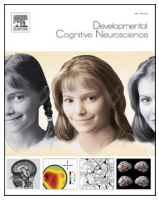
Developmental Cognitive Neuroscience 57 (2022) 101139

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: www.elsevier.com/locate/dcn



Development of cognitive control during adolescence: The integrative effects of family socioeconomic status and parenting behaviors

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ARTICLE INFO

Keywords:
Cognitive control
Adolescence
FMRI
Socioeconomic status
Parental monitoring
Parental warmth
Latent growth modeling

ABSTRACT

Cognitive control is of great interest to researchers and practitioners. The concurrent association between family socioeconomic status (SES) and adolescent cognitive control is well-documented. However, little is known about whether and how SES relates to individual differences in the development of adolescent cognitive control. The current four-year longitudinal investigation (N = 167, 13–14 years at Wave 1) used multi-source interference task performance (reaction time in interference correct trials minus neutral correct trials) and corresponding neural activities (blood oxygen level dependent contrast of interference versus neutral conditions) as measures of cognitive control. SES and parenting behaviors (warmth, monitoring) were measured through surveys. We examined direct and indirect effects of earlier SES on the development of cognitive control via parenting behaviors; the moderating effect of parenting also was explored. Results of latent growth modeling (LGM) revealed significant interactive effects between SES and parenting predicting behavioral and neural measures of cognitive control. Lower family SES was associated with poorer cognitive performance when coupled with low parental warmth. In contrast, higher family SES was associated with greater improvement in performance, as well as a higher intercept and steeper decrease in frontoparietal activation over time, when coupled with high parental monitoring. These findings extend prior cross-sectional evidence to show the moderating effect of the parenting environment on the potential effects of SES on developmental changes in adolescent cognitive control.

1. Introduction

Adolescence is a crucial transition phase of both opportunities and risks (Dahl, 2004). With the onset of puberty, adolescents are experiencing a second sensitive period of brain development, particularly in regions that are involved in higher-level cognitive process and goal-directed behaviors (Steinberg et al., 2018; Tamnes et al., 2013). However, adolescents are also facing increasing behavioral, affective and interpersonal challenges compared to childhood. For instance, previous work has identified adolescence as a time of increased risk taking (Duell et al., 2018) and greater risk for psychopathology (Lee et al., 2014). Cognitive and neurobiological models suggest that cognitive control ability is critical in decision making and promoting healthy behaviors in adolescence (Casey et al., 2005; Kim-Spoon et al., 2017). However, prospective longitudinal studies investigating the developmental changes of cognitive control during adolescence are rare. There is evidence indicating that brain function underlying cognitive control continues to develop throughout adolescence (Kim-Spoon et al., 2021; Ordaz et al., 2013), yet socioecological contexts that support or hinder within-person developmental trajectories of cognitive control during this period have not been clearly understood. The current study addresses this gap by examining the additive and interactive links between family socioeconomic status (SES) and parenting (i.e., warmth and monitoring) with developmental changes in cognitive control across mid-adolescence (from 14 to 17 years), the period of development when risky decision making and health risk behaviors emerge and increase markedly (Steinberg et al., 2018).

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https://doi.org/10.1016/j.dcn.2022.101139
Received 12 November 2021; Received in revised form 13 July 2022; Accepted 16 July 2022
Available online 20 July 2022

1.1. Socioeconomic disparities in cognitive control development

In the current study, we investigated cognitive control development during adolescence, observed by brain activation and behavioral performance during the multi-source interference task (MSIT; Bush et al., 2003). Our operationalization of cognitive control focuses on behavioral responses and neural activation needed to overcome conflict and to perform effectively (Kerns et al., 2004), largely reflecting the ability to voluntarily suppress task-irrelevant, prepotent responses in favor of goal-directed responses (Crone and Steinbeis, 2017; Luna et al., 2010). The ability to detect and respond to behavioral errors is a critical component of cognitive control and is supported by a network of regions, including the frontoparietal network previously identified as involved in cognitive control (Bush and Shin, 2006; Dosenbach et al., 2008; Luna et al., 2010). Throughout adolescence, prefrontal cortex regions—known to be critical in cognitive control—undergo maturation, including increased myelination, experience-dependent synaptogenesis and pruning, as well as strengthening of connections within prefrontal circuitry (Liston et al., 2006; Luna et al., 2015; Paus, 2005). Indeed, the notable development and specialization of prefrontal cortex regions that support higher-order cognition during adolescence marks adolescence as a neurobiological critical period (Larsen and Luna, 2018). Neuroimaging studies have implicated several regions of the frontoparietal network to be involved in cognitive control, such as the insula, inferior frontal gyrus, supplementary motor area (SMA), and the precuneus (Aron et al., 2014; Bartoli et al., 2018; Sebastian et al., 2013; Zhang et al., 2012, 2017). For example, Sebastian et al. found that frontoparietal circuits were more strongly related to interference response inhibition, compared to other components of cognitive processing, and Spielberg et al. (2015) found that activation in the dACC, MFG, and IFG exhibited change over time during the NoGo (vs. Go) task.

Further, due to the protracted development of prefrontal cortex, cognitive control is viewed to be sensitive to environmental influences. Adverse environments such as low family SES and poverty can impede cognitive control performance in adolescence (Brieant et al., 2021; Lambert et al., 2016; Lawson et al., 2018). The association between SES and cognitive control also has implications for psychopathology; specifically, poorer cognitive control contributes to the association between low SES and growth in externalizing behavior problems (McNeilly et al., 2021). Although, little is known about the association between SES and developmental change in cognitive control. Two longitudinal studies indicate that higher SES is associated with better initial and magnitude of growth in behavioral cognitive control from 11 to 19 years (Boelema et al., 2014) and that lower SES was associated with worsening cognitive control performance from 11 to 13 years (Spielberg et al., 2015).

Turning to neuroimaging studies (anatomical and functional), family SES plays an important role in the development of cognitive control-related brain regions (Noble et al., 2015; Ursache and Noble, 2016, 2019, for reviews). For instance, Spielberg and colleagues (2015) reported that lower SES was associated with worsening performance (accuracy) using the go-no-go task over a 2-year period for female adolescents, reflecting developmental changes towards decreasing behavioral cognitive control. At the same time, lower SES was related to increased no-go vs. go activation in dorsal anterior cingulate cortex (ACC), which has been shown to instantiate top-down control during inhibition. Thus, the results suggested less efficient cognitive control processing (i.e., requiring greater recruitment of the ACC) among female adolescents from lower SES families.

In another sample of adolescents, negative income slope (decreasing income) was associated with more negative connectivity between the posterior cingulate cortex (PCC) and right inferior frontal gyrus, indicating that poverty-related stress may disrupt the development of PCC connectivity (Weissman et al., 2018). Previous studies have also found that family income and parental educational attainment were associated with greater surface area in the inferior frontal gyrus and ACC, regions involved in cognitive control and executive functioning (Noble et al., 2015; Brito and Noble, 2018). Additionally, a recent meta-analysis and systematic review found that deprivation and SES were consistently associated with cortical thinning in frontoparietal networks (Colich et al., 2020). Similarly, in a sample of children and adolescents, higher family SES (using income-to-needs ratio) was associated with higher cognitive stimulation in the home, which was then positively associated with cortical thickness in the frontoparietal network (Rosen et al., 2018). These findings suggest that family SES may influence brain development during adolescence in areas of cognitive control; however, further evidence is needed to elucidate how SES may contribute to the longitudinal development of and changes in cognitive control during this sensitive period of adolescence.

1.2. Parenting behaviors and adolescent cognitive control

Family SES clearly is important, but its impacts on adolescent cognitive control development operate in transaction with more proximal features of the home, neighborhood, school and broader environment (Hyde et al., 2020). Specifically, Rakesh and colleagues found that household indicators of SES moderated the association between neighborhood indicators of SES and resting-state connectivity patterns within cognitive networks (Rakesh et al., 2021). Additionally, neighborhood disadvantage in adolescence, but not childhood, was associated with less prefrontal reactivity after accounting for other family-level adversities (Gard et al., 2021). We focused on the parenting environment in the current study, given that parenting behaviors covary with family SES across childhood and adolescence (Hoff and Laursen, 2019), and that normative variation in parenting behaviors is associated with structural and functional brain development during adolescence (Belsky and de Haan, 2011; Luby et al., 2013; Whittle et al., 2016, 2017). We propose that two specific domains of parenting— warmth and monitoring—may play particularly important roles in cognitive control development.

Parental warmth represents supportive responsiveness to children's and adolescents' behaviors (Landry et al., 2006; Romm and Metzger, 2021). The literature on children's cognitive control as well as other related aspects of executive function and self-regulation more broadly, indicates that greater parental warmth promotes internalization of self-regulation skills and strategies that guide adaptive functioning (Berthelsen et al., 2017; Hughes and Ensor, 2011). Turning to *adolescents*, although there are no prior studies of parental warmth and adolescent cognitive control specifically, there are a few studies on warmth and measures (usually questionnaires) of adolescent broad or general self-regulation of emotions, cognitions and behaviors, and the results are mixed. Several studies reported positive correlations between warmth and adolescents' self-reported self-regulation (among 10–14 year olds in Finkenauer et al., 2005, and 11–17 year olds in Moilanen, 2007) or longitudinal growth in regulation in the transition to early adolescence (from 9 to 11 years in Eisenberg et al., 2005), but other findings indicate no such associations (among 11–16 year olds in Eisenberg et al., 2005, or among 7–16 years olds in Samuelson et al., 2012). These mixed results do not appear to be due to age differences in the samples, which is an important consideration when studying the transition to and through adolescence. This issue aside, it also is important to note that there are studies showing robust associations in adolescence between parental warmth and youth outcomes that are related to cognitive control, such as academic achievement (e.g., Pinquart, 2016), anxiety/depression symptoms (e.g., Butterfield et al., 2021, which also included investigation of covarying neural activity), and antisocial behavior (e.g., Rothenberg et al., 2020, a five-year longitudinal study in 12 cultures). This broader literature on parental warmth in adolescence lends further credence to considering it in the current research.

Parental monitoring encompasses knowledge about and supervision of the adolescent's whereabouts, activities, and peers (Dishion and McMahon, 1998). Monitoring indicates parental investment in providing a consistent set of socialization experiences for their children

that promotes self-regulation skills and protection from exposure to risk factors. As with warmth, no prior studies have examined monitoring and adolescent cognitive control directly, but several have examined broader measures of self-regulation. Similar to the findings for warmth, results on monitoring have been mixed (i.e., no association, Moilanen, 2007; positive association between monitoring and self-regulation, Atherton et al., 2020; Bowers et al., 2011; Kim-Spoon et al., 2014). Thus, prior evidence is suggestive of links with cognitive control, but the current study will be the first to directly estimate these effects.

1.3. SES and parenting transactions: mediation, moderation, or both?

Contemporary views indicate that family SES and the parenting environment work together, additively and interactively, to influence brain development of the next generation (Hyde et al., 2020). As Hyde and colleagues note, little is yet known regarding how specific aspects of the home environment (including parenting behavior) mediate effects of SES, or whether those specific aspects of the environment interact with (in addition to or instead of mediating) effects of SES, on the developing brain. In the current study, we tested both of these types of SES/parenting environment transactions, in order to fully examine their interplay. First, parenting may *mediate* the link between SES and adolescent cognitive control, whereby lower SES is associated with less warm and lower monitoring parenting, which in turn is associated with lower cognitive control. Family stress theory has posited that family economic stress influences adolescent development via proximal harsher and less supportive parenting due to parental distress (Conger and Donnellan, 2007; Hoff and Laursen, 2019). Accordingly, harsher parenting contributes to heightened stress for the adolescents, which interferes with typical cognitive control development (Lengua et al., 2014). At the same time, higher levels of warm supportive parental investment in the youth (including higher levels of monitoring) promote healthy brain development (Hyde et al., 2020). One prior study (Luby et al., 2013) found that the detrimental effects of early childhood poverty on subsequent adolescent brain structure (i.e., smaller white and cortical gray matter and hippocampal and amygdala volumes) was mediated in part by less parental support and more parental hostility. However, prior evidence of mediation pertaining to adolescent cognitive control specifically, is scant. Several related studies in childhood have shown that lower family SES is related to less parental responsiveness, scaffolding and limit setting which in turn is related to children's lower executive functioning and effortful control (Hackman et al., 2015; Lengua et al., 2014; Sarsour et al., 2011). In contrast, the only relevant study of adolescents found that parental negativity did not mediate the link between SES and adolescent neural cognitive control three years later (Brieant et al., 2021).

Second, parenting may *moderate* the link between SES and adolescent cognitive control. Resilience theory states that factors in the social environment and within the individual can buffer against the negative effects of risk factors (Zimmerman et al., 2013). Broadly, an authoritative parenting environment (i.e., higher levels of warmth, moderate levels of control) is an important protective factor that can mitigate the effects of low family SES on a wide range of deleterious child outcomes (Weisleder et al., 2016), by reducing stress and providing more opportunities for learning effective self-regulation strategies (Lee et al., 2019; Rochette and Bernier, 2014). In the prior study of adolescents (9–15year olds), lower grandparent SES was associated with lower cognitive flexibility (but not cognitive control) for adolescents exposed to lower parental warmth, but not higher parental warmth (Lee et al., 2019). There are few prior studies that have examined the moderating role of parenting on SES effects, but two general brain imaging studies are suggestive. They converge to show that a supportive parenting environment buffers deleterious adolescent brain structural and functional outcomes against exposure to low family income (i.e., lower resting-state functional connectivity, Brody et al., 2019) or neighborhood socioeconomic disadvantage (i.e., amygdala volume, though only for males, Whittle et al., 2017).

1.4. The current study

To our knowledge there is not work testing mediation and moderation involving SES and parenting on neurodevelopmental growth trajectories of cognitive control across adolescence, although preliminary evidence is suggestive. To address these gaps in the literature, our main goal was to test hypotheses regarding the roles of family SES, maternal and paternal warmth and monitoring, with respect to longitudinal growth. We measured this longitudinal growth as improvements in behavioral (i.e., longitudinal reductions in reaction time difference in interference versus neutral conditions during a cognitive control task) and neural (i.e., longitudinal reductions in neural activity difference in interference versus neutral conditions during the same task) indicators of cognitive control across four years in adolescence (14–17 years) using latent growth models (LGM). Based on prior research, we hypothesized that these reductions in neural activity during cognitive control would occur in frontoparietal regions. First, we tested a mediation hypothesis: that the link between lower SES and slower growth in behavioral and neural indicators of cognitive control would be mediated by lower levels of parental warmth and monitoring. Second, we tested a moderation hypothesis: that we would observe either that a) an association between lower SES and slower growth in adolescent behavioral and neural indicators of cognitive control would be attenuated at higher levels of parental warmth and monitoring (i.e., a buffering moderation effect), or that b) an association between higher SES and faster growth in behavioral and neural cognitive control would be strengthened at higher levels of parental warmth and monitoring (i.e., an enhancing moderation effect).

2. Methods

2.1. Participants

The current sample included 167 adolescents (47 % females) who were 13–14 years at Wave 1 ($M = 14.13$, $SD = 0.54$), 14–15 years at Wave 2 ($M = 15.05$, $SD = 0.54$), 15–16 years at Wave 3 ($M = 16.07$, $SD = 0.56$), and 16–17 years at Wave 4 ($M = 16.48$, $SD = 0.53$). Median household income was $35,000 - $49,999, which is close to the median annual household income range of the area ($36,000 - $59,000 according to United States Census Bureau, 2010). Adolescent participants were primarily White (78 %), Black (14 %), Asian (1 %), American Indian (1 %), and more than one race (6 %). The current sample was generally representative of the region (Appalachian region of southwestern Virginia) regarding household income and ethnicity. There were 24 adolescents who did not participate at all four time points for reasons including: ineligibility for tasks ($n = 2$), declined participation ($n = 17$), and lost contact ($n = 5$) during the follow-up assessments. Logistic regression was conducted to compare those who provided data in all four waves and those who did not. Results revealed no differences in age, sex assigned at birth, ethnicity, parents' years of education, or family income ($ps > 0.15$).

2.2. Procedure

Adolescents and their parents were recruited via emails and flyers that were distributed through schools and other community locations. Research assistants described the nature of the study to interested individuals over the telephone and invited them to participate in the study. Data collection took place at the university's offices where adolescents and their primary caregivers were interviewed by trained research assistants and received monetary compensation for participation. All adolescent participants provided written assent and their parents provided written consent. The research protocol was approved by the institutional review board of the university.

2.3. Measures

2.3.1. Cognitive control

Adolescents' cognitive control was assessed using the Multi-Source Interference Task (MSIT; Bush et al., 2003) at all four waves. MSIT was selected because of its capability to probe brain behavior relationships (Ordaz et al., 2013). In this task, subjects were presented with sequences of three numbers for a duration of 1.75 s and asked to identify the unique number among three digits by pressing a button with the index finger. In the neutral condition, the distractor numbers were zeros, and the identity of the target was congruent with their position on the button box and screen. In the interference condition, the distractors were 1, 2, or 3 and the target's identity was incongruent with its position on the button box and screen (see Fig. 1A). Participants completed 4 blocks of 24 neutral trials interleaved with 4 blocks of 24 interference trials for a total of 96 neutral trials and 96 interference trials. The variable of interest was the interference effect, which was measured by reaction time. We calculated reaction time differences between correctly responded interference and neutral trials (i.e., averaged reaction time for the interference condition minus averaged reaction time for the neutral condition). Lower reaction time difference scores indicated greater cognitive control, characterized by faster reaction time for the interference condition relative to the neutral condition. Only reaction times for correct trials were used in the current analyses, given that response times for incorrect trials often yield excessive amounts of statistical noise that produce inaccurate representations of response patterns (Kane and Engle, 2003).

2.3.2. Neuroimaging data acquisition

Adolescents performed the MSIT task while their blood-oxygen-level-dependent (BOLD) responses were monitored using functional magnetic resonance imaging (fMRI). Neuroimaging data were acquired on a 3 T Siemens Tim Trio MRI scanner fitted 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: TR= 1200 ms, TE= 2.66 ms, field of view (FoV)= 245 × 245 mm, and 192 slices with the spatial resolution of 1x1x1 mm. Echo-planar images (EPIs) were collected using the following parameters: slice thickness= 4 mm, 34 axial slices, FoV= 220 x 220 mm, repetition time (TR)= 2 s, echo time (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. Neuroimaging data were preprocessed and analyzed using SPM8 (Wellcome Trust Neuroimaging Center). After correcting the functional scans for motion using a six-parameter rigid body transformation, the mean functional scan was co-registered to the corresponding anatomical image using a rigid-body transformation estimated to maximize the normalized mutual information between the anatomical and mean functional image. Then, the anatomical image was segmented to produce spatial normalization parameters which were then used to normalize the functional images to MNI-152 template. Normalization produced images resliced to an isotropic voxel size of 3 mm^3. Finally, the normalized functional images were smoothed using a 6 mm full-width-half-maximum Gaussian kernel.

2.3.3. Neural correlates of cognitive control

A General Linear Model (GLM) was fit to each participant's preprocessed fMRI at each time point. The interference and neutral task conditions were modeled using a boxcar convolved with a canonical hemodynamic response function (HRF). Head motion was modeled using the six realignment parameters. Framewise displacement (FD) was calculated assuming displacement across the surface of a sphere with 50 mm radius (Power et al., 2012; Siegel et al., 2014). Volumes with FD > 0.9 mm were censored by adding volumes-specific regressor to the design matrix (see Table 1 in Appendix A). We applied a high-pass filter with a cutoff of 0.006 Hz to remove low frequency noise. For each GLM, we obtained a contrast-map by subtracting the Neutral beta-map from the Interfere beta-map. These contrast maps were entered into second-level GLMs at each longitudinal time-point, using root mean FD as a regressor of no interest. We assessed how the interference effect on BOLD changed with time-point by entering data from all four waves into a longitudinal group-level model using the Sandwich Estimator Toolbox, version 2.1.0 (SwE; Guillaume et al., 2014), with root mean FD as a no-interest regressor to account for age-correlated changes to in-scanner head motion (Satterthwaite et al., 2012).

Consistent with prior literature as reported in (Kim-Spoon et al., 2021), we observed a significant interference effect on BOLD at each time point (see Fig. 1B). Our longitudinal model showed a significant linear decrease in the interference effect on BOLD in cognitive control regions identified by the MSIT. Using a cluster-defining false discovery rate (FDR) corrected threshold of $p <$ 1e-5 and a gray matter mask, the SwE derived map of time-related changes in BOLD was used to identify nine clusters of interest for an ROI analysis, including bilateral insula, bilateral middle frontal gyrus (MFG), left pre-supplementary motor area (pSMA), right pregenual anterior cingulate cortex (pACC), left inferior parietal lobule (IPL), right precuneus, and left middle occipital gyrus (see Fig. 1 C; for coordinates for peak regions within each time point, see Appendix B for Tables B1-B4). From each time point, the first eigenvariate values in the interference minus neutral contrast was obtained, after adjusting for an F-contrast of the effect of interest. Following construct validation through the longitudinal confirmatory factor analysis of brain activation during cognitive control (see Kim-Spoon et al., 2021), the "frontoparietal" latent factor scores were calculated based on left and right insula, left and right MFG, left pSMA, left IPL, and right precuneus. These seven ROIs were located in the frontoparietal network previously identified as involved in cognitive control (e.g., Dosenbach et al., 2008). Specifically, those ROIs in the fronto-parietal regions are known to be involved in attention to salience (insula), motor control (MFG and pSMA), and spatial attention and visuomotor processing (IPL and precuneus) (Sebastian et al., 2013; Spielberg et al., 2015). In a previous study, Kim-Spoon and colleagues (2021) reported that frontoparietal activation decreased with age, suggesting that lower frontoparietal activation may implicate better cognitive control. In addition, they demonstrated measurement invariance in longitudinal confirmatory factor analysis based on the multiple ROIs in the frontoparietal regions across four years, implying longitudinal reliability of ROI indicators during the MSIT task (Kim-Spoon et al., 2021; see Appendix D).

2.3.4. Family SES

Parents reported their annual income using an ordinal scale (1 = None; 2 = less than $1000; 3 = $1000 - $2999; 4 = $3000 - $4999; 5 = $5000 – $7499; 6 = $7500 - $9,999; 7 = $10,000 - $14,999; 8 = $15,000 - $19,999; 9 = $20,000 - $24,999; 10 = $25,000 - $34,999; 11 = $35,000 - $49,999; 12 = $50,000 - $74,999; 13 = $75,000 - $99,999; 14 = $100,000 - $199,000; 15 = $200,000 + a year). The median family income was between $35,000 and $49999 a year. For each family, income was estimated as the mid-point of the category parents chose. Family income was transformed from an interval variable with non-regular spacing into a continuous variable, and the approximation may obscure the nonlinear nature of the construct. Family income-to-needs ratio was calculated by dividing the family income by the federal poverty threshold for a family of that size. Participating parents reported on their and their spouse's education A parental education score was calculated as the average of parent education level. Family income-to-needs ratio and parental education were moderately correlated ($r = 0.49$, $p < .001$). A family SES index at Wave 1 was calculated by standardizing, averaging and standardizing again the scores of family income and parental education, with higher scores indicating higher family SES.

2.3.5. Parental warmth

Adolescents rated perceived positive supportive parenting separately

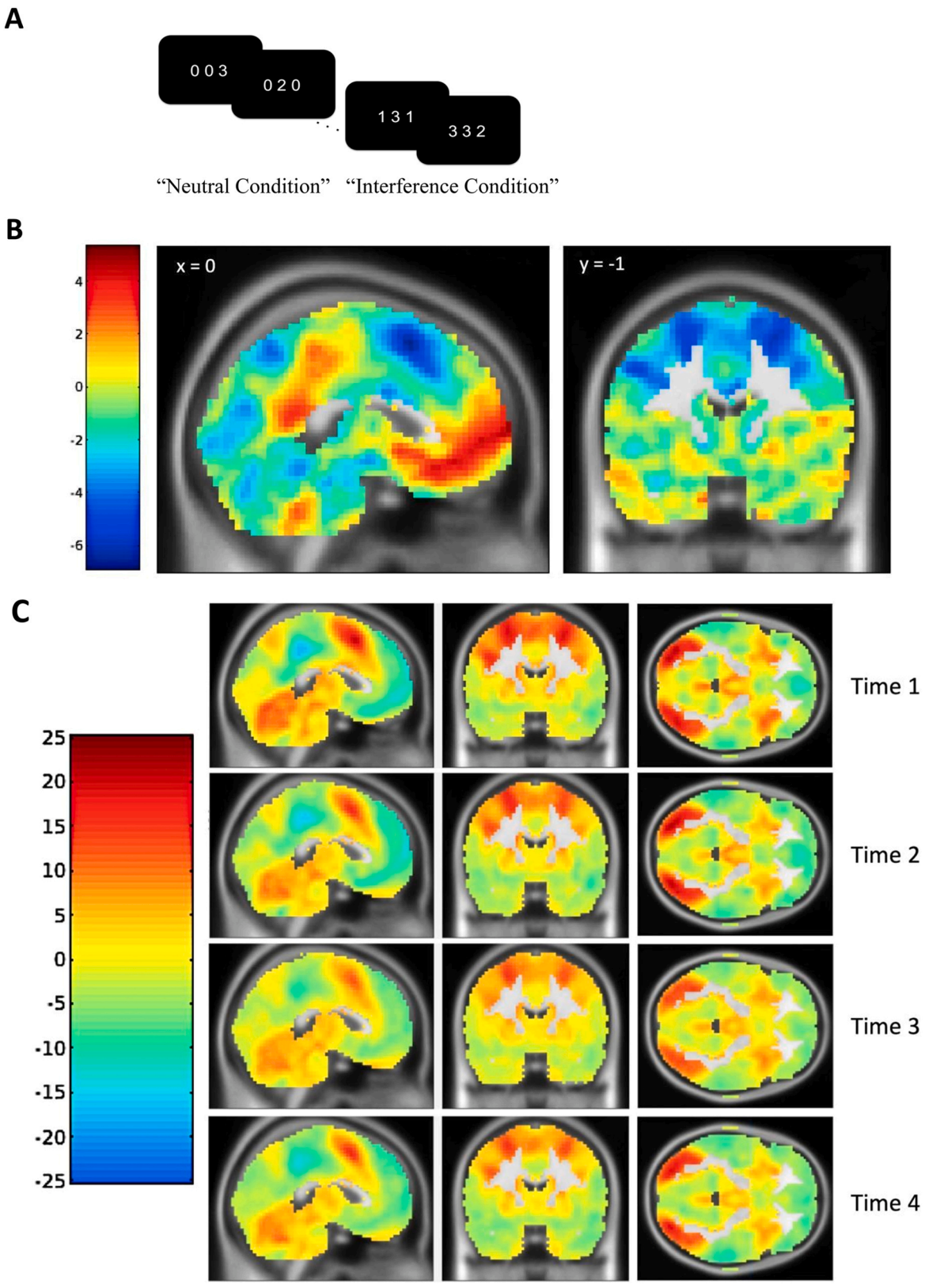

Fig. 1. Schematic Display of the Multi-Source Interference Task (MSIT) and Activation Maps Showing Significant Activation for the Interference-Neutral Contrast *Note:* A) Adolescents were instructed to identify the different digit while ignoring its position. B) Statistical *T* map showing regions of positive and negative linear change in the interference effect on BOLD responses with time point using the Sandwich Estimator Toolbox after applying a gray matter mask (unthresholded). C) Statistical *T* maps showing regions of positive (interference > neutral) and negative (neutral > interference) interference effect for each time point after applying a gray matter mask (unthresholded). Figures B) and C) are reprinted from Kim-Spoon, J., Herd, T., Brieant, A., Elder, J., Lee, J., Deater-Deckard, K., & King-Casas, B. (2021). A 4-year longitudinal neuroimaging study of cognitive control using latent growth modeling: Developmental changes and brain-behavior associations. *Neuroimage, 237,* 118134.

Table 1
Descriptive statistics and bivariate correlations of the study variables.

		1	2	3	4	5	6	7	8	9	10
1 Sex	–										
2	Family SES W1	-0.09	–								
3	Parental warmth W2	-0.01	.09	–							
4	Parental monitoring W2	.31 * *	.13	.42 * *	–						
5	MSIT RT W2	.13	-0.08	-0.18 *	-0.12	–					
6	MSIT RT W3	.22 *	-0.10	-0.07	-0.03	.63 * *	–				
7	MSIT RT W4	.18 *	-0.15	-0.06	-0.09	.47 * *	.56 * *	–			
8	MSIT FS W2	.01	-0.01	-0.03	.02	.29 * *	.13	.11	–		
9	MSIT FS W3	-0.01	.16	.05	.09	.15	.08	.05	.17	–	
10	MSIT FS W4	.01	-0.05	.06	.14	.25 * *	.12	.18	.34 * *	.27 * *	–
	M	44 % (Female)	.00	3.84	3.98	.40	.37	.34	-0.62	-0.83	-0.88
	SD		1	.63	.60	.07	.07	.06	.81	.91	.74

Note. W1 = Wave 1, W2 = Wave 2, W3 = Wave 3, W4 = Wave 4.
* $p < .05$, * * $p < .01$

for mother and father using the Inventory of Parent and Peer Attachment (IPPA; Raja, McGee, and Stanton, 1992) on a 5-point Likert scale, ranging from 1 (almost never true) to 5 (almost always true). This scale includes 12 items capturing three subscales (four items each): parent-child communication, trust and alienation. The average score of the 12 items was calculated, with higher scores indicating higher levels of perceived warmth. High internal consistency scores (Cronbach's $\alpha = 0.82 - 0.92$) were found for the total score across all four waves. Within each wave, adolescent-report maternal warmth and paternal warmth were moderately to substantially correlated (*rs* =0.43 to.56, *ps* <0.001). Therefore, a global parental warmth score was computed by averaging maternal warmth and paternal warmth at each wave. For our main analyses, we used the mother-father composite of warmth at Wave 2 ($r = 0.56$, $p < .001$), based on the recommendation that longitudinal mediation models use a temporally spaced sequence of predictor (i.e., SES), mediator (i.e., parenting), and outcome (i.e., changes in inhibitory

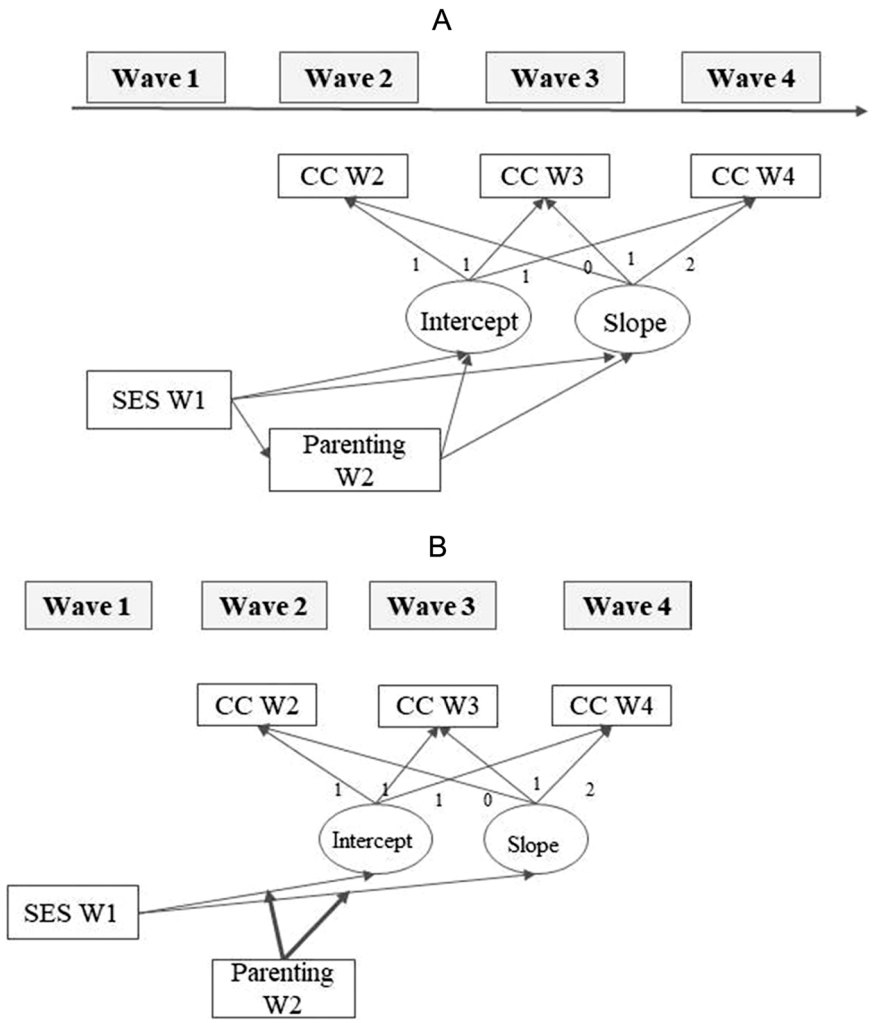

Fig. 2. A. Mediation model of parenting between SES and cognitive control development. *Note*: SES = Socioeconomic status, CC = Cognitive control. *B*. Moderation model of parenting for SES effects on cognitive development. *Note*: SES = Socioeconomic status, CC = Cognitive control.

control) whenever possible (e.g., Preacher, 2015).

2.3.6. Parental monitoring

Adolescents reported different aspects of parental monitoring using the Parental Monitoring Scale (Stattin and Kerr, 2000). This scale included 25 items, capturing subscales pertaining to parental knowledge (9 items), child disclosure (5 items), parent solicitation (5 items), and parental control (6 items). The average score of the 25 items at each wave was calculated, with higher score representing higher parental monitoring. This overall monitoring score showed good reliability across four waves (Cronbach's alpha =0.90 −0.94). For our main analyses, we used the score at Wave 2.

2.4. Data analysis plan

A latent growth modeling (LGM) approach was used via M*plus* 8.1 software package (Muthén and Muthén, 1998–, 2018) with Full Information Maximum Likelihood (FIML) estimation to account for missing data (see Appendix C). Model fit was assessed by χ^2 value, degrees of freedom, corresponding *p*-value, Root Mean Square Error of Approximation (RMSEA) and its 90 % Confidence Intervals (CI), and Confirmatory Fit Index (CFI). RMSEA values less than.08 and CFI values greater than.90 were taken to reflect acceptable fits (Little, 2013).

To test direct and indirect effects of family SES on the developmental trajectories of cognitive control via parenting behaviors, we fit conditional LGMs with family SES at Wave 1 predicting growth parameters of cognitive control (from Waves 2–4) directly, as well as indirectly through parenting behaviors at Wave 2 (see Fig. 2-A). Given that the cognitive control growth trajectories are based on only three time points with equal intervals, we used a linear growth model. We tested the significance of indirect effects by calculating bias-corrected bootstrap confidence intervals using 10,000 bootstrapping samples (Preacher and Hayes, 2008).

Next, to examine whether the association between family SES and the developmental trajectory of adolescent cognitive control is moderated by parenting behaviors, we fit conditional LGMs with family SES at Wave 1, parenting behaviors at Wave 2, and the interaction between family SES and parenting behavior as predictors (see Fig. 2-B[1]). Significant interactions were probed using simple slopes analysis, in which the association between SES and the growth parameters was estimated at low (−1 SD) and high (+1 SD) levels of the parenting behavior variable.

3. Results

Descriptive statistics and bivariate correlations among the study variables are presented in Table 1.

3.1. Mediation hypothesis

We began by testing the proposed mediation hypothesis: that the link between lower SES and slower growth in behavioral and neural indicators of cognitive control would be mediated by lower levels of parental warmth and monitoring. Results are reported separately for parental warmth and monitoring (for behavioral then neural indicators of cognitive control).

[1] The main and interaction effects of SES and parenting on the intercept and slope growth factors produced the conditional mean of the cognitive control variable (y) as a function of SES (x1) and parenting (x2): $Yt|x1, x2 = (\mu\alpha + \gamma1 \times 1 + \gamma2 \times 2 + \gamma3x1x2) + (\mu\beta + \gamma4 \times 1 + \gamma5 \times 2 + \gamma6x1x2)\ \lambda t$Where α = intercept factor, β = slope factor, μ = latent variable mean, λt = the value of time t (i.e., slope factor loading), γ = coefficient of main and interaction effect of the predictor.

3.1.1. Parental warmth

For behavioral cognitive control (MSIT reaction time), the mediation model fit the data well ($\chi^2 = 2.36$, $df = 3$, $p = .502$, CFI = 1.00, and RMSEA =0.00). As shown in Table 2, the mean of the slope factor was negative and significant, indicating significant decreases in reaction time over time. Parental warmth was negatively associated with the intercept, indicating the significant link between higher warmth and lower reaction time at Wave 2. This suggests that higher parental warmth is associated with higher cognitive control. However, there was no evidence of significant direct or indirect effects of family SES on adolescent cognitive control intercept (CI: −0.023, .000) or slope (CI: −0.002, .011) through parental warmth.

For neural cognitive control (frontoparietal activation), the mediation model fit the data well ($\chi^2 = 3.47$, $df = 3$, $p = .325$, CFI =0.97, and RMSEA =0.03). In this model, the slope had a small, nonsignificant negative residual (−0.041) that was fixed to zero. The resulting model fit was excellent ($\chi^2 = 3.92$, $df = 5$, $p = .561$, CFI = 1.00, and RMSEA =0.00). The mean of the slope factor was negative and significant, indicating significant decreases in frontoparietal activation over time, suggesting improvement in cognitive control over time. However, the link between parental warmth and frontoparietal activation intercept or slope was not significant. Thus, there was no evidence of significant direct or indirect effects of family SES on adolescent cognitive control intercept (CI: −0.160, .144) or slope (CI: −0.065, .097) through parental warmth (see Table 2).

3.1.2. Parental monitoring

For behavioral cognitive control (MSIT reaction time), the model fit the data well ($\chi^2 = 2.86$, $df = 3$, $p = .414$, CFI = 1.00, and RMSEA =0.00). However, the link between parental monitoring and reaction time intercept or slope was not significant, suggesting that there was no evidence of significant direct or indirect effects of family SES on adolescent cognitive control intercept (CI: −0.016, .006) or slope (CI: −0.004, .000) through parental monitoring (see Table 2).

For neural cognitive control (frontoparietal activation), the model fit the data well ($\chi^2 = 3.40$, $df = 3$, $p = .334$, CFI =0.98, and RMSEA =0.03). In this model, the slope had a small, nonsignificant negative residual (−0.037) that was fixed to zero. The resulting model fit was excellent ($\chi^2 = 3.75$, $df = 5$, $p = .585$, CFI = 1.00, and RMSEA =0.00).

Table 2
Indirect effects of family SES on adolescent cognitive control development via parenting behaviors.

	Parental warmth		Parental monitoring	
	b	*SE*	*b*	*SE*
Reaction Time				
Regressions				
Family SES → Parenting behavior	0.092	0.084	0.131	0.084
Parenting behavior → RT intercept	-0.012*	0.006	-0.006	0.006
Parenting behavior → RT slope	0.005	0.003	0.001	0.003
Family SES → RT intercept	-0.005	0.006	-0.005	0.006
Family SES → RT Slope	-0.003	0.003	-0.002	0.003
Means				
Intercept	.406 ***	0.006	.406 ***	0.006
Slope	-0.033***	0.003	-0.033***	0.003
Frontoparietal Activation				
Regressions				
Family SES → Parenting behavior	0.091	0.084	0.13	0.084
Parenting behavior → FPA intercept	0.006	0.070	0.066	0.071
Parenting behavior → FPA slope	0.029	0.044	0.017	0.044
Family SES → FPA intercept	0.032	0.067	0.025	0.067
Family SES → FPA Slope	-0.031	0.042	-0.034	0.042
Means				
Intercept	-0.635***	0.079	-0.639***	0.07
Slope	-0.135**	0.043	-0.134**	0.043

Note. SES = socioeconomic status, RT = reaction time, FPA = frontoparietal activation.
*$p < .05$, ** $p < .01$, *** $p < .001$.

The link between parental monitoring and frontoparietal activation intercept or slope was not significant, suggesting that there was no evidence of significant direct or indirect effects of family SES on adolescent cognitive control intercept (CI: −0.107, .219) or slope (CI: −0.067, .110) (see Table 2).

3.2. Moderation hypothesis

Next, we tested the proposed moderation hypothesis: the association between lower SES and slower growth in adolescent behavioral and neural indicators of cognitive control would be either attenuated or strengthened at higher levels of parental warmth and monitoring. Results are reported separately for parental warmth and monitoring (for behavioral then neural indicators of cognitive control).

3.2.1. Parental warmth

For behavioral cognitive control (MSIT reaction time), the moderation model fit the data well ($\chi^2 = 2.21$, $df = 4$, $p = .697$, CFI = 1.00, and RMSEA =0.00). As shown in Table 3, the mean of the slope factor was negative and significant, indicating significant decreases in reaction time over time. A significant interaction effect of family SES-by-parental warmth was found in predicting the intercept but not the slope. Significant main effects of parental warmth on the intercept of MSIT reaction time was observed. We used simple slopes in post-hoc probing of the interaction, with family SES regressed on the intercept of MSIT reaction time at various standard deviation (SD) thresholds above and below the mean of parental warmth. Results indicated that lower family SES was associated with higher MSIT reaction time at low levels of parental warmth (b = -.018, SE =.008, $p = .037$ at −1 SD). In contrast, the link between family SES and the intercept of MSIT reaction time was nonsignificant at higher levels of parental warmth (b = −0.006, SE =.006, $p = .303$ at mean; b =.006, SE =.008, $p = .447$ at +1 SD).

Regarding the neural cognitive control (frontoparietal activation), model fit of the moderation model was good ($\chi^2 = 3.95$, $df = 4$, $p = .413$, CFI = 1.00, and RMSEA =0.00). In this model, the slope had a small negative residual variance (−0.061) that was fixed to zero. The resulting model fit was excellent ($\chi^2 = 5.09$, $df = 6$, $p = .53$, CFI = 1.00, and RMSEA =0.00). As shown in Table 3, the mean of the slope factor was negative and significant, indicating significant decreases in frontoparietal activation over time. The interaction and main effects on the intercept and slope were not significant.

3.2.2. Parental monitoring

For MSIT reaction time, the models fit the data well ($\chi^2 = 3.73$, $df = 4$, $p = .444$, CFI = 1.00, and RMSEA =0.00). As shown in Table 3, the mean of the slope factor was negative and significant, indicating significant decreases in reaction time over time. A significant interaction effect of family SES-by-parental monitoring was found in predicting the slope of MSIT reaction time. We used simple slopes in post-hoc probing of the interaction, with family SES regressed on the intercept of MSIT reaction time at various standard deviation (SD) thresholds above and below the mean of parental monitoring. Results indicated that higher family SES was associated with steeper decreases of MSIT reaction time at high levels of parental monitoring (b = -.009, SE =.004, $p = .018$ at +1 SD). In contrast, the link between family SES and the slope of MSIT reaction time was not significant at lower levels of parental monitoring (b = -.001, SE =.003, $p = .646$ at mean; b =.007, SE =.004, $p = .124$ at −1 SD).

Regarding frontoparietal activation, the model fit the data well ($\chi^2 = 4.64$, $df = 4$ $p = .327$ CFI =0.97, and RMSEA =0.03). However, the slope had a small negative residual variance (−0.065) that was fixed to zero. The resulting model fit was excellent ($\chi^2 = 5.81$, $df = 6$, $p = .445$, CFI = 1.00, and RMSEA =0.00). As shown in Table 3, the mean of the slope factor was negative and significant, indicating significant decreases in frontoparietal activation over time. Significant interaction effects of family SES by parental monitoring were found in predicting both the intercept and the slope of frontoparietal activation. Simple slope analyses revealed that higher family SES was associated with higher intercept (b =0.184, SE =0.090, $p = .042$ at +1 SD) and steeper decreases in frontoparietal activation at high levels of parental monitoring (b = −0.132, SE =0.056, $p = .019$ at +1 SD). This suggests that at high levels of parental monitoring, higher SES was associated to lower initial cognitive control but faster improvement in cognitive control over time. In contrast, family SES was not significantly associated with intercept (b =.003, SE =.069, $p = .964$ at mean; b = −0.178, SE =.105, $p = .090$ at −1 SD) or slope (b = -.016, SE =.044, $p = .706$ at mean; b =.099, SE =.069, $p = .151$ at −1 SD) of frontoparietal activation at lower levels of parental monitoring.

4. Supplemental analyses

In supplemental analyses, we report in Appendix E the traditional ROI-based analyses conducted by one of the co-lead authors as part of a dissertation thesis (Li, 2020). The results were consistent with those reported in the current manuscript.

In addition, as supplemental exploratory analyses, we tested sex effects on growth trajectories of cognitive control. We tested mediation and moderation models with sex (male = 0 and female = 1) predicting intercept and slope of cognitive control for reaction time and frontoparietal activation. For the behavioral cognitive control, significant effects of sex were found for the mediation and moderation models of SES and parental monitoring and the moderation model of SES and parental warmth, indicating higher levels of cognitive control at Wave 2 for girls. In terms of the mediation and moderation effects between SES and parenting, the significant effects were consistent with the findings from the original model (without the covariate of sex). These results are reported in Appendix F.

5. Discussion

Cognitive control slowly develops through adolescence and is critical for regulating impulses and risk-taking behaviors at all stages of the

Table 3
Effect of family SES on the development of cognitive control moderated by parenting behaviors (parental warmth and parental monitoring).

	Reaction time				Frontoparietal activation			
	Intercept		Slope		Intercept		Slope	
	B	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*
SES	-0.006	.006	-0.002	.003	.028	.069	-0.027	.044
Parental warmth	-0.011*	.006	.005	.003	.012	.070	.015	.043
SES X Parental warmth	0.012 *	.006	-0.004	.003	.121	.068	-0.057	.044
SES	-0.006	.006	.001	.003	.003	.069	-0.016	.044
Parental monitoring	-0.006	.006	-0.001	.003	.052	.070	.020	.043
SES X Parental monitoring	.010	.006	-0.008**	.003	.181 **	.069	-0.115*	.045

Note. SES = socioeconomic status.
* $p < .05$, ** $p < .01$.

lifespan (Kim-Spoon et al., 2016; Nigg, 2017). The emergence and development of cognitive control in childhood has been widely studied, yet much remains unknown about environmental factors contributing to developmental changes in cognitive control across adolescence. Family SES and parenting behaviors are important contextual factors influencing individual differences in cognitive control (Farah et al., 2006; Noble et al., 2007). The current investigation attempted to clarify ways in which SES and parental warmth and monitoring work together to contribute to adolescent cognitive control development.

5.1. SES effects mediated via parenting

Utilizing a four-wave longitudinal sample of adolescents, we found no evidence for direct or indirect effects (i.e., mediation) of family SES via parental warmth or monitoring on growth in cognitive control task performance or neural activation. This null finding appears to be inconsistent with family stress theory as well as previous cross-sectional research using samples of children showing significant correlations between family SES and cognitive control (Farah et al., 2006; Noble et al., 2007), and of mediation effects via parenting on behavioral measures of executive function or effortful control (Lengua et al., 2014; Sarsour et al., 2011). Future longitudinal research should investigate whether mediating effects of parenting behaviors vary depending on the specific developmental periods in question, and the specific levels of family income (e.g., mediating effects may be more prominent in the face of chronic poverty). This null finding also could indicate that there are other key factors (e.g., neighborhood, peer, school) that were not measured or investigated in the current study, that might be functioning as mediators between SES and adolescent cognitive control. Importantly, as we discuss in the following section, our results comparing mediation versus moderation models clearly support the theorized buffering roles of parenting behaviors that promote affiliative bonding (Feldman, 2021), as opposed to parenting behaviors acting as mediating processes between environmental contexts and adolescent outcomes. For example, buffering effects have been suggested in previous neuroimaging research suggesting that a supportive caregiver can buffer against the elevated threat-related processing that is common among people who have experienced trauma (Gee et al., 2014).

5.2. SES effects moderated by parenting

Regarding our moderation hypothesis, there was some evidence to reject the null hypothesis. Specifically, a significant interaction involving parental warmth emerged but only for the behavioral (not neural) measure of cognitive control in early adolescence. Lower family SES was associated with a higher MSIT reaction time intercept (indicating poorer behavioral cognitive control performance at 14 years), but only in families with low levels of parental warmth; the SES-behavioral cognitive control link was nonsignificant at higher levels of parental warmth. This “buffering effect” is consistent with resilience theory (Zimmerman et al., 2013), highlighting that the potential risk-inducing effect of low SES can be mitigated by a supportive parenting environment. Our interpretation is that parenting behavior that is warm and supportive protects youth from negative cognitive outcomes in lower SES contexts arising from the chronic stress that is induced by lack of access to resources and insecurity in income, housing, food and other essential needs (Mullainathan and Shafir, 2013). Our data further indicated that the effect of parental warmth was prominent during early adolescence, suggesting possibly differential developmental timing of the effects of warmth with regard to cognitive control development. Parental warmth makes adolescents feel calmer when they are on their own (Steinberg, 2014), and it provides a foundation for cognitive control development, instead of shaping the developmental trajectory of cognitive control across adolescence.

We also found evidence of a significant moderating effect of parental monitoring, for behavioral as well as neural indicators of cognitive control. With regard to behavior, higher family SES was associated with greater growth in behavioral cognitive control performance across four years, suggesting an enhancing, salutary effect of parental monitoring on the benefits of access to socioeconomic resources to adolescent brain functioning. Turning to the analysis of the neural measure of cognitive control, we observed an enhancing effect of higher monitoring on the link between higher SES and greater frontoparietal activation at intercept (age 14 years), and on larger slope decreases in that activation (indicative of greater developmental improvements in the efficiency of neural processing) from 14 to 17 years. Although the enhanced (at higher levels of monitoring) positive association between higher family SES and higher frontoparietal activation at intercept seems counterintuitive (i.e., we did not expect to find higher SES to be associated with an indicator of weaker neural cognitive control), there was clear evidence of anticipated conjoint beneficial effect of higher SES and higher parental monitoring predicting stronger developmental improvements in the neural processing measure of cognitive control. Considering the pattern in which higher SES and higher parental monitoring are together related to greater activation (i.e., less efficient processing) at age 14, as well as greater developmental improvements in neural activation from age 14–17, there may be differential effects dependent on developmental timing. Prior work suggested a link between stress exposure and acceleration of the development of brain regulatory processes (e.g., Tottenham and Galván, 2016). Conversely, it is plausible that among higher SES families, higher parental monitoring allows the brain to occupy a more immature state for longer in early adolescence, but then facilitates more substantial maturational improvements as part of the developmental progression when those gains are achieved.

To our knowledge, our results also provide the first evidence of a benefit-enhancing effect of higher parental monitoring on the development of both cognitive control behaviors and their neural underpinnings at higher levels of SES. Our data revealed decreasing developmental trajectories of reaction time and frontoparietal activation across adolescence, suggesting age-related improvement in cognitive control performance and more refined and more efficient neural functioning with development (Luna et al., 2010). This result is in line with prior cross-sectional research reporting larger reaction time differences between the neutral versus the interference condition among adolescents compared to adults during the MSIT (Fitzgerald et al., 2010). Further, there was a consensus between behavioral and neural indicators of cognitive control, suggesting that the positive association between high SES and change rates of cognitive control development was most evident among adolescents with higher parental monitoring. The behavioral finding corroborates previous behavioral research providing evidence for beneficial effects of parental monitoring on adolescent self-control (e.g., Kim-Spoon et al., 2014).

Importantly, our findings regarding neural processes of cognitive control illustrate how the socio-ecological context can contribute to adolescent cognitive brain development that is known to play a key role in risky decision making and health risk behaviors during adolescence (see Kim-Spoon et al., 2017 for a review). As such, our findings clarify prior research suggesting protective effects of positive parenting behaviors on the link between lower SES in adolescence and resting-state connectivity in adulthood (Brody et al., 2019). The findings further add to a growing literature proposing that the mechanisms reflected in statistical cross-sectional and longitudinal associations may operate at various levels (e.g., family environment, larger social context, physiological level) and involve the interplay of multiple contextual factors (Blair et al., 2011; Hyde et al., 2020; Sarsour et al., 2011). It is essential to conduct analyses at multiple levels of the developing system (e.g., behavior, neural activation) to better understand the multi-faceted and complex nature of cognitive control. Future longitudinal studies measuring behavioral performance and BOLD responses using different cognitive control tasks are needed to replicate the current findings before more definitive assertions can be made regarding the mechanisms by which family SES and parenting behaviors influence the

development of adolescent cognitive control.

From a methodological viewpoint, we believe that latent variable modeling of multiple ROIs is superior to traditional individual ROI-based analyses, particularly because it improves test-retest reliability and thereby improves between-subjects inferences (see Cooper et al., 2019 for review). As prior fMRI studies have demonstrated, a single region can be involved in a broad range of tasks (Kanai and Rees, 2011) and brain regions do not function in isolation, but rather as parts of larger collections of interacting brain regions (Fox et al., 2005). Therefore, using latent factor modeling to analyze multiple ROIs that are related to a particular function during a behavioral task is a promising way to represent associations of functionally related brain regions, which resembles other functional approaches used in the field to assess brain systems and networks (e.g., Woo et al., 2017). Furthermore, in a previous study, the latent factor modeling approach allowed testing for longitudinal measurement invariance to confirm that the repeated measures fMRI data of the MSIT yielded reliable variance in intra-individual changes (Kim-Spoon et al., 2021), a critically important aspect of measurement quality in longitudinal research that is not readily addressed using traditional individual ROI-based analyses.

5.3. Limitations and future directions

Our results should be considered in light of several limitations. First, although the longitudinal data is a strength, these are still correlational data and therefore prevent us from inferring causality. Second, because nearly all of the mothers and fathers were the genetic parents of the adolescents, the family design did not allow us to test competing hypotheses regarding passive gene-environment correlation effects (i.e., that parents genetically transmit cognitive control as well as displaying covarying buffering or enhancing parenting behaviors; Bridgett et al., 2015). Third, we relied exclusively on adolescents' reports of the parenting environment, which are known to be distinct from parents' perceptions and reports—sometimes substantially so (Human et al., 2016). Fourth, youth or their parents self-identified as participants based on recruitment through schools and community settings, which could introduce bias in the results with regard to whether the results would generalize to a broader representation of adolescents (i.e., including those whose families are not inclined to volunteer to participate in research studies). Fifth, we did not examine other crucial social relationships in adolescents' lives such as best friends and peer group influences. We also did not take into account other key aspects of the home, neighborhood and school environments that may be as or more important to development of adolescent cognitive control (Holmes et al., 2019; Tomlinson et al., 2020). Finally, for head motion correction, we used both motion regression (the 6 motion regressors) and motion censoring. We did not add the *expansion* of motion regressors, as suggested by Hagler et al. (2019) based on the Adolescent Brain Cognitive Development study. The expansions do not add notable value over and above the censoring itself, as indicated by studies that directly compared alternative approaches (Siegel et al., 2014; Soares et al., 2021; Yang et al., 2019), and we would lose statistical power by adding another 6, or 18 regressors to our first level models. We suggest that future studies, especially involving a large sample of children, may consider using both the expanded motion regressors and censoring to be able to more accurately reflect actual head motion (e.g., Hagler et al., 2019).

6. Conclusions

Notwithstanding the limitations, the current investigation contributes in several key ways. First, the relatively large multiple-wave longitudinal sample and analysis of behavioral and neural indicators of cognitive control provided a unique opportunity to systematically investigate the developmental trajectories and statistical predictors of cognitive control development. Second, the current investigation is the first to test the additive and interactive statistical effects of family SES and parenting behaviors on adolescent cognitive control growth. The results provide some evidence that parenting practices play important transactional roles with SES during adolescence. Third, two different dimensions of parenting practices (warmth and monitoring) were considered in the current investigation. Though correlated with each other, these dimensions showed differential moderating effects on the association between family SES and adolescent cognitive control.

The current findings broaden understanding of how family SES and parenting behaviors together shape the development of behavioral performance and neural functioning related to cognitive control during adolescence. Our findings provide evidence of vulnerability to suboptimal cognitive control behaviors among adolescents living in impoverished environments laden with less supportive parenting and stress associated with low family SES. Our findings also provide evidence of enhanced development of neural and behavioral processes of cognitive control among adolescents living in well-resourced family environments enriched with social and cognitive stimulation inputs provided through interactions with involved parents. Such social inputs and cognitive stimulation have been argued as crucial to many forms of learning (e.g., Sheridan et al., 2017). Identifying the role of specific parenting behaviors informs prevention and intervention programs for improving adolescent cognitive control across a wide range of family SES. Furthermore, policies that aim to reduce family poverty may have greater impact if they also take into consideration the importance of enhancing resources to support parental warmth and monitoring, to optimize adolescents' behavioral and neural cognitive self-regulation.

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.

Acknowledgements

This work was supported by grants from the National Institute on Drug Abuse (R01 DA036017) to Jungmeen Kim-Spoon and Brooks King-Casas. We thank the former and current JK Lifespan Development Lab members for their help with data collection. We are grateful to the adolescents and parents who participated in our study.

Appendices A–F. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.dcn.2022.101139.

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