then administered the protocol by trained research assistants. The procedures took approximately five hours total. Adolescents and their parents received monetary compensation for their time. These procedures were reviewed and approved by the university's Institutional Review Board.

### 2.3. Measures

#### 2.3.1. Socioeconomic status

At Time 1, caregivers completed a demographic interview which included questions about their and their spouse's (if applicable) income and education. Total household income before taxes for the previous year was used to calculate an income-to-needs (ITN) ratio for each family. Specifically, income was divided by the poverty threshold for the given family size (according to guidelines by the U.S. Census Bureau). Number of years of education was averaged across the primary caregiver and their spouse (when applicable). Education and ITN were significantly correlated ( $r = .48$ ,  $p < .01$ ) and so these two variables were standardized and then averaged to create a composite SES score.

#### 2.3.2. Perceived stress

At Time 2, perceived stress was measured by adolescent report on the Perceived Stress Scale short form (PSS; [Cohen et al., 1983](#); [Cohen and Williamson, 1988](#)). The short form of the scale includes ten items which ask participants to indicate how often they had thought or felt a certain

way in the last month from "0 = never" to "4 = very often". Example items include, "In the last month, how often have you felt nervous and 'stressed'?" and "In the last month, how often have you felt that you were on top of things?" (reverse scored). The ten items were averaged, with higher scores representing higher levels of perceived stress. The scale demonstrates good reliability in our sample ( $\alpha = .83$ ).

#### 2.3.3. Household chaos

At Time 2, household chaos was measured by adolescent report on the short version of the Chaos, Hubbub, and Order Scale (CHAOS; [Matheny et al., 1995](#)). The scale includes six items that ask about characteristics of the home environment, including noise, crowding, and lack of structure or routine. Responses are given on a 5-point Likert-type scale from "1 = definitely untrue" to "5 = definitely true" with higher scores indicating higher levels of chaos in the home. Example items include, "We are usually able to stay on top of things" and "You can't hear yourself think in our home". The scale demonstrates relatively low reliability within the current sample ( $\alpha = .64$ ), which is consistent with previous research using the six-item CHAOS ([Coldwell et al., 2006](#); [Pike et al., 2006](#)).

#### 2.3.4. Parent-adolescent relationship

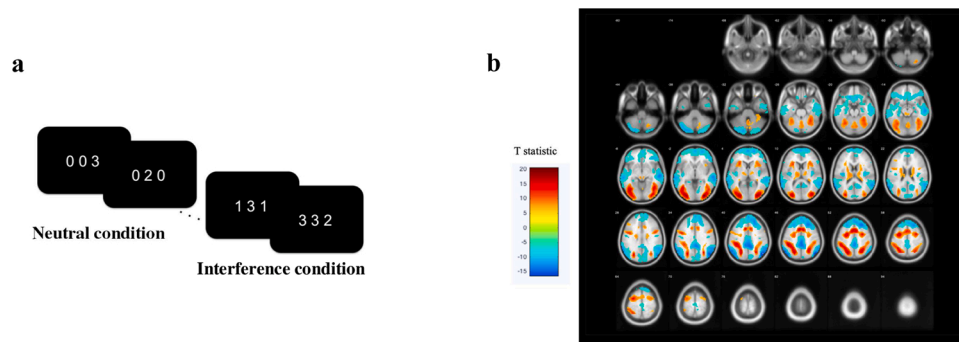
At Time 2, adolescents reported on the quality of their relationship with the caregiver who was participating in the study with them. The Parent-Child Relationship Scale ([Hetherington and Clingempeel, 1992](#)) includes seven items that ask about negativity within the relationship. Adolescents were asked to rate each item on a 5-point Likert-type scale, from "1 = extremely" to "5 = not at all". Example items include, "How much does this person yell at you after you've had a bad day?" and "How much do you criticize this person?". Responses were reverse-scored such that higher scores represented greater negativity within the parent-adolescent relationship. The scale demonstrates good reliability in our sample ( $\alpha = .86$ ).

#### 2.3.5. Cognitive control

Parent and adolescent cognitive control was evaluated with the Multi-Source Interference Task (MSIT; [Bush and Shin, 2006](#)), a computerized cognitive interference task. Participants were presented with three digits, two of which were identical, and asked to indicate the identity of the oddball digit using a button press. In neutral trials, a target's identity matched the digit's presented location, but in interference trials, the target's identity was incongruent with the digit's presented location ([Fig. 1a](#)). The task consisted of 96 neutral trials and 96 interference trials, interleaved in blocks of 24 trials. Preceding and following the task-active blocks were 26 s periods of rest.

**2.3.5.1. Behavioral data.** In line with previous studies ([Bush et al., 2003](#)), we found significant MSIT interference effects in reaction time for correct responses, such that reaction time was higher for interference compared to neutral trials ( $t_{(147)} = 47.68$ ,  $p < .001$ ). We calculated intraindividual variability in reaction time, indexed as intraindividual standard deviations (ISDs) for correct responses in the interference condition ([MacDonald et al., 2012](#)). Greater variability in response time indicated lower cognitive control ability. We used parents' ISDs as a measure of their cognitive control performance at Time 2, and adolescents' ISDs as a measure of their cognitive control performance at Time 4.

**2.3.5.2. Neuroimaging data.** Adolescents completed the MSIT at Time 4 while blood oxygenation level dependent (BOLD) responses were recorded using functional magnetic resonance imaging (fMRI). Prior neuroimaging work has shown that the MSIT is reliably associated with activation in regions of the prefrontal cortex involved in cognitive control ([Deng et al., 2018](#)), and that behavioral performance is correlated with frontal activation during the MSIT, such that better



**Fig. 1.** A) In the multi-source interference task (MSIT), participants were asked to identify the digit that differed from two other concurrently presented digits, ignoring its position in the sequence. B) Statistical parametric map of the Interference – Neutral contrast, displayed at a voxel-threshold of  $p < .001$  (Family-Wise Error correction).

performance is associated with lower BOLD response (Bush et al., 2003).

**2.3.5.3. Imaging acquisition and analysis.** Functional neuroimaging data were acquired on a 3 T Siemens Tim Trio MRI scanner with a standard 12-channel head matrix coil. Structural images were acquired using a high-resolution magnetization prepared rapid acquisition gradient echo sequence with the following parameters: repetition time (TR) = 1200 ms, echo time (TE) = 2.66 ms, field of view (FoV) = 245 × 245 mm, and 192 slices with the spatial resolution of 1 × 1 × 1 mm. Echo-planar images were collected using the following parameters: slice thickness = 4 mm, 34 axial slices, FoV = 220 × 220 mm, TR = 2 s, TE = 30 ms, flip angle = 90 degrees, voxel size = 3.4 × 3.4 × 4 mm, 64 × 64 grid, and slices were hyperangulated at 30 degrees from anterior-posterior commissure. Imaging data were preprocessed and analyzed using SPM8 (Wellcome Trust Neuroimaging Center). For each scan, data were corrected for head motion using a six-parameter rigid body transformation and realigned. The mean functional image was co-registered to the anatomical image, then the anatomical image was segmented and registered to the MNI template and functional volumes were normalized using parameters from the segmented anatomical image, and were smoothed using a 6 mm full-width-half-maximum Gaussian filter.

For each participant, the preprocessed functional data were submitted to a General Linear Model (GLM) using the SPM8 toolbox. Interference and neutral task conditions were modeled as boxcars convolved with the canonical hemodynamic response function (HRF), using pre-task and post-task as an implicit baseline. A high-pass filter with cutoff of 0.006 Hz was used to remove the effect of low-frequency noise. Six realignment parameters were included to model head motion. A contrast map was obtained by subtracting the beta map from the neutral condition from the beta map of the interfere condition.

A group level analysis was performed in SPM8 on the first-level Interference-Neutral contrasts to identify peak regions of interference effect. For each participant, first eigenvariate values were extracted from individual-level regions-of-interest (ROI) corresponding to 6 mm-radius spheres centered at coordinates of peak activation in the interference trial minus neutral trial group-level contrast (see Table 1 for MNI coordinates; Fig. 1b for activation map). Among these ROIs, the left middle frontal gyrus (MFG), corresponding to Brodmann area 9, was selected as the outcome variable given both its involvement in cognitive control and demonstrated associations with SES (Finn et al., 2017; Rosen et al., 2018). We examined correlations with other prefrontal ROIs but there were no significant associations with SES (see Table S1). Consistent with findings by Bush et al. (2003), we found that lower variability in reaction time (indicating better cognitive control) was related to lower BOLD responses during the MSIT.

#### 2.4. Plan of analysis

Skewness and kurtosis were examined for all variable distributions

**Table 1**

Areas of significant activation for the contrast of interference minus neutral blocks of the Multi-Source Interference Task.

Cluster #	Region	Size	Peak MNI Coordinates			T
			x	y	z	
1	R Inferior Occipital Gyrus	1938	33	-88	-2	18.71
	R Middle Occipital Gyrus		39	-85	7	17.80
	R Angular		27	-58	52	16.25
2	L Middle Occipital Gyrus	2267	-30	-88	1	17.84
	L Inferior Parietal Lobule		-42	-37	43	16.94
	L Inferior Occipital Gyrus		-42	-73	-8	16.58
3	L Pre-Supplementary Motor Area	1487	-6	14	46	16.48
	L Middle Frontal Gyrus		-27	-4	58	15.85
	L Inferior Frontal Gyrus		-45	2	31	13.35
4	L Insular Cortex	146	-27	20	7	11.61
5	R Inferior Frontal Gyrus	110	48	8	31	11.24
6	R Insula	167	33	20	7	10.36
	R Putamen		24	11	7	7.90
7	R Putamen	349	27	2	13	6.86
	R Cerebellum Posterior Lobe		6	-73	-17	9.61
	Culmen		0	-55	-29	9.50
8	R Cerebellum Posterior Lobe	30	-70	-50	9.20	
	L Thalamus		162	-12	-19	13
9	Extra-Nuclear	40	-18	-7	25	8.29
	Extra-Nuclear		-30	-34	4	6.63
	Midbrain		-3	-28	-11	8.44
10	Midbrain	111	6	-28	-11	7.50
	Extra-Nuclear		21	-1	22	7.83
	R Thalamus		15	-13	13	7.76
11	Extra-Nuclear	9	24	-31	16	7.69
	Extra-Nuclear		30	-37	10	7.67
12	<b>L Middle Frontal Gyrus</b>	<b>34</b>	<b>-45</b>	<b>32</b>	<b>25</b>	<b>7.31</b>
13	Corpus Callosum	2	-3	8	19	6.42
14	Culmen	3	3	-49	-5	6.17

*Note:* MNI, Montreal Neurological Institute; L, Left; R, right. Size refers to the number of voxels in the cluster. All activations reported here survive whole-brain family-wise error multiple comparisons correction at a threshold of  $p < .001$ . Selected region of interest is in boldface.

and acceptable levels were less than 3 and 10, respectively (Kline, 2011). All variables were normally distributed. Prior to analysis, univariate outliers for study variables were identified, defined as values  $\geq 3$  SD from the mean. In these cases ( $n = 4$ ), values were winsorized to retain statistical power and attenuate bias resulting from elimination. Demographic variables (i.e., sex and race) were not associated with mediator or outcome variables ( $ps > .05$ ). The hypothesized model was tested via Structural Equation Modeling (SEM) using Mplus version 8 (Muthén and Muthén, 1998–2019; Muthén and Muthén, 1998).

and Muthén, 1998–2019). RMSEA values of less than .05 were considered a close fit while values less than .08 were considered a reasonable fit (Browne and Cudeck, 1993), and CFI values of greater than .90 were considered an acceptable fit while values greater than .95 were considered an excellent fit (Bentler, 1990). Little's MCAR test (Little, 1988) indicated that patterns of missing data on study variables were completely random ( $\chi^2 = 17.13$ ,  $df = 16$ ,  $p = .38$ ); thus, full information maximum likelihood estimation was used to handle missing data. To test significance levels of mediated effects, asymptotic and resampling strategies were used with bootstrapping, with 10,000 iterations with bias-corrected bootstrap estimations of the 95 % confidence interval (Preacher and Hayes, 2008).

### 3. Results

Descriptive statistics and correlations for all study variables are presented in Table 2. First, we fit a longitudinal parallel mediation model that included all four mediators at Time 2 (household chaos, parent cognitive control, perceived stress, and parent-adolescent relationship) simultaneously, as well as the direct effect of SES at Time 1 on MFG activation at Time 4. This model demonstrated poor fit ( $\chi^2 = 53.93$ ,  $df = 6$ ,  $p < .001$ , CFI = .30, RMSEA = 0.22); thus, we examined non-significant paths to trim the model to improve fit and parsimony. When considering all mediators simultaneously, SES at Time 1 significantly predicted parent cognitive control ( $b = -0.01$ ,  $SE = 0.004$ ,  $p = .03$ ), household chaos ( $b = -0.13$ ,  $SE = 0.06$ ,  $p = .03$ ) and perceived stress ( $b = -0.12$ ,  $SE = 0.06$ ,  $p = .04$ ) measured at Time 2. However, neither household chaos ( $b = 0.03$ ,  $SE = 0.07$ ,  $p = .67$ ) or perceived stress ( $b = -0.06$ ,  $SE = 0.07$ ,  $p = .36$ ) predicted MFG activation measured at Time 4. Furthermore, SES did not predict parent-adolescent relationship quality ( $b = -0.08$ ,  $SE = 0.06$ ,  $p = .21$ ) and parent-adolescent relationship quality did not predict MFG activation ( $b = -0.08$ ,  $SE = 0.06$ ,  $p = .14$ ). Finally, the direct effect from SES at Time 1 on MFG activation at Time 4 was not significant ( $b = -0.06$ ,  $SE = 0.05$ ,  $p = .22$ ). We trimmed all mediators that did not meet requirements for an indirect effect (i.e., were not significantly associated with the SES predictor or MFG activation outcome) as well as the non-significant direct effect, leaving parent cognitive control as the only mediator in the final, trimmed model (see Fig. 2).

The trimmed model included the association between SES at Time 1 and MFG activation at Time 4 via parent cognitive control at Time 2. This model had good fit ( $\chi^2 = 1.14$ ,  $df = 1$ ,  $p = .29$ , CFI = .99, RMSEA = 0.03) and fit was significantly better than the full, untrimmed model ( $\Delta\chi^2 = 53.79$ ,  $\Delta df = 5$ ,  $p < .001$ ). In this model, SES at Time 1 significantly predicted parent cognitive control at Time 2 ( $b = -0.01$ ,  $SE = 0.004$ ,  $p = .03$ ), and in turn, parent cognitive control significantly predicted adolescent MFG activation at Time 4 ( $b = 2.79$ ,  $SE = 0.79$ ,  $p < .001$ ). Bias-corrected bootstrapped confidence intervals indicated that the indirect effect of SES on MFG activation via parent cognitive control was significant ( $b = -0.03$ ,  $SE = 0.02$ ,  $\beta = -.05$ , 95 % CI [-0.06; -0.002]). Standardized estimates for this final model are presented in

Fig. 2.

#### 3.1. Supplementary behavioral results

In addition to the neural results, we tested whether SES was indirectly related to behavioral indices of cognitive control based on intra-individual variability in response time on the MSIT. Following the same procedure, we tested all mediators simultaneously; this model demonstrated poor fit ( $\chi^2 = 53.92$ ,  $df = 6$ ,  $p < .001$ , CFI = .33, RMSEA = 0.22). Only parent cognitive control ( $b = 0.18$ ,  $SE = 0.08$ ,  $p = .03$ ) and perceived stress ( $b = 0.01$ ,  $SE = 0.01$ ,  $p = .04$ ) were significantly associated with the behavioral outcome. Thus, all other mediators (i.e., household chaos, parent-adolescent relationship) were trimmed. The direct effect of SES on behavioral cognitive control was also not significant ( $b = -0.01$ ,  $SE = 0.004$ ,  $p = .10$ ) and thus was trimmed from the model. This trimmed model demonstrated acceptable fit ( $\chi^2 = 3.04$ ,  $df = 2$ ,  $p = .22$ , CFI = .95, RMSEA = 0.06) which was significantly better than the original model ( $\Delta\chi^2 = 50.88$ ,  $\Delta df = 4$ ,  $p < .001$ ). In this final model, SES was significantly associated with both parent cognitive control ( $b = -0.01$ ,  $SE = 0.004$ ,  $p = .03$ ) and adolescent perceived stress ( $b = -0.12$ ,  $SE = 0.06$ ,  $p = .04$ ). In turn, both parent cognitive control ( $b = 0.21$ ,  $SE = 0.08$ ,  $p = .01$ ) and perceived stress ( $b = 0.02$ ,  $SE = 0.01$ ,  $p = .01$ ) were significantly associated with behavioral cognitive control. Bias-corrected bootstrapped confidence intervals indicated that the indirect effects of SES on behavioral cognitive control via parent cognitive control ( $b = -0.002$ ,  $SE = 0.001$ , 95 % CI [-0.01; 0.00],  $\beta = -.04$ ) and perceived stress ( $b = -0.002$ ,  $SE = 0.001$ , 95 % CI [-0.004; 0.00],  $\beta = -.03$ ) were significant.

### 4. Discussion

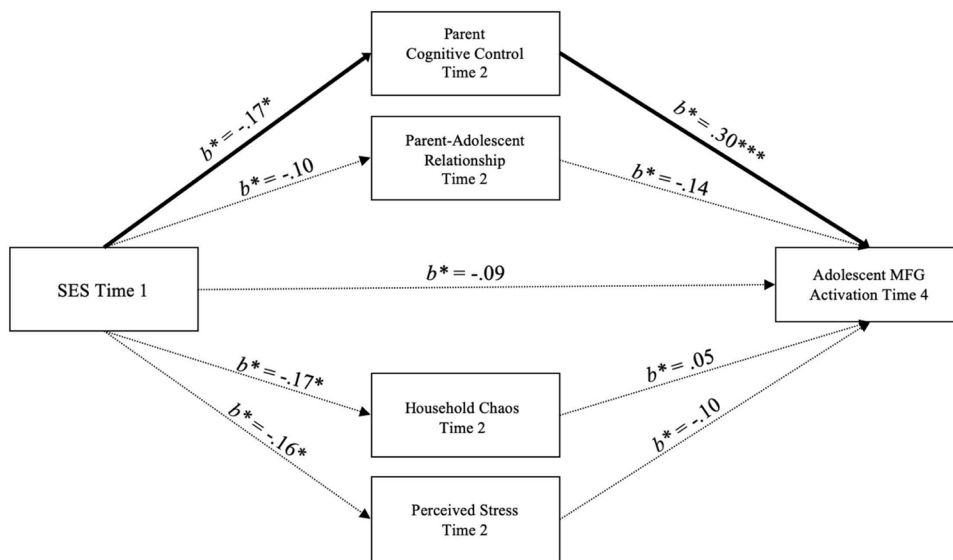
Prior empirical work suggests that SES may have broad implications for child and adolescent development via its more proximal association with intraindividual, familial, and environmental factors. Indeed, many contextual factors associated with SES have been linked to individual differences in child or adolescent cognitive control performance or task-based functional activation. However, few efforts have been made to integrate these findings in order to better understand what it is about SES that drives changes in the developing brain. Available studies that have attempted to address this question with longitudinal mediation models (e.g., Javanbakht et al., 2015; Kim et al., 2013; Luby et al., 2013) have not done so in adolescent samples. Given that adolescence is a developmental period characterized by heightened sensitivity to contextual influences (Blakemore, 2008), the present longitudinal study sought to explain how SES may be associated with neural correlates of cognitive control in adolescence. By examining four possible mediating processes relevant to adolescent development (perceived stress, household chaos, parent-adolescent relationship quality, and parent cognitive control) simultaneously, we aimed to clarify the indirect pathways whereby SES is associated with cognitive control in adolescence. We examined these associations among families from a largely rural region

**Table 2**  
Correlations and descriptive statistics for socioeconomic status, proximal mediators, and neural and behavioral cognitive control.

	1	2	3	4	5	6	7	8	<i>M (SD)</i>	<i>Min</i>	<i>Max</i>
1. Education	–								14.83 (2.42)	0.00	25.00
2. Income-to-Needs Ratio	.48**	–							2.49 (1.89)	0.00	8.39
3. SES Composite	.86**	.86**	–						0.00 (0.86)	-1.73	2.54
4. Parent Cognitive Control	-.18*	-.12	-.18*	–					0.20 (0.04)	0.08	0.32
5. Parent-Adolescent Relationship	-.07	-.11	-.10	.08	–				1.99 (0.71)	1.00	4.14
6. Household Chaos	-.13	-.17*	-.17*	.16	.39**	–			1.60 (0.63)	0.02	3.10
7. Perceived Stress	-.21*	-.07	-.16*	.04	.29**	.40**	–		2.45 (0.65)	1.17	4.00
8. Adolescent Neural Cognitive Control	-.17	-.05	-.13	.30**	-.13	.02	-.11	–	0.29 (0.42)	-0.56	1.56
9. Adolescent Behavioral Cognitive Control	-.21*	-.11	-.20*	.23**	.03	.18*	.22**	.17	0.18 (0.04)	0.10	0.31

\*  $p < .05$ .

\*\*  $p < .01$ .



**Fig. 2.** Standardized estimates of the longitudinal associations among socioeconomic status, proximal mediators, and adolescent middle frontal gyrus (MFG) activation during cognitive control. Parent cognitive control was measured by intraindividual variability in reaction time and higher values reflect worse cognitive control performance. Dashed lines represent paths that were trimmed, bold lines were retained in the final model.

\* $p < .05$ ; \*\* $p < .01$ ; \*\*\* $p < .001$ .

in the United States, a relatively understudied population in fMRI research.

When considering the four mediators together, parent cognitive control emerged as the strongest factor underlying the association between SES and neural and behavioral correlates of cognitive control. Specifically, lower SES was associated with lower parent cognitive control, which in turn predicted higher prefrontal activation during cognitive control as well as poorer cognitive control performance in adolescents. This finding is consistent with the heuristic model of the intergenerational transmission of self-regulation (Deater-Deckard, 2014) which stipulates that individual differences in self-regulation (including cognitive control) are transmitted from parents to their children through both socialization and biological processes. Prior research has only considered transmission of behavioral outcomes, and empirical evidence for intergenerational transmission has largely focused on younger children (e.g., Cuevas et al., 2014). However, initial findings demonstrate that these transmission processes continue into adolescence (Briant et al., 2017; Jester et al., 2009). Our results provide additional support for the transmission of self-regulation during this important developmental period, as well as novel evidence of transmission across multiple levels of analysis. That is, better behavioral cognitive control in parents predicted better behavioral *and* neural cognitive control among adolescents.

We first consider the possibility that these results reflect genetic effects. In the extant literature, behavior genetics studies have demonstrated that cognitive abilities related to interference and inhibition are genetically influenced and heritable (50–60 %; e.g., Anokhin et al., 2004; Malone and Iacono, 2002). A twin study which similarly used the Multi-Source Interference Task found moderate heritability (i.e., 37 % of the variance attributable to genetic effects) in activation in the dorsal anterior cingulate cortex (dACC), whereas behavioral responses on the task (i.e., difference in reaction time between interference and neutral condition) were not significantly influenced by genetic factors (Matthews et al., 2007). While there is evidence for heritability in some domains, this does not necessarily mean that environmental factors are not at play (Friedman et al., 2008), and the similarity we observed between parent performance on the task and adolescent performance and activation is likely not entirely genetic in nature. Though the present study cannot discount the potential genetic mechanisms at play, we adopt a psychobiological model of development which also emphasizes environmental, non-genetic influences. In addition to genetic transmission, concurrent socialization and parenting behaviors account in part for intergenerational similarity in self-regulation. For example,

stronger parent self-regulation is associated with behaviors such as sensitivity and responsiveness (Barrett and Fleming, 2011) which in turn shape children's own self-regulation, thereby fostering intergenerational similarity. There is also evidence that in early childhood, maternal parenting behaviors (e.g., facilitating attention, intrusiveness) explained the association between maternal and child EF (Cuevas et al., 2014). Nonetheless, further disentangling the biological and ecological mechanisms of transmission processes during adolescence, as well as elucidating their interplay, will be an important direction for future research.

Importantly, the heuristic model of intergenerational transmission of self-regulation (Deater-Deckard, 2014) is embedded within the broader home and family context, suggesting that exposure to any form of chronic stress may disrupt healthy development of self-regulation abilities. That is, as parents' own cognitive control abilities are affected by their SES, this will in turn impact their children's cognitive control. Consistently, our findings illustrate how low SES can impair adolescents' cognitive control indirectly via parent cognitive control. This effect may emerge in part due to the cognitively taxing demands associated with socioeconomic disadvantage (Mani et al., 2013; Mullainathan and Shafir, 2013) and may be particularly salient in our sample of largely rural families given unique financial stressors and access to resources in rural communities (Conger et al., 1994). These findings suggest that parents' own cognitive control may be a useful target for intervention and prevention efforts aimed at strengthening cognitive control abilities in adolescents. Such interventions may be particularly beneficial for parents in low SES households in order to facilitate adaptive regulatory responses to the demands associated with lower SES environments. The modeling of such skills at home is likely to promote better cognitive control related abilities and corresponding brain functioning in their adolescents. At the same time, it is possible that broader contextual factors associated with SES may be affecting both parent and adolescent cognitive control. Indeed, adolescent neighborhood disadvantage has demonstrated effects on later reward-related brain function (Gonzalez et al., 2016); however, findings related to prefrontal functioning in adolescence have been inconsistent across samples (Gard et al., 2020).

Our results also demonstrated a direct association between SES and adolescent perceived stress, corroborating previous literature (e.g., Finkelstein et al., 2007). In turn, perceived stress impaired behavioral cognitive control, illustrating one pathway whereby SES may disrupt cognitive resources with possible consequences for later adjustment. While perceived stress predicted adolescent behavioral cognitive control, it did not predict neural cognitive control. This finding appears to

be inconsistent with the neuroscience literature documenting the deleterious effects of stress on prefrontal functioning (Kim et al., 2013; Liston et al., 2009; Mueller et al., 2010). The discrepancy in findings may indicate that *how* stress effects are measured (e.g., subjective versus objective; concurrent versus early; traumatic versus not) have differential predictive validity for brain functioning during adolescence. Prior research examining the impact of stress on brain development has focused on objective, chronic stress experienced in early life, whereas the present study assessed subjective, recent stress. Indeed, temporal patterns of stress may be associated with distinct behavioral and neurobiological outcomes (Sheth et al., 2017) and chronic stress is likely a stronger predictor of neural alterations in adolescence.

Results demonstrated a direct association between lower SES and higher household chaos, consistent with previous literature. Income and education have broad implications for family contexts, including limited physical and psychological resources (e.g., knowledge of optimal parenting practices, social support networks) that can create a more challenging home environment for youth to navigate (Evans and English, 2002; Wachs and Evans, 2010). One way that these challenges may manifest is through noise, crowding, and lack of structure in the home. However, the effects of SES on household chaos did not extend to either behavioral or neural cognitive control outcomes. Increased time spent in contexts outside of the family (e.g., peers, school, neighborhood, extracurricular) during adolescence may mean that higher levels of chaos in the home contribute less to cognitive control in adolescence relative to earlier developmental periods. Furthermore, prior studies suggest that household chaos may play an important role as a moderator of effects on adolescent cognitive functioning (Brieant et al., 2017; Kim-Spoon et al., 2017), rather than a direct predictor. However, replication—particularly across development—is warranted, especially considering that this is the first study to examine whether household chaos may impinge upon development of the brain.

Negativity in the parent-adolescent relationship was not predicted by SES nor was it predictive of behavioral or neural cognitive control. Prior evidence suggests that lower SES may place a strain on both the physical and emotional resources of parents, creating a disruption in optimal parenting that may have implications for parent-adolescent relationship quality (Kotchick and Forehand, 2002). However, given adolescents' increasing reliance on peers for support compared to parents (Brown and Larson, 2009), and developmentally normative increases in negativity and decreases in closeness between parents and adolescents (Branje, 2018), SES may not play as large of a role in negative parent-adolescent relationships relative to other factors. The non-significant association between parent-adolescent relationship quality and adolescent neural cognitive control appears to be inconsistent with previous research identifying links between particular facets of the parent-adolescent relationship and adolescent neural cognitive control (McCormick et al., 2016). However, comparing research on parent-adolescent relationships is difficult in that the parent-adolescent relationship is a multifaceted construct and different studies measure different facets of parent-adolescent relationships. For example, in contrast to studies which assessed conflict and family cohesion in relation to neural cognitive control (McCormick et al., 2016), the present study measured negativity within the parent-adolescent relationship. While negativity in the parent-adolescent relationship may impair optimal socialization processes by which parents may transmit adaptive cognitive control related behaviors to their adolescents, results across studies are inconsistent (see Li et al., 2019 for a meta-analysis) and may depend on factors such as age of the adolescent and parent/adolescent gender.

Taken together, our findings demonstrate the effect of SES on intraindividual, familial, and environmental levels of influence (Masten and Garmezy, 1985) such that SES may confer risk or resilience for more specific features of adolescents' lives such as stress, household chaos, and parents' cognitive control. However, the familial factor represented by parents' cognitive control was the only factor that was significantly

associated with adolescent brain function during cognitive control, highlighting the importance of the family (via both genetic influence and behavior) in shaping adolescent neurocognitive development. Other relational and environmental pathways (i.e., the parent-adolescent relationship and household chaos) had weaker associations with adolescent neural cognitive control. This may be due in part to the fact that we measured negativity in the parent-adolescent relationship and chaotic home environments as risk factors for lower cognitive control. Rather, factors that directly reflect deprivation (e.g. Sheridan et al., 2017), such as neglectful parenting behaviors that fail to promote cognitive enrichment or home environments lacking cognitive stimulation and learning resources, may make greater contributions to neurocognitive development during adolescence. Future research may benefit by directing attention to intergenerational influences, as well as environmental and relational factors that reflect cognitive impoverishment.

#### 4.1. Limitations and future directions

Results should be interpreted in light of several limitations that may offer potential directions for future research. First, intergenerational similarity in cognitive control may indicate passive gene-environment correlations. Given that SES is confounded with cognitive abilities (e.g. Luo and Waite, 2005; Peng et al., 2019) and individuals with better cognitive control tend to achieve higher levels of education and income, the home environment may not be causally associated with offspring cognitive control outcomes. Future research using a parent-offspring behavior genetic design will be better able to ascertain unique contributions of family SES while simultaneously considering genetic transmission of self-regulation. Second, there are potential mediators, such as cognitive enrichment in the family environment (e.g., Amso et al., 2019), that we were not able to account for in this study. Consideration of both stress and enrichment pathways is an important future direction in understanding the effects of SES on adolescent development. Future work will also benefit from considering shared environmental experiences that are related to low SES (e.g., neighborhood disadvantage) and may impact both parent and adolescent cognitive control. Next, although we used a sample with diverse SES representation from understudied communities, racial diversity was limited. Given the degree to which SES systemically intersects with race in the United States, it will be important for future studies with fewer sample restrictions to test these associations and generalize to socioeconomically disadvantaged samples that are more racially diverse. Finally, we acknowledge that not all adolescents facing socioeconomic disadvantage will develop deficits in cognitive control. Thus, future research will benefit from considering additional individual and contextual factors that may moderate these associations and promote resilience.

## 5. Conclusions

The present longitudinal study provides support for an indirect association between SES and behavioral and neural cognitive control during adolescence, and also tested several potential underlying mechanisms (i.e., perceived stress, household chaos, parent-adolescent relationship quality, and parent cognitive control) to elucidate why this association may exist. Given that adolescents are particularly sensitive to environmental influences (Blakemore, 2008), identifying such pathways using multiple competing mediators is an imperative step toward facilitating healthy development for youth facing socioeconomic disadvantage. Furthermore, given the well-established link between self-regulatory abilities and psychopathology, identifying how SES and its more proximal factors contribute to ontogeny and individual differences in cognitive control has important implications for intervention efforts. Given evidence for intergenerational transmission of self-regulation (Deater-Deckard, 2014), parents who consistently demonstrate better cognitive control will transmit such skills to their adolescents. Moreover, our findings suggest that intervention efforts

targeting cognitive control may be especially important for adolescents facing socioeconomic disadvantage, ultimately contributing to reductions in disparities in psychosocial outcomes for adolescents facing challenges related to growing up in low SES families.

### Datasharing Agreement

The datasets generated and/or analyzed during the current study are not currently publicly available but are available from the corresponding author on request.

### Declaration of Competing Interest

The authors report no declarations of interest.

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### Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:<https://doi.org/10.1016/j.dcn.2021.100935>.

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