DEVELOPMENT OF NEURAL AND BEHAVIORAL INHIBITORY CONTROL DURING ADOLESCENCE: THE INTEGRATIVE EFFECTS OF FAMILY SOCIOECONOMIC STATUS AND PARENTING BEHAVIORS

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DEVELOPMENT OF NEURAL AND BEHAVIORAL INHIBITORY CONTROL
DURING ADOLESCENCE: THE INTEGRATIVE EFFECTS OF FAMILY
SOCIOECONOMIC STATUS AND PARENTING BEHAVIORS

A Dissertation Presented

by

MENGJIAO LI

Submitted to the Graduate School of the
University of Massachusetts Amherst in partial fulfillment
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DEVELOPMENT OF NEURAL AND BEHAVIORAL INHIBITORY CONTROL 
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ABSTRACT

DEVELOPMENT OF NEURAL AND BEHAVIORAL INHIBITORY CONTROL DURING ADOLESCENCE: THE INTEGRATIVE EFFECTS OF FAMILY SOCIOECONOMIC STATUS AND PARENTING BEHAVIORS

FEBRUARY 2020

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Inhibitory control (IC) has drawn great attention from researchers and practitioners and the concurrent association between family socioeconomic status and IC in adolescence is well-documented. However, little is known about whether and how family socioeconomic status influence the individual differences in the development of adolescent IC. The current investigation aimed to address this gap in knowledge by employing two multiple-wave longitudinal studies of IC. In the early adolescent sample (N = 311), color-word Stroop task performance was assessed as a measure of IC when individuals were 10 and 13 years old. In the middle adolescent sample (N = 167), multi-source interference task performance and corresponding neural activities were assessed as measures of IC, annually for four years from 14 to 17 years of age. Family socioeconomic status and three dimensions of parenting behaviors were measured through informant-rating surveys in both studies. In both samples, the longitudinal development of IC was examined first. Next, the direct and indirect effects of earlier
family socioeconomic status on the development of IC via different parenting behaviors was studied. Also, the independent and interactive associations between family socioeconomic status and several parenting behaviors in the prediction of adolescent IC were examined. Results across these two studies revealed that IC continued to improve through adolescence. In the early adolescent sample study, family socioeconomic status showed significant indirect effects on behavioral IC via its influences on parenting behaviors--especially parental warmth and parental negativity. In the middle adolescent sample study, the interactive effects of family socioeconomic status and parenting behaviors were significant in predicting neural functioning related to IC. The association between family socioeconomic status and the intercept of neural correlates of IC was significant only among families with low parental warmth, high parental rejection and low parental monitoring. The current investigation extended the prior literature in systematically testing the longitudinal associations between family socioeconomic status, parenting behaviors and the development of behavioral and neural functioning of IC during adolescence. Implications of the current study for prevention and intervention were also discussed.
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<td>BOLD</td>
<td>Blood-oxygen-level dependent</td>
</tr>
<tr>
<td>dACC</td>
<td>Dorsal anterior cingulate cortex</td>
</tr>
<tr>
<td>DLPFC</td>
<td>Dorsolateral prefrontal cortex</td>
</tr>
<tr>
<td>EF</td>
<td>Executive function</td>
</tr>
<tr>
<td>fMRI</td>
<td>functional magnetic resonance imaging</td>
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<tr>
<td>IC</td>
<td>Inhibitory control</td>
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<tr>
<td>MSIT</td>
<td>Multi-source interference task</td>
</tr>
<tr>
<td>SES</td>
<td>Socioeconomic status</td>
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<tr>
<td>SMA</td>
<td>Supplementary motor area</td>
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<tr>
<td>VLPFC</td>
<td>Ventrolateral prefrontal cortex</td>
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CHAPTER I

GENERAL OVERVIEW

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). Heightened neuroplasticity also supports accelerated growth in academic, physical, and social capabilities. 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 (Braams, van Duijvenvoorde, Peper, & Crone, 2015; Duell et al., 2018; Steinberg, 2008) and greater risk for psychopathology, such as depression, anxiety and delinquent behaviors (Conger, Ge, Elder, Lorenz, & Simons, 1994; Dishion & Patterson, 2016; Moffitt, 2018). During adolescence, individuals begin to experiment with substances such as alcohol, tobacco and marijuana, which can have adverse health, social and developmental outcomes (Squeglia & Gary, 2016; Volkow et al., 2016). For example, longitudinal studies indicate that individuals who are exposed to drugs such as cannabis during adolescence are more likely to experience neurocognitive decline in IQ (Meier et al., 2012), and lower rate of high-school graduation (Castellanos-Ryan et al., 2017). Neuroimaging studies have identified that moderate-to-high alcohol use in adolescence may impede the structural and functional development of the brain systems that are related to complex social and cognitive functions (Meruelo, Castro, Cota, & Tapert, 2017; Spear, 2018). Teenagers also initiate sexual intercourse and sometimes unprotected sexual behaviors, which may result
in unintended pregnancies and dramatically change adolescents’ life course (Ellis et al., 2003; Meinzer et al., 2017). Negative experience during this phase not only shapes an individual’s health, social and career development along the life course, but also influences the overall wellbeing of an entire society.

A number of cognitive and neurobiological models have suggested that inhibitory control (IC), the ability to suppress impulses in favor of long-term goals and contextual requirements, is critical in decision making and promoting healthy behaviors (Casey, Tottenham, Liston, & Durston, 2005; Reyna & Farley, 2006). Empirical research has associated IC with a variety of risk-taking behaviors in children and adolescents, such that adolescents with better IC tend to make less risky decisions (Kim-Spoon et al., 2016; Lahat et al., 2012; Nigg, 2017; Steinberg, 2008). IC has therefore drawn great research attention and an accumulating number of studies have revealed that IC develops dramatically during early childhood (Garon, Bryson, & Smith, 2008; McClelland & Cameron, 2012; Montroy, Bowles, Skibbe, McClelland, & Morrison, 2016). However, relatively little is known about whether and how IC develops through adolescence. Cross-sectional studies suggest that regulatory capacities are still developing during adolescence and do not reach adult levels until early adulthood (Liu, Angstadt, Taylor, & Fitzgerald, 2016; Luna, Garver, Urban, Lazar, & Sweeney, 2004; Steinberg et al. 2008). However, longitudinal studies investigating the developmental changes of IC from late childhood to adolescence are rare and there is no systematic attempt in the published literature to examine the mechanisms that support or hinder the development of IC during this period (Luna, Marek, Larsen, Tervo-Clemmens, & Chahal, 2015; Zanolie & Crone, 2018).

Given the impacts of adolescence experience on later life, it is critical to understand the
individual differences in normative development of brain regions and behaviors that are related to IC, as well as the contextual factors that may influence their development.

The current investigation employed two longitudinal study samples to measure and depict developmental changes in adolescent IC and address the mechanisms through which important social contextual factors, such as family socioeconomic status (SES) and parenting behaviors, influence IC development during adolescence. The two study samples were distinct, with each focusing on a specific age group and a specific set of research questions and methods. The first study sample was a group of 311 youths recruited from Durham, North Carolina and followed multiple times during the transition from late childhood to early adolescence (from 9 to 13 years of age). Development of behavioral IC during this transition was examined using computer tasks and questionnaires. The second study sample was a group of 167 adolescents recruited from Southwest Virginia and assessed four times on behavioral performance and neural correlates of IC during middle adolescence (from 14 to 17 years of age). Growth trajectories of behavioral IC and neural correlates of IC were examined using computer tasks and brain imaging. In both studies’ samples, we investigated whether the effects of earlier family SES on adolescent IC development would be moderated or mediated by different parenting behaviors. In addition to the common research questions mentioned above, we were also able to test specific research hypotheses in each sample depending on the research designs. These two samples were recruited from similar geographical area and matched on many of the key variables’ measures, which reduce the influence of these factors on the results. Furthermore, utilizing two longitudinal samples allows us to
examine the development of IC on a much wider age range and to test a broader range of research questions.

A heuristic model is presented in Figure 1 to provide a framework for guiding the literature review and statistical modeling in the current dissertation. Different paths in this model indicate the specific links and mechanisms investigated among main study variables. In the following sections, the main study variables and measurements will be introduced, theory for each part of the model will be reviewed, and empirical cross-sectional and longitudinal studies of that link in childhood and adolescence will be summarized.

Elucidating the underlying mechanisms of family SES, parenting and IC development also has practical implications on intervention and prevention. Adolescence is a critical period when youths show heightened risk taking behaviors and start the experimentation of substance use. Promoting healthy behaviors and protecting young people from health risks during adolescence has been a major focus of public policy and intervention (World Health Organization, 2018). Within the developmental literature, deficits in EF (including IC) has been theorized to contribute to risk taking as a result of poor cognitive and behavioral regulation (Giancola & Mezzich, 2003; Kim-Spoon et al., 2017; Lahat et al., 2012). A variety of training and intervention programs have been developed to improve child and adolescent EF. However, the results are rather mixed regarding to the effectiveness of EF trainings direct to children. A few studies found significant training effects in young children with poor inhibitory skills (Dowsett & Livesey, 2000; Röthlisberger, Neuenschwander, Cimeli, Michel, & Roebers, 2011), however, studies of typically developing children failed to find any improvement in IC
nevertheless, family-based training program engaging larger contexts of parenting and the home environment have been found to be effective to support EF development in young children (Neville et al., 2013). Therefore, understanding the mechanism underlying family SES and IC development may help refine and improve prevention and intervention efforts by targeting more specific contextual and neurobehavioral buffers.

**Defining Inhibitory Control**

IC is one component of executive function (EF), which is an umbrella term of several dissociable subcomponents: working memory (i.e. the ability to hold and manipulate information in mind), inhibition (i.e. the ability to inhibit a prepotent response for the purpose of a contextual-appropriate response) and set shifting (i.e. the capacity to flexibly update the mindset) (Miyake et al., 2000). The three major components of EF work together to support goal-directed behavior, but each of them characterizes specific processes and may have distinct profiles of cognitive development (Luna et al., 2004). EF shows protracted and gradual development through childhood and is essential for mental health, academic achievement, and individual wellbeing (Giancola & Mezzich, 2003; Luna, Padmanabhan, & O’Hearn, 2010; Schmeichel & Tang, 2015).

IC enables top-down, goal-driven, and voluntary control of one’s attention, behavior, and thoughts. It allows individuals to suppress automatic prepotent responses to achieve long-term goals and fulfill contextual requirements (Miyake et al., 2000). IC represents a family of functionally similar inhibitory process and has been used interchangeably with other terms, such as self-control or response inhibition (which refers
to inhibiting prepotent response), interference control (which refers to control of attention) or cognitive inhibition (which refers to suppressing irrelevant information) (Diamond, 2013; Nigg, 2000). Behavioral studies have found that behavioral inhibition and suppression of interference are strongly correlated and fall into a single latent factor (Friedman & Miyake, 2004). Neurobiological evidence also suggests that these inhibition-related functions share similar neural bases and involve overlapping brain regions, such as right inferior frontal gyrus, supplementary motor area (SMA) and pre-SMA (Aron, Behrens, Smith, Frank, & Poldrack, 2007; Bartoli, Aron, & Tandon, 2018; Crone & Steinbeis, 2017; Zheng, Geng, & Lee, 2017).

IC is a prominent component of EF and has drawn great research attention given its profound impact in cognitive, behavioral and social development (Diamond, 1990). During early childhood, the development of IC sets stage for school readiness, academic success, and social competency, and forms a critical foundation for higher cognitive processes developed later in life (Allan, Hume, Allan, Farrington, & Lonigan, 2014; Blair & Razza, 2007; Duncan et al., 2007; Moffitt et al., 2011; Skibbe, Montroy, Bowles, & Morrison, 2019). For adolescents, IC plays a key role in minimizing the risk of developing negative outcomes (Braams et al., 2015), such as peer victimization (Fahie & Symons, 2003; Oberle & Schonert-Reichl, 2013; Holmes, Kim-Spoon, & Deater-Deckard, 2016) and risk-taking behaviors (Kim-Spoon et al., 2016; Lahat et al., 2012; Luna et al., 2010).

**Measuring Inhibitory Control**

There is considerable variability in the methods used to measure IC. Many behavioral tasks and questionnaires have been developed to measure IC at different age
groups. A detailed discussion of age-appropriate IC tasks for young children was reviewed by Garon and colleagues (2008). This review differentiated between simple IC tasks and complex IC tasks, with the former focusing on withholding a proponent response, whereas the latter requiring individuals to hold a rule in mind, inhibit a proponent response and respond to a conflict option. IC tasks used for adolescents and adults are typically complex IC tasks and require individuals to inhibit a prepotent response and generate an incompatible response. Common IC tasks with adolescents and adults include the go/no-go (Roberts & Pennington, 1996), flanker (Rueda, Posner, & Rothbart, 2005), stop signal (Logan, 1994), antisaccade (Hallett, 1978), Stroop (Stroop, 1935), and multi-source interference tasks (MSIT, Bush, Shin, Holmes, Rosen & Vogt, 2003). Though these tasks present different types of cognitive conflicts, they all require detecting the conflicts, inhibiting dominant responses, and activating sub-dominant responses. For example, in the classic color-word Stroop task, participants are presented with a series of words printed in a color of “ink” that is different from their semantic meaning (e.g. the word “red” printed in blue ink). Participants must inhibit the prepotent response (i.e., reporting the semantic meaning of the word) and initiate a subdominant response which is interfering (i.e. naming the color of the ink). In most of the performance-based tasks, accuracy (or error rate) and reaction time are the two most common indicators used to measure the efficiency of IC, with higher accuracy (or less error) and lower reaction time suggesting greater ability to inhibit interference.

A handful of questionnaires and rating scales also have been developed to measure IC, especially for young children; this is because standard IC tasks are too difficult for them to complete, and informant ratings of children’s IC behaviors can
provide more ecologically valid measurement of IC compared to brief tasks. One commonly used questionnaire is the IC subscale in the Child Behavior Questionnaire (Rothbart et al., 2001), which captures the ability to exercise control over inappropriate approach responses under instruction or in novel situations. For adolescents and adults, the Brief Rating Inventory of Executive Function (Gioia, Isquith, Guy, & Kenworthy, 2000) is a commonly used rating scale to measure IC and EF. Impulsivity ratings also have been used as an indicator of poorer IC (Enticott et al., 2006; Shuster & Toplak, 2009). However, empirical studies have often found a modest association between performance-based and rating-scale/questionnaire measures of EF (including IC) (Blair, 2003; Toplak, West, & Stanovich, 2013). Researchers have proposed that performance-based measures and questionnaire measures may capture slightly different aspects of cognitive abilities, with the former assessing processing efficiency when the goals are explicitly presented and the latter indicating the success of goal pursuits (Toplak et al., 2013). In the current investigation, we used performance-based IC measured by two common behavioral tasks in order to reduce the risk of subjective interpretation on adolescent behavior.

**Development of Inhibitory Control across Childhood and Adolescence**

As shown in the conceptual model (Figure 1), investigating the development of IC is the first goal of the current investigation. A large body of cross-sectional and longitudinal research has investigated the emergence and early development of IC. It is widely acknowledged that the early form of the inhibitory process emerges at the end of the first year when infants are able to inhibit neonatal reflexes and prepotent reaching response following a caregiver’s requests (Diamond, 1990). Psychophysiological studies
also have provided evidence of early IC by showing that 10-month-olds recruit distinct frontal resources during tasks that require inhibitory process (Cuevas, Swingler, Bell, Marcovitch, & Calkins, 2012). Voluntary inhibitory control begins to emerge in the second year of life when toddlers become able to comply with caregiver instructions and to independently control their behaviors under external supervision (Carlson, 2005; Kochanska, Murray, & Harlan, 2000). From toddlerhood to the preschool period, IC undergoes rapid development, which coincides with the maturation and strengthened connectivity in the frontal lobe (Rothbart & Posner, 2001; Moriguchi, & Hiraki, 2013). Better inhibitory capabilities allow young children to inhibit their behaviors for a longer period of time and inhibit both automatic responses and responses with an external reinforcer (Garon et al., 2008). During middle to late childhood, performance on some relatively simple IC tasks develops rapidly and reaches a plateau phase (Carlson & Moses, 2001) whereas behavioral performance of complex IC tasks continues to improve (Simonds, Kieras, Rueda, & Rothbart, 2007; Troller-Renfree et al., 2019).

Even though the early development of IC has been well-researched, a systematic investigation of how IC develops through adolescence is lacking. Adolescence is an important period when dramatic brain changes in brain structure and functioning occur as a result of pubertal maturation and rapid physical growth (Blakemore & Choudhury, 2006; Fuhrmann, Knoll, & Blakemore, 2015). Neuroimaging studies revealed that cerebral white matter increases linearly and grey matter declines in both volume and thickness in frontal, parietal and temporal cortices through adolescence (Crone, van Duijvenvoorde, & Peper, 2016; Giorgio et al., 2010). Moreover, brain regions that are closely related to cognitive control, including the lateral prefrontal cortex, parietal cortex
and medial prefrontal cortex, showed different magnitude of activation compared to children and adults (Luna et al. 2010; Lei et al., 2015; Velanova, Wheeler, & Luna, 2008). However, empirical research on how IC develop throughout adolescence is rather limited.

To date, most of the studies on adolescents have adopted cross-sectional designs and have yielded mixed results. Some studies have suggested that behavioral performance of IC capacity continues to improve well into adolescence (Liu et al., 2016; Luna et al., 2004; Steinberg et al. 2008). However, other studies have claimed that children’s performance on some of the inhibition tasks reach adult levels by late childhood or early adolescence (Carlson & Moses, 2001; Welsh et al., 2006). Researchers have posited that the age at which IC reaches adult-level performance largely depends on the task complexity. Performance on simple control of prepotent responses develops rapidly and reaches adult levels in late childhood. However, for IC tasks that require more complex inhibitory processes, performance continues developing through childhood and adolescence (Garon et al., 2008).

Cross-sectional studies on complex IC tasks have provided initial, preliminary evidence that IC shows steady development in the transition from childhood to adolescence. Leon-Carrion and colleagues (2004) tested performance on the classic color-word Stroop task in a cross-sectional sample of children and adolescents aged 6 to 17 years. In this task, participants were presented with a fixed order of three conditions: single word displayed in black color, color rectangle, and incongruent color-word. Participants were asked to name the single words, the color of the rectangle, or the printed color of a word while ignoring its meaning in each condition. Interference error
and interference reaction time were computed as the difference in both response accuracy and time between naming color rectangle and naming incongruent word. Results suggested that both interference errors and interference times decreased linearly from age 6 to 17, suggesting that IC developed gradually through adolescence. Another cross-sectional study examined the effects of age on behavioral performance and brain activation during the MSIT task in a sample of 8- to 19-year-old youths (Liu et al., 2016). In this task, participants were asked to identify the number that differs from the other two by pressing a button corresponding to the unique number. Results revealed significant age effects on task performance, with increasing age being associated with greater accuracy and faster response times. Similar improvements in IC reaction time and accuracy were observed in studies using other IC tasks (Best & Miller, 2010; Duell et al., 2018; Huizinga, Dolan, & van der Molen, 2006; Luna et al., 2004).

However, there have been a few exceptions. For example, one cross-sectional study compared the color-word Stroop task performance across youths (ages 7 - 13) and adults (ages 19 - 29), and found age differences in the Stroop interference effects were only observed for response time, not for accuracy (Schroeter, Zysset, Wahl, & von Cramon, 2004). In another study, no significant differences were found for either accuracy or response time in the color-word Stroop task when comparing adolescents (ages 14 - 17) to adults (ages 18 - 25) (Andrews-Hanna et al., 2011). Differences in samples’ age ranges, variations in IC tasks, and differences in how accuracy and reaction time were measured all may have contributed to the discrepancies in these findings.

Though cross-sectional behavioral studies on IC have been fruitful, they are limited in their ability to distinguish cohort effects from true developmental changes and
they cannot test the developmental shape and rate of IC change and individual differences in developmental trajectories over time. These are problems that can be addressed with longitudinal designs. First, longitudinal studies can help replicate the cross-sectional findings and demonstrate true developmental changes of behavioral IC through the transition from late childhood to adolescence and across adolescence. Second, it will be beneficial to identify predictors of developmental changes in behavioral and neural correlates of IC in order to better understand the individual differences of IC development across this age period; this can only be done with a longitudinal design.

Turning to longitudinal study designs, there have been only a handful of studies in adolescence, and the results generally support the cross-sectional literature showing age differences in IC. Ordaz and colleagues (2013) examined performance on an antisaccade task in a longitudinal sample of 9 – 26 years old. In this task, a visual cue was presented either on the left or right side of a screen, and participants were instructed to inhibit the saccade toward the stimulus and to look instead to the opposite direction. Corrected error rate (i.e. mistakenly look at the stimuli followed by a saccade to the correct location) and reaction times on corrected trials were measured to indicate IC. Longitudinal growth modeling examined the linear and quadratic growth pattern of corrected error rate and response time on corrected trials. Results revealed that both the corrected error rate and response times on corrected trials decreased significantly with age, suggesting that adolescent IC performance improved with age. Moreover, the rate of improvement decelerated with age yet persisted into early adulthood. Significant variability was only observed at the intercept of corrected error rate, not the slope of corrected error rate or the developmental parameters of response times on correct trials. Results suggested that
individuals show similar growth trajectories of the corrected error rate and the response times on corrected trials across adolescence. Boelema et al. (2014) investigated the developmental changes of IC in a large sample of adolescents from early adolescence to late adolescence. In this study, IC was measured twice using a subtask of Amsterdam Neuropsychological Tasks at age 11 and then at age 19. Participants were presented with a square jumping randomly left/right on a horizontal bar. In the compatible condition, a series of ten squares were shown and one of them was green. If the green square jumps left, the participant has to press the left mouse button and the right mouse button if it jumps right. In the incompatible condition, a series of ten squares are shown and one of them is red. If the red square jumps left, the participant has to press the right mouse button and the left button if it jumps right. Reaction time differences between the compatible and incompatible conditions were used to indicate inhibition capacity. Results revealed significant decreases in reaction time over time, which indicated salient improvement in inhibition capacity from early adolescence to late adolescence. In this study, IC was only measured twice (at age 11 and 19), therefore the shape of the developmental trajectories of IC and the individual differences in the developmental trajectories of IC could not be estimated. It is also worth noting that both the antisaccade task and the subtask in Amsterdam Neuropsychological Tasks measure a relatively simple form of inhibition, which is the ability to inhibition of prepotent responses (Garon et al., 2008). As described earlier, adolescence is a key developmental period of complex IC. Thus, it is important for longitudinal studies to use complex IC tasks, such as Stroop task and MSIT task used in the current study, to investigate the developmental trajectories of complex IC capacity, which relies on cognitive interference and requires
more mental processing, such as holding a rule in mind and responding to a conflict option.

**Socioeconomic Disparities in Inhibitory Control Development**

The protracted development of IC across childhood and adolescence has been revealed as sensitive to environmental influences and may allow for adjustment to contextual demands. The evidence for, and implications of, environmental inputs are twofold. First, IC task performance, as well as its underlying neural processes, could be improved through cognitive training programs or school curriculum, and set the foundation for better academic and behavior performance in the future (Diamond & Lee, 2011; Karbach & Unger, 2014; Raver et al., 2011). Second, IC development is particularly vulnerable to disadvantageous environmental influences. Evidence is accumulating that adverse environmental factors, such as family SES and negative parenting, can impede IC performance not only in early childhood but also in adolescence, which is another critical period of changes in brain development (Blair & Raver, 2012; Hackman & Farah, 2009; Ursache & Noble, 2016).

The second goal of the current study focuses on the association between family socioeconomic status (SES) and the developmental changes of IC (Figure 1, path ‘a’). Extant research has identified family SES as an important contextual factor to influence early IC development (Blair et al., 2011; Hughes, Ensor, Wilson, & Graham, 2010; Rochette & Bernier, 2014; Obradović, Portilla, & Ballard, 2016; Ruberry et al., 2016). Family SES represents family’s access to social and economic resources spanning education, income, and social prestige (McLoyd, 1998). Low family SES have long been recognized as sources of variance in individual differences in cognitive development and
academic performance in children and adolescents (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005; Kim et al., 2013; Lawson, Hook, & Farah, 2018; Noble et al., 2015; Sarsour et al., 2011; Ursache & Noble, 2016). Theories emphasize that low family SES is related to limited resources and increased levels of stress, which produce greater cognitive load on everyone in the household and result in impaired IC and related EF skills. Farah and colleagues (2006) investigated how childhood poverty influenced five main neurocognitive systems (i.e., language, EF, spatial cognition, memory and visual cognition system). They found that poverty is associated with these neurocognitive systems differentially, with disproportionally stronger influence on language and EF development (including IC). Specifically, in two different studies they found significant differences in IC performance between low and middle SES kindergarteners (Noble, Norman, & Farah, 2005) and 11-year-olds (Noble, McCandliss, & Farah, 2007).

Though the concurrent association between family SES and level of IC has been reported, much less is known about the association between family SES and developmental changes in IC. A few longitudinal studies of preschoolers investigated the association between SES and developmental changes of EF, including IC, and yielded mixed results. While Moilanen and colleagues (2010) found that extreme family poverty was association with slower growth of IC from 2 to 4 years old, whereas Hughes and colleagues (2010) revealed that early family income predicted only the intercept of EF (including IC) at age 4, but not the developmental changes of EF from age 4 to 6.

To date, only two longitudinal studies has examined the association between SES and IC development across adolescence. In a two-wave longitudinal study of adolescents, Spielberg et al. (2015) examined the impact of SES on the maturation of IC and brain
function. The results showed that higher family SES was associated with increases in behavioral inhibition and lower family SES was associated with decreased behavioral inhibition accuracy measured by a Go/No-go task from age 11 to 13. This study provides initial evidence that family SES plays a critical role in the development of IC during adolescence. Boelema et al., (2014) explore the effects of family SES on the developmental changes of IC during adolescence, and the results revealed that comparing to middle and low family SES groups, high family SES group had the smallest intercept (of reaction time differences between compatible and incompatible conditions) at age 11 and the largest developmental changes from age 11 to age 19, indicating a better performance at baseline and more maturation of IC performance from early adolescence to late adolescence.

**Parenting Behaviors and Adolescent’s Inhibitory Control**

Another important contextual factor that has been linked to IC development is parenting behaviors (Figure 1, path ‘c’; Blair et al., 2011; Hughes et al., 2010; Rochette & Bernier, 2014). Parenting practices can be broadly categorized on several dimensions: parental warmth, parental rejection and parental monitoring. Though moderately correlated, these parenting dimensions are said to distinctly contribute to general cognitive ability and inhibition processes in offspring (Blair et al., 2011; Rhoades, Greenberg, Lanza & Blair, 2011). A moderate association between parental warmth and better EF performance (including better IC performance) has been repeatedly detected in young children (Blair et al., 2011; Karreman, van Tuijl, van Aken, & Deković, 2006; Meuwissen & Carlson, 2015; Rhoades et al., 2011; Rochette & Bernier, 2014). Meanwhile, maternal discipline, inconsistency and negative controlling behaviors were
negatively linked to IC development in young children (Moilanen et al., 2010; Roskam, Stievenart, Meunier, & Noël, 2014). It has been suggested that low SES families are often characterized by limited access to social and economic resources, which are also associated with less warm and supportive, harsher and more controlling parenting behaviors. These parenting behaviors may contribute to heightened stress for adolescents, and act as a proximal contextual factors of IC development.

The above studies provide preliminary supports for the link between parenting behaviors and IC in young children (Morris, Silk, Steinberg, Myers, & Robinson, 2007; Valcan, Davis, & Pino-Pasternak, 2018), however, these relationships have yet to be systematically examined in late childhood or adolescence, and have yet to be examined with the consideration of the effects of other important family contexts, such as family SES. Next, the current evidence about the association between various parenting behaviors and adolescent IC is reviewed. However, it is worth noticing that in many of the studies cited, adolescent IC was mostly measured through self or parent ratings, rather than cognitive task performance measures. More objective measures, such as performance-based IC tasks, are needed to confirm the magnitude of the association between adolescent IC and various dimensions of parenting behaviors.

**Parental Warmth**

Parental warmth represents supportive, sensitive and consistent responsiveness to children’s behaviors (Landry et al., 2006). It has been repeatedly found to promote general cognitive development in young children (for a review, see Fay-Stammbach, Hawes, & Meredith, 2014). This could be due to that parents who are warm and supportive are more likely to establish appropriate rules and provide consistent feedback
to guide child’s behavior. Children growing up in such a sensitive and supportive environment are more likely to understand these rules and internalize the external standards to monitor their own behaviors and develop strategies to guide those behaviors (Hughes & Ensor, 2009; Roskam et al., 2014).

By comparison to the childhood literature, there are few studies on parental warmth and adolescent IC, and results are mixed. In a small sample of 7- to 16-year-olds who had been exposed to traumatic family environments (e.g., domestic violence), positive maternal parenting as perceived by youth was associated with their “planning” performance (measured by the Tower of London task), but was not associated with IC using the Stroop task (Samuelson, Krueger, & Wilson, 2012). However, adolescent studies that measured IC using questionnaires have generally reported positive correlations between warm parenting practices and adolescent IC (Finkenauer, Engels, & Baumeister, 2005; Moilanen, 2007).

Turning to longitudinal evidence, one study found that warm and expressive parenting behaviors were related to longitudinal change in children’s self-control when children transitioned from childhood to adolescence (from 7-12 years old to 9–14 years old) but this pattern was not repeated when children were followed-up again two years later at age 11–16 years (Eisenberg et al., 2005). These results were echoed by another longitudinal study which revealed that greater paternal positive parenting predicted better child effortful control (including self-reported IC) during the transition from late childhood to adolescence (from ages 7 to 11–12 years) (Tiberio et al., 2016).
**Parental Rejection**

Rejecting parenting encompasses harsh punishment, rejection, and intrusive discipline toward the children (Rohner, 2005). It has been consistently associated with negative outcomes, such as poor cognitive ability, emotional dysregulation and behavioral problems in children (Blair et al., 2011; Cuevas et al., 2014; Meuwissen & Carlson, 2015). A recent meta-analysis examined the strength of the association between parenting and child EF (including IC) and found moderate association between parental rejection and IC in children aged 0 to 8 years, with higher rejecting parenting associated with lower IC (Valcan et al., 2018). This could occur via the elevated family stress. Harsh punishment and rejection from parents increase stress in the family environment, which elicits anxiety and stress in the child and does not provide appropriate scaffolding for the development of IC capacity. In addition, parent’s inconsistent discipline makes it difficult for children to learn to guide their behaviors and internalize them to response to challenges (Hughes & Ensor, 2011; Roskam et al., 2014).

Prior research on the association between rejecting parenting and IC during adolescence is limited. A recent cross-sectional study of adolescents found that high levels of parental inconsistent discipline are associated with lower EF performance (including IC) in a group of 11-14-year-olds (Sosic-Vasic et al., 2017). This finding is also found in non-Western adolescent sample (Fatima, Sheikh, & Ardila, 2016). Longitudinal evidence about rejecting parenting and IC development has been reported in studies using informant ratings of IC. Two-wave longitudinal studies indicated that higher levels of parental rejection predicted decreases in informant-reported effortful control/self-regulation in early adolescence (Moilanen, Rasmussen, & Padilla-Walker,
Another longitudinal study examined the trajectory of mother- and self-rated IC in children transitioning from late childhood to adolescence. It also investigated whether and to what extent the growth trajectory of IC was linked to parenting behaviors. Results revealed that rejecting parenting (e.g., harshness, inconsistency and physical discipline) was related to individual differences in initial level of effortful control, but not the trajectory of effortful control over time, suggesting that children growing up in high parental hostile environment showed lower levels of self-regulation in childhood (King, Lengua, & Monahan, 2013).

**Parental Monitoring**

Parental monitoring encompasses knowledge about children’s whereabouts and supervision of children’s activities and surroundings (e.g. friends, after-school activities) (Dishion & McMahon, 1998). Higher monitoring may allow parents to supervise children’s behaviors and provide guidance for the emergence and improvement of IC. It may also minimize risk factors that may deteriorate the neurocognitive functioning development. Therefore, parental monitoring has been linked to a variety of child outcomes, with some of them are highly correlated with IC. For instance, high parental monitoring was associated with lower alcohol and marijuana use among adolescents (Dever et al., 2012; Rusby, Light, Crowley, & Westling, 2018), lower levels of antisocial behaviors (Crocetti et al., 2016; Wertz et al., 2017) and less risky sexual behaviors (DelPriore, Schlomer, & Ellis, 2017; Parkes, Henderson, Wight, & Nixon, 2011).

As for IC capacity and closely related EF and self-regulation skills, the results from prior studies are less consistent. In a cross-sectional study involving adolescents between 11 and 17 years of age, parental monitoring perceived by adolescents was not
predictive of adolescents’ self-reported regulatory abilities (Moilanen, 2007). However, Bowers and colleagues (2011) investigated and identified four classes of developmental trajectories of cognitive and behavioral regulation in pursuit of goals (adolescents’ self-rating on the Selection, Optimization, and Compensation questionnaire, Freund & Baltes, 2002), longitudinally from the fifth to eleventh grades. A higher level of parental monitoring in fifth grade was related to elevated trajectories of self-regulation across adolescence.

Taken together, the studies of parenting and adolescent IC (and related outcomes) have provided initial evidence that parental warmth, rejection, and monitoring may play distinct roles in adolescent IC development. However, most of the literature is based on informant-ratings of IC (rather than task performance) and parenting environments, which has a higher risk of subjective interpretation and inflates effects due to shared method variance including informant biases (Doty & Glick, 1998). Moreover, most of prior literature on parenting style has focused on maternal parenting, with only a few exceptions considered the effects of fathering on adolescent’s development. Accumulating empirical evidence suggested that maternal and paternal parenting may have similar influences on children and adolescents’ inhibitory process (Moilanen et al., 2015). It is essential that future studies, including the current research, should replicate and extend the literature by considering both maternal and paternal parenting and minimizing shared method variance through incorporating more IC indicators across methods (e.g., behavioral tasks, neural measures).
Family Socioeconomic Status, Parenting and Adolescent Inhibitory Control

Prior research has emphasized the importance of family SES and parenting in the development of IC (Farah et al., 2006; Noble et al., 2005; Sarsour et al., 2011; Spielberg et al. 2015; Ursache & Noble, 2016), yet the developmental pathways from family socioeconomic disparities to adolescents’ IC are not fully elucidated in the extant literature. According to Bronfenbrenner’s (2005) bioecological theory of human development, human development is a product of the interplay among four elements: Process (e.g. parent-child interaction), Person (e.g. neurobiological function), Context (e.g. family SES) and Time. Following this approach, researchers have proposed several ways these contextual factors work together to influence cognitive development. First, parenting may mediate the effect of family SES on IC development (Figure 1, paths ‘b’ and ‘c’). Low family SES, as characterized by high financial stress, may be associated with less warm, more hostile and less monitoring during parent-child interaction, which in turn is associated with low IC performance. Second, family SES and parenting behaviors also showed interactive effects on IC development. That is, the association between family SES and child IC varies depending on the level of parenting (Figure 1, path ‘d’).

Of relevance to the theorized mediating mechanism in the current project, family stress theory posited that family economic disadvantage may impact children’s self-regulation development through more proximal contextual factors to the child, including quality and sensitivity of parenting practices (Conger & Donnellan, 2007; Lengua, 2009; Masarik & Conger, 2017). Parents with low economic background are experiencing more emotional stress and are less likely to provide quality and supportive care to their
adolescents. Meanwhile, parents with lower education may have less knowledge of providing sensitive responsiveness to their children, which may hinder the development of neurocognitive functioning (Blair et al., 2011; Holochwost et al., 2016). Empirical evidence supporting this mechanism has been found among children and adolescents. Blair and colleagues (2011) examined the mechanisms underlying family SES, parenting practices, and young children’s EF performance in a longitudinal study of 3-year-olds. Parents’ intrusiveness and negative regard was found to mediate the associations between early family income-to-needs ratio and EF (including IC). Thus, family socioeconomic disadvantage was related to higher negative parenting behaviors and harsh negative feelings toward child, which in turn was associated with lower EF abilities. In a more recent cross-sectional study, Sarsour et al., (2011) found that lower family SES was associated with poorer IC among 10-year-olds. Moreover, parental responsivity (i.e. parental emotional and verbal sensitivity to child) and companionship (i.e. parental involvement in providing companionship) partially mediated the SES-IC association.

As to the moderation mechanism, resilience theory suggests that environmental, social and individual factors may moderate the negative effects of risks for predicting child outcomes (Zimmerman et al., 2013). Positive relationship with parenting has been found as an important protective factor that can mitigate the consequences of low family SES on child outcomes (Weisleder et al., 2016). Parents who are supportive and sensitive may be able to create a warm and supportive family environment that reduce the negative effect of low family SES on child cognitive development. Nurturing parents are also likely to engage child in learning activities and teach self-regulatory strategies, which promotes EF capacity. The instructions from parents may be important especially for low
family SES child who has relatively few learning resources (Lee et al., 2019; Rochette & Bernier, 2014). Relatively few empirical studies have investigated the moderation mechanism on IC development and the results are less consistent. Rochette and Bernier (2014) found that family SES and maternal parenting behaviors may have interactive effects on young children’s inhibitory capacities (at age 3). Specifically, family SES and high-quality maternal behaviors (e.g. response to distress, response to positive signal and physical proximity) showed significant interactive effect in predicting children’s ability to delay or suppress impulse responses. The link between the quality of maternal behavior and child impulse control was stronger among children from lower-SES families, but the link was negligible and not significant for children from higher SES families. The results suggested that high quality parenting may provide a buffering effect on child EF against the disadvantage associated with lower family SES. A recent study examined the interactive effects of grandparent SES and positive parenting on adolescent EF (around 12 years old) and found that positive parenting moderates the link between grandparent SES and cognitive flexibility, but not inhibitory control (Lee et al., 2019).

**The Current Study**

To address the limitations and gaps in knowledge in the literature, the main goal of the current investigation was to test hypotheses regarding family SES, parenting dimensions, and growth in IC in adolescence using longitudinal designs with multiple methods. Specifically, we aimed to address these gaps by: 1) characterizing the developmental pattern of IC in two longitudinal samples spanning late childhood to middle adolescence; 2) investigating the association between family SES and IC development during this period, 3) understanding the mechanisms by which family SES
influence IC development by testing the roles of family global parenting behaviors (i.e. parental warmth, rejection and monitoring) that include both maternal and paternal parenting behaviors. We examined the direct and indirect effects of family SES on the development IC task performance via different dimensions of parenting behaviors, and the interactive effects between family SES and different parenting behaviors on the development of IC task performance.

In the following chapters, we described each study separately. In Chapter 2 and 3, we presented the studies for early adolescent sample and middle adolescent sample, respectively. In each chapter, we introduced prior research evidence for IC development during this phase and common and specific research hypotheses for each sample, followed by detailed descriptions of the sample and measurements for key variables. Research findings were then presented and discussed in each chapter. In Chapter 4, we integrated the findings from both samples and discussed further on the current limitations, which cast light on future directions of research in the development of IC and further implications on the prevention and intervention of negative outcomes associated with poor IC during adolescence.
CHAPTER II

EARLY ADOLESCENT SAMPLE

Introduction

Though the development of IC has been well studied in young children, there is a gap in the literature about IC development transitioning from late childhood to adolescence. With the onset of puberty, substantial structural and functional changes occur in the brain, especially prefrontal cortex, and concomitant changes in the hormonal system significantly impact the development of cognitive capacities (Fuhrmann et al., 2015). How IC develops through such a critical transitional period is vastly understudied, and existing findings are mostly from cross-sectional studies, which confound age differences with potential cohort effects, and do not allow estimating of within-person changes. One goal of the current study was to investigate the development of IC during the transition to adolescence.

A handful of studies have examined the association between family SES and IC during this transition (Boelema et al., 2014; Spielberg et al., 2015). However, these studies usually did not control for potential confounding variables, such as race or ethnicity. National surveys suggest that African American and Hispanic American families have disproportionately lower income, lower education and higher unemployment rates, compared to White families (National Center for Education Statistics, 2015; U.S. Census Bureau, 2015). Therefore, family SES and ethnicity has been confounded in such a way that their unique effects on child outcomes are obscured (Hill, 2006). Another goal of the current study was to address this gap in knowledge, by examining family SES and adolescent IC development with a unique sample that had
roughly equal numbers of White, Black and Hispanic families, and that controlled for race/ethnicity in statistical analyses when analyzing potential SES effects.

**Inhibitory Control Development during the Transition to Adolescence**

Prencipe and colleagues (2011) examined youth EF task performance cross-sectionally across four age groups (from age 8 - 9 to 14 - 15 years) and revealed that substantial improvements in performance on the color word Stroop emerged relatively early in the age range tested, with the largest improvements occurring between age 10 - 11 years. These findings are in line with other cross-sectional studies that found age-related improvement in samples across even larger age ranges (Leon-Carrion et al., 2004; Liu et al., 2016; Schroeter et al., 2004). One prior longitudinal study has examined age-related changes in IC, which was assessed using the antisaccade task in a longitudinal sample of 9 - 26 years old (Ordaz et al., 2013). Results revealed that the task error rates and reaction times on corrected trials decreased significantly with age, suggesting that adolescent IC performance improved with age. Moreover, the improvements in performance were more rapid during the early assessments and decelerated with age. Significant variability was only observed at the intercept of the error rate, not the slope of the error rate or the developmental parameters of reaction times on correct trials. However, even in this study, the sample size representing the transition to adolescence were limited and one third of the participants were only measured once.

Other longitudinal studies examining the IC development during this transition revealed that IC changes were influenced by contextual factors, such as family SES (Boelema et al., 2014; Spielberg et al., 2015). As described in the previous chapter, this study showed that higher family SES was associated with increases in behavioral
Inhibition accuracy (but not reaction time) in a Go/No-go task from age 11 to 13, but lower family SES was associated with decreased behavioral inhibition accuracy (but again, not reaction time), especially for the “go” condition. This study provides initial evidence that family SES plays a critical role in the development of IC during the transition to adolescence; though correlational data, it is unlikely that early adolescent IC skills would be causally influencing family SES.

Ethnicity as a Confounding Variable of Socioeconomic Status

Race/ethnicity and SES are highly correlated and interplayed in complex ways to affect individual development and health disparity (Geronimus, Hicken, Keene, & Bound, 2006; Williams, 2012). Prior literature suggested substantial racial differences in SES and income and education may not be equivalent across race. Racial differences may account for a big portion of the observed socioeconomic disparities. In spite of the fact that family SES is highly correlated with race/ethnicity, prior studies usually did not consider the effects of race or ethnicity, while examining the association between family SES, parenting, and IC. Little of the prior research has examined racial or ethnic group differences in IC in childhood or adulthood (Zimmerman & Messner, 2013). However, ethnic and racial group differences in child cognitive and behavioral outcomes that are closely related to IC have been reported. For example, compared to White adolescents, African Americans and Hispanic youth have higher ADHD symptoms (Lee, Oakland, Jackson, & Glutting, 2008; Miller, Nigg, & Miller, 2009; Zuckerman, & Pachter, 2019), higher emotional problems and aggressive behaviors (McLaughlin, Hilt, & Nolen-Hoeksema, 2007; Vitoroulis & Vaillancourt, 2015), and higher impulsivity and sensation seeking that drive earlier alcohol use among minority adolescents (Choukas-Bradley,
With regard to parenting, racial and ethnic differences have also been found in child-rearing goals and behaviors among parents of adolescents. Prior literature has shown that African American families have lower levels of maternal involvement and warmth, as well as less authoritative parenting, compared to White non-Hispanic families (Holmes, Dunn, Harper, Dyer, & Day, 2013; Moilanen et al., 2009). Moreover, compared to African American and European American families, Hispanic parents of adolescents have higher expectations for compliance and higher monitoring of child’s behaviors (Dearing, 2004).

In the literature of family SES and IC development, White children and adolescents have comprised the majority of the samples, and African American and Hispanic families are underrepresented. Whether or not the results of IC development obtained from White samples could be generalized to other racial and ethnic groups is unknown (Hackman & Farah, 2009). In the current investigation, we mainly focused on European-American, African American, and Hispanic families as they are the three largest ethnic groups in the United States. Including sizable African American and Hispanic participant samples, the current study statistically controlled the effect of ethnicity while exploring the effect of family SES on IC development. Furthermore, the current findings of the development of IC and its associations with important family contextual factors could be generalized to a more diverse population.
Socioeconomic Status, Parenting and Inhibitory Control Development During the Transition to Adolescence

The transition from childhood to adolescence is characterized as dramatic physiological, psychosocial and cognitive development. Adolescents are undergoing substantial changes in the brain and the hormonal system as a result of pubertal development (Fuhrmann et al., 2015). In addition, the transition into adolescence is also accompanied by challenging social and emotional experiences that may further influence the improvement of cognitive control. Thus, late childhood and early adolescence may mark a period of particular vulnerability to contextual influences. As reviewed in the previous chapter, accumulating evidence has been found suggesting that youth IC development is linked to family SES and parenting during this transition (Sarsour et al., 2011).

As noted in previous chapter, family stress model emphasizes the role of proximal contextual factors, including quality and sensitivity of parenting practices, in the association between family economic disadvantage and child outcomes, including self-regulation development (Conger & Donnellan, 2007; Lengua, 2009). Parents with a low SES are likely to experience more emotional stress and are less likely to provide quality and supportive care to their adolescents. Economic press also leaves parents with less time and effort to make investments in their adolescents to promote cognitive and emotional development. (Blair et al., 2011; Holochwost et al., 2016).

Resilience theory argues that the negative effects of risks on child outcomes may be moderated by environmental, social and individual factors (Zimmerman et al., 2013). Following this line of reasoning, warm and supportive parenting may act as a buffer and
mitigate the negative effect of socioeconomic disadvantage on youth healthy development. Parents who are aware of their youth behaviors and knows what may be going wrong can undertake appropriate action. They can also guide youth behaviors and teach self-regulatory strategies, which promotes EF capacity (Lee et al., 2019; Rochette & Bernier, 2014; Weisleder et al., 2016). To date, empirical investigations of the mechanism underlying the association among SES, parenting and IC development during the transition to adolescence are relatively limited. The current longitudinal study was aimed to address the knowledge gaps mentioned above by directly examining the developmental changes in IC and investigating the association among SES, parenting and IC development during the transition into adolescence.

**Hypotheses**

In the current longitudinal study of the transition to early adolescence (i.e., from 9 to 13 years), the changes of IC task performance with age were first examined, following by the examination of the statistical prediction from family SES (the predictor) at age 9 to IC task performance (the outcome) at age 13, via parenting behaviors (the mediators) at age 10. This was done using a series of longitudinal mediation models. Based on the findings from the literature, the following hypotheses were proposed:

*Hypothesis 1.1:* Adolescents’ IC task performance will improve (i.e., increases in accuracy and decreases in reaction time) from age 10 to age 13.

*Hypothesis 1.2:* Lower family SES at age 9 years will be associated with poorer IC performance at age 10 and 13.

*Hypothesis 1.3:* Parenting behaviors will mediate the hypothesized SES effect (hypothesis 1.2) such that lower SES at age 9 will be related to
less parental warmth and monitoring, and more parental rejection at age 10, which in turn will be associated with less IC improvement from 10 to 13 years across early adolescence.

In the interest of fully examining the potential role of parenting behaviors in the association between family SES and IC development, statistical moderation models were tested as post-hoc analyses to examine the main and interactive effects of family SES and parenting on the IC during the transition to adolescence. Specifically, the moderation models tested whether the link between family SES at age 9 and IC task performance (level scores at age 10 and age 13, and changes from age 10 to 13) varied as a function of variance in parenting behaviors at age 10 (the moderators).

**Methods**

**Participants**

The sample was drawn from an international longitudinal study of parenting and child/adolescent development, Parenting Across Cultures (Lansford & Bornstein, 2011). This project is a collaboration across nine countries: the United States (Durham, North Carolina, \(N = 311\)), China (Jinan and Shanghai, \(N = 240\)), Colombia (Medellín, \(N = 108\)), Italy (Rome and Naples, \(N = 203\)), Jordan (Zarqa, \(N = 114\)), Kenya (Kisumu, \(N = 100\)), Philippines (Manila, \(N = 120\)), Sweden (Trollhättan/Vänersborg, \(N = 103\)), and Thailand (Chiang Mai, \(N = 119\)). Approximately 1,417 children and their parents were recruited when the children were 8 years old and assessed annually in “waves”, to assess family context, parenting behaviors and children’s adjustment.

The US sample (49.6% male, 50.4% female) was recruited in Durham, North Carolina, which in the past was a major manufacturing center in the tobacco industry and
remains a working-class industrial city. Durham has a population of 250,000, with 43% being European American, 14% being Hispanic American and 37% being African American (U.S. Census Bureau, 2018). The current sample (N = 311), included 111 European Americans, 97 Hispanic Americans and 103 African Americans. The sample is broadly representative of the proportion of White and African Americans, but with an overrepresentation of Hispanic Americans, so all three groups have roughly equal number of participants in the current study. Group comparisons indicated no differences among the ethnic groups on youth age or gender. However, European American parents were older and more highly educated than were Hispanic American and African American parents, and African American parents were older in age and more highly educated than were Hispanic American parents (See Appendix A for details).

For the purposes of the current investigation, family SES at wave 2 (M = 10.23 year-old, SD = .72), parenting behaviors at wave 3 (M = 11.04 year-old, SD = .78) and wave 5 (M = 13.95 year-old, SD = .65), and youth IC task performance at wave 3 and wave 6 (M = 14.76 year-old, SD = .77) were analyzed. Retention rates were high: 90% of the participants from the initial sample provided data at wave 2, and 76% of the sample still participated at wave 6. T-test was conducted to compare those who remained from those who dropped out of the study; there were no differences for youth’s age, gender, ethnicity, parents’ years of education, or family income (ps > .38).

**Procedure**

Adolescents were recruited from fifteen public and two private schools across the city, to ensure socioeconomic and ethnic diversity. After acquiring consent from parents and assent from the adolescents, the parent and youth completed a 1.5 to 2 hour
assessments including a demographic questionnaire and self-reported measures of family context (in wave 2) and parenting behaviors (in waves 3, 5 and 6), and behavioral tasks of cognitive control (in waves 3 and 6). These sessions were completed individually in participants’ homes, schools, or other locations chosen by the participants. Adolescents were given gifts and parents were given monetary compensation for their participation.

Measures

**Family SES.** At wave 2, parents reported their families’ annual gross income in a scale ranging from 1-10 (1 = less than $5,000; 2 = between $5,000 and $10,000; 3 = between $11,000 and $15,000; 4 = between $16,000 and $29,000; 5 = between $30,000 and $40,000; 6 = between $41,000 and $50,000; 7 = between $51,000 and $60,000; 8 = between $61,000 and $70,000; 9 = between $71,000 and $80,000; 10 = more than $81,000), and number of people living in the household. The median income in each category was used as a proxy of that family annual income. The family income-to-needs ratio was calculated using the family income divided by the federal poverty threshold for a family of that size (U.S. Census Bureau, 2018). It is worth noting that family income was transformed from an interval variable with non-regular spacing into a continuous variable, and the approximation may obscure the nonlinear nature of the construct. Father and/or mother each reported the years of education they each completed. Parental education level was calculated as the average of father’s and mother’s years of education. Family income-to-needs ratio and parental education were moderately correlated ($r = .52, p < .001$). Family income-to-needs ratio and parental education were standardized, averaged and standardized again to create family SES score, with a higher value indicating higher socioeconomic status.
**Inhibitory Control.** At wave 3 and wave 6, IC was measured using a computerized color–word Stroop task (See Figure 2; Banich et al., 2007). On each trial, the participant was presented with either a color-word (e.g. ‘YELLOW’) or a neutral word (e.g. ‘MATH’, ‘ADD’) and instructed to quickly press the button that matches the color in which the word was printed (i.e., its “ink”), and ignoring the meaning of the word. In this task, all the words were presented in a way that the ink color of the word was discordant with its semantic meaning (e.g. the word ‘YELLOW’ printed in red ink). Participants completed two experimental blocks of 48-trials each. The first block included an equal number of neutral and incongruent trials, and the second block included a greater number of neutral trials than incongruent trials (75% neutral vs. 25% incongruent). In each block, response accuracy was calculated as the proportion of correct responses on incongruent trials relative to all trials. Higher scores indicated better IC. Response time for each trial was also measured and averaged for neutral trials and incongruent trials, separately. The average response time for incongruent trials was used to indicate resistance to interference, with higher scores indicating poorer resistance to interference, or poorer IC. Within both waves, accuracy and response time were negatively correlated ($r = -.15, p = .03$ for wave 3 and $r = -.23, p = .002$ for wave 6); because the covariation was modest, these were analyzed separately.

**Parental Warmth and Rejection.** Mother (wave 3, 5 and 6), father (wave 3, 5 and 6) and child (wave 3 and 5) each rated aspects of parenting behavior using the Parental Acceptance-Rejection Questionnaire (Rohner, 2005) that is rated using a 4-point Likert scale, ranging from 1 (Almost never) to 4 (Everyday). This questionnaire includes 29 items capturing parental warmth (8 items, e.g. “My mother/father says nice things
about me” or “I say nice things about my child”), parental hostility (6 items, e.g. “My mother/father hits me, even when I do not deserve it” or “I hit my child, even when (s)he does not deserve it”), parental neglect (6 items, e.g. “My mother/father pays no attention to me” or “I pay no attention to my child”), parental rejection (4 items, e.g. “My mother/father sees me as a big nuisance” or “I see my child as a big nuisance”) and parental control (4 items, e.g. “My mother/father is always telling me how I should behave” or “I always tell my child how (s)he should behave”).

The parental acceptance-rejection theory (PARTheory) and confirmatory factor analysis in Rohner and Cournoyer (1994) supported a two-factor structure, with the eight items from the warmth subscale loaded on a parental warmth factor and 16 items from the hostility, neglect and rejection subscales all loaded on a parental rejection factor. Therefore, a parental warmth score was computed as the average of eight items from the warmth subscale, and a parental rejection score was computed as the average of all the items from the hostility, neglect and rejection subscales. In the current sample, internal consistency (Cronbach’s α) scores were .74 - .89 (depending on informant and wave) for parental warmth and .72 - .79 for parental rejection.

Within each wave, youth-report parenting behaviors were only modestly correlated with parents’ self-report (.08, ns, to .29, p < .001 for wave 3 fathering; .04, ns, to .31, p < .001 for wave 5 fathering; .00, ns, to .15, p = .02 for wave 3 mothering; .06, ns, to .23, p < .001 for wave 5 mothering). Youth-report parenting behaviors were used in the following analysis, given than youth’s perception of parental behaviors has been found to be a more proximal and stronger correlate than parents’ self-perceptions of their parenting behaviors, with youth behavioral outcomes (Abar, Jackson, Colby, & Barnett,
Turning to whether adolescents perceived maternal and paternal behaviors similarly, the magnitudes of correlations between youth-reported mothering and fathering were moderate to high ($rs = .57 - .64$, $ps < .001$ for parental warmth; $0.62 - .69$, $p < .001$ for parental rejection). Therefore, a global parental warmth score was computed by averaging youth-reported maternal and paternal warmth for each wave, and a global parental rejection score by averaging maternal and paternal rejection at each wave.

**Parental Monitoring.** Parents (mothers at wave 3, 5 and 6) and youth (wave 3 and 5) rated 10 items on parents’ knowledge of youth behaviors (5 items, e.g. “How much do you/your parents try to know who your child/you spend time with?”) and parents’ monitoring of youth behaviors (5 items, e.g. “How often do you/your parents set rules or limits on who your child/you spend time with?”). These first 5 items were rated on a scale from 0 (I/They don’t try) to 2 (I/They try a lot), and the second set of five items were rated on a scale from 0 (Never) to 3 (Always). Responses for the first five items were recoded into 0, 1.5 and 3 (to align its scale with the second item set) and a parental monitoring total score was computed as the average of the 10 items, with higher scores indicating greater parental monitoring. Within each wave, youth-reported parent monitoring was only modestly correlated with parents’ self-reported monitoring ($rs = .01 - .30$ for wave 3; $rs = .13 - .31$ for wave 5; $r = .23$ for wave 6). Youth’s perception of parental monitoring at wave 3 and 5 were used in the analyses, because (as noted above) the adolescents’ perceptions of their parenting environment is most likely to be associated with their development outcomes.
Data Analysis Plan

**Descriptive analyses.** Descriptive analyses were performed to examine the normality of distributions and outliers. Outliers are values more than 3 SD from the mean. For skewness and kurtosis, the acceptable levels are less than 3 and less than 10, respectively (Kline, 2005). Bivariate correlation analyses were used to examine the associations among variables. Paired sample t-tests were conducted to compare adolescent IC across the two waves that it was assessed.

**Predictive Models.** Next, to test the study hypotheses, path analyses were conducted to examine the association among family SES, parenting behaviors and adolescents’ Stroop task performance using Mplus 7.4 software package (Muthén & Muthén, 1998 - 2012). Six path analysis models were performed to examine the effects of family SES on two Stroop task performance indicators via three parenting behaviors, separately. As shown in Figure 3, there were ten sets of parameters in each path analysis model: 1) regression parameters estimating the effects of family SES at wave 2 on parenting behaviors and adolescent IC at wave 3 and wave 5/6; 2) auto-regressive parameters that estimated the rank-order stability of individuals on parenting behaviors and Stroop performance from wave 3 to wave 5/6; 3) cross-lagged regression parameters estimating the predictive effects of one construct (i.e. parenting behaviors/adolescent IC) on the other construct (i.e. adolescent IC/parenting behaviors) in the following assessment; and 4) the parameters that estimated the concurrent associations between parenting behaviors and adolescent IC. This model was capable of capturing potential indirect effects of family SES on adolescent IC via parenting behaviors, while controlling
the indirect effects of family SES on parenting behaviors via adolescent IC, and long-term stabilities of and concurrent links between parenting behaviors and adolescent IC.

Overall model fit was evaluated by three indices: chi-square ($\chi^2$) value, Root Mean Square Error of Approximation (RMSEA), and Confirmatory Fit Index (CFI). A nonsignificant chi-square value indicates a good model fit. RMSEA values of less than .08 are considered an acceptable fit while values less than .05 are considered an excellent fit (Browne & Cudeck, 1993). CFI values of greater than .90 are considered an acceptable fit while values greater than .95 are considered a good fit (Bentler, 1990).

Indirect effects were calculated using the IND command in MPlus. Bias-corrected bootstrap confidence intervals (CIs) for the indirect effects were calculated using 10,000 bootstrapping samples (MacKinnon, Lockwood, & Williams, 2004). These CIs take non-normality of the estimates into account and are therefore not necessarily symmetric. Full information maximum likelihood (FIML) estimation procedure was used to estimate the missing data. Compared to listwise deletion or other ad hoc methods, FIML reduces the potential bias elicited by missing data and decreases the likelihood of Type I error (Arbuckle, 1996; Schafer & Graham, 2002).

**Moderation Models.** Three sets of multiple linear regression analyses were conducted to examine the moderation effects of parenting behaviors on the association between family SES and adolescent IC (Figure 4). Set 1 examined the main and interaction effects of family SES at wave 2 and parenting behaviors at wave 3 on adolescent IC at wave 3. Set 2 examined the main and interaction effects of family SES at wave 2 and parenting behaviors at wave 3 on adolescent IC at wave 6. In set 3, we first calculated a residualized change score for adolescent IC by regressing IC at wave 6 on IC.
at wave 3, and then tested the main and interaction effects of family SES at wave 2 and parenting behaviors at wave 3 on the residualized change score of adolescent IC. The residualized change scores represent the change across time, and compared to simple difference scores, have the advantage that they adjust for baseline differences (MacKinnon et al., 2013).

**SES and Race/Ethnicity Confound.** Given the significant association between ethnicity and family SES, additional analyses were conducted to determine whether the effects of family SES detected in the analyses would hold when statistically controlling for race/ethnicity. In order to do that, two dummy variables (Hispanic American = 0 (No) or 1 (Yes); African American = 0 (No) or 1 (Yes)) were created to represent three ethnic groups and made the European American group the reference group (Hispanic American = 0 and African American = 0). The mediation models and moderation models were then re-estimated while controlling for the effects of ethnic groups on parenting behaviors and adolescent IC indicators.

**Results**

**Descriptive and Correlational Analyses**

Descriptive statistics and bivariate correlations among the main study variables are presented in Table 1. All study variables were normally distributed with skewness values below 3 and kurtosis values less than 10. Family SES at wave 2 correlated with parenting behaviors at wave 3 to a modest degree, such that higher family SES was related to higher youth-report parental warmth, and lower levels of parental rejection. Greater family SES at wave 2 was associated with lower reaction time at wave 6.
Accuracy and reaction time were negatively correlated at each wave, and they were modestly to moderately stable over the two assessments.

**Hypothesis 1.1**

Paired-sample $t$-tests indicated that accuracy significantly increased ($t(171) = -8.70, p < .001$) and reaction time significantly decreased ($t(171) = 12.84, p < .001$) across the two waves, suggesting that IC improved from 10 to 13 years.

**Hypothesis 1.2, 1.3: Socioeconomic Status, Parenting, and Adolescent Inhibitory Control**

A set of path analysis models was utilized to examine the associations between family SES and adolescent IC, with parenting warmth, rejection and monitoring as mediators. In these models, we were able to test the indirect path from family SES at wave 2 $\rightarrow$ parenting behaviors at wave 3 $\rightarrow$ adolescent IC at wave 6 while controlling for the effects of family SES on adolescent IC and parenting behaviors at wave 6 (that is, the autoregressive effects of and the concurrent correlations between parenting behaviors and adolescent IC) and the potential indirect path from family SES at wave 2 $\rightarrow$ adolescent IC at wave 3 $\rightarrow$ parenting behaviors at wave 6. The initial models were fully saturated ($0 df$), as they estimated all the possible paths.

**Parental warmth as mediator.** The model for accuracy was fully saturated as it estimated all the possible paths. Results (see Figure 5 showing standardized path estimates; unstandardized path estimates are presented in text) indicated that family SES at wave 2 was associated with parental warmth at wave 3 ($b = .25, SE = .06, p < .001$), which in turn, was marginally associated with accuracy at wave 6 ($b = .09, SE = .05, p = .09$). Higher parental warmth at wave 3 was associated with higher parental warmth at
wave 5 ($b = .37, SE = .07, p < .001$). Higher accuracy at wave 3 was associated with higher accuracy at Wave 6 ($b = .18, SE = .06, p = .004$). Family SES at wave 2 was not directly associated with parental warmth at wave 5 ($b = .02, SE = .07, p = .82$), or accuracy at wave 3 ($b = .02, SE = .07, p = .76$) or wave 6 ($b = .06, SE = .05, p = .28$). Accuracy at wave 3 was not associated with parental warmth at wave 5 ($b = .11, SE = .07, p = .11$). Parental warmth did not covary with accuracy ($b = -.05, SE = .06, p = .45$ at wave 3; $b = .02, SE = .05, p = .66$ at wave 5/6). The indirect effect of family SES at wave 2 on parental warmth at wave 5 via adolescent IC at wave 3 was not tested given the link between family SES and accuracy at wave 3 was not significant.

The bias corrected bootstrap test revealed that the indirect effect from SES at wave 2 to adolescent IC (accuracy) at wave 6 via parental warmth at wave 3 was significant ($b = .03, SE = .01, 95\% CI [.003, .068]$). Results suggested that higher family SES at wave 2 was associated with higher parental warmth at wave 3, which was linked to greater IC accuracy at wave 6 even while controlling for prior IC performance at wave 3.

For reaction time (see Figure 6), the model was also fully saturated. Higher family SES at wave 2 was directly associated with higher parental warmth at wave 3 ($b = .25, SE = .08, p = .002$) and lower reaction time at wave 6 ($b = -.13, SE = .06, p = .03$). Higher parental warmth at wave 3 was associated with higher parental warmth at wave 5 ($b = .37, SE = .07, p < .001$). Lower reaction time at wave 3 was associated with lower reaction time at wave 6 ($b = .40, SE = .08, p < .001$). Family SES was not associated with reaction time at wave 3 ($b = .001, SE = .07, p = .98$) or parental warmth at wave 5 ($b = .02, SE = .07, p = 80$). The cross-lagged effects between parental warmth and reaction
time were not significant (reaction time wave 3 → parental warmth at wave 5: $b = .03$, $SE = .06$, $p = .58$; Parental warmth at wave 3 → reaction time wave 6: $b = -.02$, $SE = .07$, $p = .80$). Therefore, neither of the indirect effects (i.e., family SES at wave 2 → reaction time at wave 3 → parental warmth at wave 5; family SES at wave 2 → parental warmth at wave 3 → reaction time at wave 6) was tested.

**Parental rejection.** As presented in Figure 7 for accuracy, the model was saturated. Family SES at wave 2 was associated with parental rejection at wave 3 ($b = -.19$, $SE = .07$, $p = .016$). In turn, parental rejection at wave 3 was marginally associated with accuracy at wave 6 ($b = -.12$, $SE = .07$, $p = .06$). Higher parental rejection at wave 3 was associated with higher parental rejection at wave 5 ($b = .37$, $SE = .07$, $p < .001$). Higher accuracy at wave 3 was associated with higher accuracy at wave 6 ($b = .17$, $SE = .07$, $p = .011$). Family SES was not directly associated with parental rejection at wave 5 ($b = .06$, $SE = .05$, $p = .25$), or accuracy at wave 3 ($b = .02$, $SE = .06$, $p = .73$) or wave 6 ($b = .06$, $SE = .06$, $p = .31$). Accuracy at wave 3 was not associated with parental rejection at wave 5 ($b = -.06$, $SE = .07$, $p = .37$). Parental rejection did not covary with accuracy ($b = .02$, $SE = .05$, $p = .97$ at wave 3; $b = -.009$, $SE = .03$, $p = .80$ at wave 5/6). An indirect effect of family SES at wave 2 on parental rejection at wave 6 via adolescent accuracy at wave 3 was not tested given the nonsignificant link between family SES and accuracy at wave 3. However, the bias corrected bootstrap test indicated that the indirect effect from early family SES to accuracy at wave 6 via parental rejection at wave 3 was significant ($b = .03$, $SE = .01$, 95% CI [.001, .068]), suggesting that higher family SES at wave 2 was associated with lower parental rejection at wave 3, which in turn was associated with higher accuracy at wave 6, even while controlling for prior accuracy.
The model fit for the parental rejection and reaction time (see Figure 8) model was also saturated, family SES at wave 2 was directly associated with parental rejection at wave 3 ($b = -.19, SE = .06, p = .002$) and reaction time at wave 6 ($b = -.11, SE = .06, p = .08$). Parental rejection at wave 3 also marginally associated with reaction time at wave 6 ($b = -.19, SE = .07, p = .016$). Parental rejection and reaction time showed moderate stability across waves ($b = .38, SE = .06, p < .001$ for Parental rejection; $b = .41, SE = .07, p < .001$ for reaction time). Family SES at wave 2 was not associated with reaction time at wave 3 ($b = .001, SE = .07, p = .99$) or parental rejection at wave 5 ($b = .05, SE = .06, p = .31$). Reaction time at wave 3 was not associated with parental rejection at wave 5 ($b = -.03, SE = .05, p = .47$). Parental rejection and reaction time did not covary ($b = .01, SE = .06, p = .81$ at wave 3; $b = .04, SE = .05, p = .42$ at wave 5/6).

The indirect effect of family SES at wave 2 on parental rejection at wave 6 via adolescent IC at wave 3 was not tested given the nonsignificant link between family SES and reaction time at wave 3. The bias corrected bootstrap test showed that the indirect effect of family SES at wave 2 on reaction time wave 6 via parental rejection at wave 3 was significant ($b = -.03, SE = .02, 95\% CI [-.083, -.001])$, even while controlling for prior reaction time.

**Parental monitoring.** In Figure 9, results for parental monitoring and accuracy are presented. Family SES at wave 2 was not directly associated with parental monitoring or accuracy at either wave. The cross-lagged effects between parental monitoring and accuracy were not significant. Thus, the indirect effect from family SES to accuracy at wave 6 via parental monitoring at wave 3 was not tested.
For reaction time (see Figure 10), family SES at wave 2 was associated with reaction time at wave 6 (\(b = -0.14, \ SE = .06, \ p = .03\)). However, the indirect effect of family SES at wave 2 on reaction time at wave 6 via parental monitoring at wave 3 was not tested, given the nonsignificant association between family SES at wave 2 and parental monitoring at wave 3 (\(b = .003, \ SE = .07, \ p = .99\)) and between and parental monitoring at wave 3 and reaction time at wave 6 (\(b = .05, \ SE = .07, \ p = .43\)).

To address potential confounds with race/ethnicity and SES, the models were re-estimated with the previously identified significant indirect effects but now including two dummy coded ethnicity variables into the path analysis models. Results did not change. The indirect effects of family SES at wave 2 on accuracy and reaction time at wave 6 via parental warmth and rejection at wave 3 were significant, while controlling for the potential effects of ethnicity on performance and parental behaviors.

In a post-hoc analysis, the indirect effect models were re-run but with the income and parental education indicators of SES separated out, in order to determine whether the effects were due to specific SES variables. Results are shown in the Appendix B. In summary, results showed significant indirect effects of family income at wave 2 on accuracy at wave 6 via parental warmth at wave 3, such that higher family income was linked to higher parental warmth, which in turn was associated with greater accuracy (\(b = .02, \ SE = .01, \ 95\% \ CI \ [.002, .049]\)). Family income at wave 2 was also associated with parental rejection at wave 3, which was marginally associated with accuracy at wave 6, however, the indirect effect of family income on accuracy via parental rejection was not significant (\(b = -.19, \ SE = .07, \ 95\% \ CI \ [-.001, .055]\)). Parental education did not show any direct or indirect effects on accuracy or reaction time at either wave.
Parenting Behaviors as Moderators

A series of regression analysis were conducted to test the main and interactive effects of family SES at wave 2 and parenting behaviors at wave 3 (i.e. parental warmth, rejection and monitoring) on adolescent accuracy and reaction time at wave 3, wave 6 and the changes between two waves (using residualized change scores by regressing accuracy/reaction time at wave 6 on accuracy/reaction time at wave 3). Detailed results are presented in the Appendix C. No significant interaction effect was detected out of eighteen regression analyses, suggesting that parenting behaviors did not moderate the association between SES and adolescent Stroop performance.

We also reran all the parenting moderation models while controlling for the potential effects of race/ethnicity. Adding the previously described dummy-coded ethnicity variables as predictors did not change the results (i.e., there still were no significant parenting-by-SES interaction effects).

Discussion

This study had three main goals, to examine: 1) the development of IC during the transition from late childhood to adolescence (hypothesis 1.1); 2) whether early family SES would directly or indirectly influence development of IC in early adolescence via parenting behaviors (hypotheses 1.2 and 1.3); and 3) whether various parenting practices would statistically moderate the link between family SES and IC development. To address these aims, the current study used a multiple-wave longitudinal sample, of which adolescents were assessed at ages 9, 10, 12 and 13 years annually. Path analysis models were used to test several hypotheses regarding direct and indirect links between SES, parenting behaviors and adolescent IC development.
The results supported the first hypothesis—that adolescent IC improves from age 10 to age 13. Specifically, IC task performance showed significant increases in accuracy and decreases in reaction time during the transition from late childhood to early adolescence. The results echoed the findings of previous cross-sectional studies spanning 10 to 19 years of age, which have found that both IC response accuracy and response time develop gradually from childhood into adolescence (Best & Miller., 2010; Duell et al., 2018; Huizinga et al., 2006; Leon-Carrion et al., 2004; Luna et al., 2004).

The study also provided evidence for the second and third hypotheses. Family SES at age 9 showed modest direct association with IC performance, especially reaction time at age 13. Specifically, higher family SES at age 9 was associated with lower reaction time scores at age 13 (hypothesis 1.2). Also as hypothesized (hypothesis 1.3), there were significant indirect effects of higher family SES at age 9 on greater IC accuracy at age 13, via the mediator of higher parental warmth and lower parental rejection at age 10. These findings generally supported the idea that family socioeconomic disadvantage may impact self-regulation development in the transition to puberty, through more proximal factors to the child such as harsher and less supportive parenting practices (Lengua, 2009). Our results revealed that the indirect effects of early family SES on youth IC via parental warmth and parental rejection were significant, which are in line with the findings from previous studies investigating young children (Blair et al., 2011; Holochwost et al., 2016) and adolescents (Sarsour et al., 2011). It is worth mentioning that the current study’s results were significant even after controlling for the effect of earlier IC on later parenting behaviors, the concurrent association between parenting behaviors and IC, and the longitudinal stabilities of parenting.
behaviors and youth IC across time. The results provide stronger longitudinal evidence for potential parenting effects on rank-order changes in youth’s IC over time; in contrast, there was no evidence suggesting “child effects” of IC on subsequent rank-order changes in parenting behaviors during the transition to adolescence. A notable exception in the findings is that parental monitoring, which has been found to be related to adolescent self-regulation (Bowers et al., 2011), did not mediate the association between family SES and IC development. The discrepancy between this null result and the presence of this association in prior studies could be due to the different methods used across studies to measure parental monitoring and adolescent IC.

It is worth noting that the above results were obtained even while statistically controlling for the potential confound between SES and family race/ethnicity. Previous studies usually have unbalanced and nonrepresentative proportions of members of various racial/ethnic groups, which makes it hard to isolate specific SES effects from the effects of racial and ethnic background (Hackman & Farah, 2009). In the current sample, roughly equal number of White, African American and Hispanic families participated. Therefore, the sample was maximally powered for statistically separating any potential SES effects from race/ethnicity effects, by controlling for the effects of ethnicity while we are testing the effects of family SES. Results revealed that family SES was associated with race/ethnicity; White families had significantly higher incomes and parent education levels than African American families, whose SES was significantly higher than the Hispanic families. Adding ethnicity to the path analysis models did not change the pattern of the results, which suggested that even though ethnicity is confounded with SES, the indirect effects of family SES on the development of adolescent IC via parenting.
practices were independent of race/ethnicity variation. This result is in line with results found for other EF capacities in younger children (Hackman, Gallop, Evans, & Farah, 2015).

The interaction effects between parenting behaviors and family SES on IC development were tested, and the results were not significant. SES at age 9 and parenting behaviors at age 10 did not show any interactive effects on IC task performance at either wave or across waves (and this pattern did not change when race/ethnicity was included in the models). Together with the findings form mediation analyses, our results provide evidence for family stress model rather than resiliency model and suggested that family economic disadvantage is associated with behavioral IC development through parenting behaviors, which are more proximal contextual factors.

The current findings should be interpreted with a few limitations. First, the longitudinal design in the current study only measured IC task performance twice (at age 10 and 13), which does not allow us to test the shape and slope of developmental trajectories of IC task performance through the transition from late childhood to early adolescence; three or more assessments are required to do that. The early adolescence has been characterized as a rapid period of developmental changes in brain functioning and behavioral IC (measured by antisaccade task) has been found to develop dramatically during this time (Ordaz et al., 2013). IC measured using other behavioral tasks are needed to further confirm the developmental trend. Moreover, a longitudinal design with three or more time points that spans development before and during the entry into puberty will be to model the shape of the developmental changes, and to model variance in the developmental changes during this key transition. Second, parenting behaviors were
measured at Wave 3 and Wave 5 while behavioral IC indicators were measured at Wave 3 and Wave 6. The lag between the two assessments of parenting is shorter than that of IC performance, and parenting has also been found to be relatively stable across development (Van Heel et al., 2019). Parenting showed slightly higher stability than IC performance, especially accuracy, across the two waves. Higher stability of parenting from Wave 3 to Wave 5 may be one of the reasons that IC indicators at Wave 3 fail to account for any additional variance in parenting at Wave 5 (Taris & Kompier, 2006).
CHAPTER III
MIDDLE ADOLESCENT SAMPLE

Introduction

As described in the previous chapter, prior studies focusing on adolescence are limited and yield mixed findings. Some studies have revealed significant age effects on task performance, as older age was associated with greater accuracy and faster response time across a variety of IC behavioral tasks (Best & Miller., 2010; Duell et al., 2018; Liu et al., 2016; Luna et al., 2004). IC performance does not reach a plateau until early adulthood (Steinberg et al., 2018). However, in other studies, age-related differences have been found in only one of several IC indicators (Schroeter et al., 2004), or in no IC indicators (Andrews-Hanna et al., 2011). The mixed findings in prior behaviors studies may be due in part to differences in the way age was treated (i.e. as a continuous vs. categorical variable). It could also be due to the specific experimental paradigms (i.e., the use of cognitive interference versus response inhibition tasks) used in different studies. Researchers have long suggested investigating the neural basis of IC development, which will advance our understanding of the developmental changes observed in behavioral measures of IC processes and potentially explain the discrepancies observed between cross-sectional studies. The first goal of the current study is to examine the development of behavioral and neural correlates of IC during middle adolescence. Characterizing the brain circuitry underlying IC development can help us to better understand the inconsistent findings in prior literature, and elucidate the neural functioning related to adolescent decision making (Luna, Padmanabhan, & O’Hearn, 2010; Nigg, 2017). The
second goal of this study is to explore the contextual sources of individual differences in IC development and investigate the mechanisms underlying the association among family SES, parenting and the development of behavioral and neural correlates of IC. Prior neuroimaging studies (mostly cross-sectional) have also demonstrated that family context is an important source of variation that contributes to individual differences in brain development and neurocognitive processing (Foulkes & Blakemore, 2018; Noble et al., 2015), however, little is known about how important family contexts, such as family SES and parenting, are linked to the neurodevelopmental trajectory of IC during middle adolescence.

**Neural Correlates of Inhibitory Control**

Neuroimaging research has suggested that the maturation of the frontal cortex underlies the emergence of inhibition process and higher order EF during early childhood (Colombo & Cheatham, 2006; Cuevas et al., 2012). Throughout childhood, the brain regions and neural circuitries associated with IC are undergoing dramatic changes, along with the salient improvement in behavioral performance in IC tasks (Moriguchi, & Hiraki, 2013; Zelazo, 2015). Meta-analyses of healthy adult samples have revealed that dorsal anterior cingulate cortex (dACC), dorsolateral prefrontal cortex (DLPFC), ventrolateral prefrontal cortex (VLPFC), and supplemental motor area (SMA) and pre-SMA were consistently activated in a range of IC tasks that tax interference control, response inhibition, and response selection (Simmonds, Pekar, & Mostofsky, 2008; Nee, Wager, & Jonides, 2007). dACC is linked to detecting error and sending signals to adjust motor responses (Kerns, 2006; Iannaccone et al., 2015; Weiss et al., 2018). DLPFC and VLPFC are critical for coordinating and planning goal-directed behaviors (Badre &
As a sub-region of posterior medial frontal cortex, pre-SMA has extensive pre-frontal connectivity and is important for selecting an appropriate behavior to execute, or inhibiting an inappropriate response (Zhang, Ide, & Li., 2012).

However, prior developmental neuroimaging studies have revealed mixed results with regard to the relative activation of these brain regions at different ages. Several studies have reported higher activation in frontal lobes in adults compared with children or adolescents during the color-word Stroop task (Adleman et al. 2002), Go-NoGo task (Bunge et al. 2002), and other IC tasks (Rubia et al. 2000; Rubia et al. 2006). These cross-sectional results generally support a maturational hypothesis of brain function, which posits that later-maturing brain regions are crucial in the development of behavioral improvements in IC seen from childhood to adulthood. However, some IC studies have found higher frontal lobe activation in children and adolescents than in adults, which may reflect decreased effort needed to reach similar behavioral performance with age increases (Alahyane, Brien, Coe, Stroman, & Munoz, 2014; Velanova et al., 2008). Other studies have identified distinct nonlinear developmental trends for different prefrontal regions. A recent neuroimaging study focusing on a large sample of 8-to 19-year-olds found distinct activation patterns for different regions across age: activity in pre-SMA was lower among older adolescents, but activity in dACC was highest among the youngest and oldest participants (i.e., U-shaped pattern in relation to cross-sectional age groupings (Liu et al., 2016). Developmental neuroimaging studies of IC have been almost entirely cross-sectional, which limits the ability to detect the
developmental trajectories of neural correlates of IC and individual differences in those trajectories.

Indeed, longitudinal neuroimaging studies focusing on the developmental changes in functional brain regions have been rare. Existing research has shown that different brain circuits show distinct developmental rates of change from childhood to adulthood. Ordaz et al (2013) estimated the growth curves of IC-related brain regions elicited by an antisaccade task, using a longitudinal sample of participants between ages 9 to 26 years who participated 1-6 times. Results revealed that dACC showed continued increases in activation from late childhood to adulthood, and there was no significant variability in slopes across development. In contrast, activity in DLPFC decreased with age until adolescence, but again, the individual differences in slopes were stable across development. Mean growth curves of activation in the pre-SMA revealed no changes with age, but significant individual differences in slopes were observed with a pattern of trajectories converging over time. These results echo the findings from cross-sectional studies showing that brain functioning for motor response control may be mature by late childhood (Rubia et al., 2003), brain function associated with error processing (e.g. dACC) may continue to mature throughout adolescence (Buzzell et al., 2017; Velanova et al., 2008), and brain regions associated with inhibitory control (e.g. DLPFC and VLPFC) decrease in activity throughout adolescence (Durston & Casey, 2006; Steinbeis & Crone, 2016; Velanova et al., 2008). However, more conclusive longitudinal research is needed to replicate and extend the preliminary evidence from cross-sectional studies.
Contextual Factors and Brain Function

Recent neuroimaging studies suggest that contextual factors, such as family socioeconomic status (SES), are crucial for the development of IC-related brain regions. Specifically, family SES not only influences the brain at both anatomical and functional levels, but also impacts the relations between brain and behavior (Noble et al., 2015; Spielberg et al., 2015; Ursache & Noble, 2016). Noble and colleagues (2015) directly examined the role of brain structure in the association between SES and EF performance, including IC, in a large sample of typically developing individuals between 3 to 20 years old. Results indicated that family income was strongly related to brain surface area, especially among disadvantaged children. Specifically, among children from lower income families, small differences in income were associated with relatively large differences in surface area—and this association was most salient in regions supporting language, reading, EF and spatial skills. Furthermore, children’s whole-brain surface area partially mediated the link between family income and children’s performance on EF tasks (mainly capturing IC and working memory), after controlling for age and other confounding factors. Specifically, low family income was related to smaller surface area, which in turn was associated with poorer IC performance in individuals. Although there is a growing body of cross-sectional research on this topic, the mechanisms through which family SES influence the neurobiological development of IC have not yet been elucidated (Brito & Noble, 2014; Ursache & Noble, 2016). Evidence from cross-sectional studies is not able to test whether these potential effects of family SES emerge during adolescence or are due to the impact of SES earlier in life.
In a two-wave longitudinal neuroimaging study of young adolescents (11 and 13 years), Spielberg and colleagues (2015) examined the role of SES on the development of IC and related brain functioning. Results revealed that lower SES was associated with both decreased behavioral IC performance (as indexed by No-go vs. Go accuracy) and a concurrent increase in ACC activity over this 2-year period. Moreover, lower SES was related to decreased ACC and DLPFC coupling over time. These findings imply that adolescents from lower SES backgrounds appear to develop less efficient inhibitory processing in DLPFC and ACC. However, this study only examined a 2-year interval in early adolescence and had only two waves, and therefore was limited in its ability to examine how family SES might be linked with individual differences in developmental trajectories of neural and behavioral IC measures across adolescence.

Parenting behaviors are other important contextual factors that have drawn research attention. There is accumulating evidence suggesting that normative variation in parenting behaviors is not only associated with adolescent cognitive, emotional and behavioral development (Schwartz et al., 2014; Callaghan et al., 2017), but also linked to structural and functional brain development during adolescence (Belsky & de Haan, 2011; Luby et al., 2013; Whittle et al., 2016; Whittle et al., 2017). A handful of studies investigating the association among SES, parenting and adolescent brain development have provided evidence for both mediation and moderation mechanisms. Luby and colleagues (2013) examined the association between exposure to poverty, parenting behaviors and brain development during adolescence. Results suggested that exposure to poverty during early childhood is associated with poor developmental outcomes in adolescence (i.e. smaller white and cortical gray matter and hippocampal and amygdala
volumes). Moreover, parental support/hostility mediates the link between poverty and hippocampal volume, such that early poverty is associated with less parental support and more parental hostility, which are linked to smaller hippocampal volume. A recent longitudinal study provides evidence for the interactive effects between parenting and family SES in influencing adolescent brain development. Whittle and colleagues (2017) investigated the association among neighborhood-and family-level SES, positive parenting and adolescent brain development in a group of adolescents measured 3 times from ages 11 to 20 years. Results revealed positive parenting buffers the negative effects of low family-level SES on the development of the amygdala. It also mitigates the effects of neighborhood disadvantage on the development of dorsal frontal and orbitofrontal cortices, which are important for executive functioning. However, direct evidence examining the mechanism of family SES, parenting and behavioral and neural correlates of IC during adolescence is still lacking.

One primary goal of the present study was to delineate developmental changes in behavioral and neural functioning associated with IC during middle adolescence. Latent growth models (LGM) were used to characterize the growth trajectories of behavioral IC performance and related neural activity in a sample of 14-to 17-year-olds. Empirical studies also provided initial evidence that family SES may influence the neural functioning related to IC during adolescence. However, whether and how early family SES influence the behavioral and neural IC is largely unknown. The second goal of the study is to investigate whether family SES and parenting practices influence the individual differences in the developmental trajectories of behavioral and neural IC. To this end, family SES and parenting behaviors were added into the LGMs as predictors to
examine their direct effects on IC development and indirect effects of family SES on the intercept and slope of IC development via different parenting behaviors. Lastly, interactions of family SES and different parenting behaviors on IC development were tested to examine whether parenting practices moderate the association between family SES and IC development. Based on the prior literature, we proposed the following hypotheses:

**Hypothesis 2.1**: Behavioral IC task performance will show a significant increase and neural activity will show a significant decrease from age 14 to 17 years. In addition, significant individual differences in the intercept and slope of behavioral and neural indicators of IC will be found.

**Hypothesis 2.2**: Family SES at age 14 will statistically predict adolescent’s IC, such that low SES is associated with poorer IC performance at age 15 (intercept), and slower IC growth from age 15 to 17 (slope).

**Hypothesis 2.3**: Parenting behaviors will mediate the hypothesized SES effect (hypothesis 2.2). Specifically, lower SES will be linked with more negative parenting behaviors and less supportive parenting behaviors, which in turn will be associated with poorer IC intercept and slower IC growth (slope) from 15 to 17 years.

Just as the prior chapter, a series of statistical moderation models were tested as post-hoc analyses to examine the main and interactive effects of family SES and parenting on the IC growth during middle adolescence. Specifically, the moderation
models tested whether the link between family SES at age 14 and IC growth (intercept at age 15 and slope from age 15 to 17) varied as a function of variance in parenting behaviors at age 15 (the moderators).

**Methods**

**Participants**

The current middle-adolescent aged sample is drawn from a longitudinal study of adolescent health risk behaviors (Kim-Spoon et al., 2016). A total of 167 adolescents (47% females) were recruited from southwest Virginia, US and assessed annually four times. Adolescents were 13 - 14 years of age at Wave 1 ($M = 14.13, SD = 0.54$), 14 - 15 years of age at Wave 2 ($M = 15.05, SD = 0.54$), 15 - 16 years old at Wave 3 ($M = 16.07, SD = 0.56$), and 16 - 17 years old 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 - $59,000 according to United States Census Bureau, 2010). Adolescent participants were primarily White (80%), African American (13%), or “other” (7%). The current sample was generally representative of rural southwest Virginia regarding household income and ethnicity. There were 26 families who withdrew from the study between Wave 1 and Wave 4 for reasons such as: declined further participation, lost contact with the study, move out of the area, and “other”. Logistic regression was conducted to compare those who withdrew and those who provided data in all four waves, and results revealed no differences in age, gender, ethnicity, parents’ years of education, or family income ($ps > .15$).
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 participants provided written consent for a protocol approved by the institutional review board of the university.

Measures

Inhibitory Control. Adolescents’ IC 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 seconds and asked to identify the unique number among three digits by pressing a button with the index. 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 Figure 11). Subjects 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 accuracy (i.e., difference in percentage of correct responses of the neutral and interference trials) and reaction time (i.e., reaction time differences between correctly responded interference and neutral trials). 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 & Engle, 2003). Greater adolescent IC was characterized by higher accuracy and faster (i.e., lower) reaction time.

**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 3T 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, FoV=245x245 mm, and 192 slices with the spatial resolution of 1x1x1 mm. Echo-planar images (EPIs) were collected using the following parameters: slice thickness=4mm, 34 axial slices, field of view (FoV)=220 x 220mm, repetition time (TR)= 2 s, echo time (TE)=30 ms, flip angel=90 degrees, voxel size=3.4 x 3.4 x 4 mm, 64x64 grid, and slices were hyperangulated at 30 degrees from anterior-posterior commissure. Neuroimaging 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. Functional volumes were normalized using parameters from a segmented anatomical image coregistered to the average EPI and smoothed using a 6mm full-width-half-maximum Gaussian filter.

**Neural correlates of Inhibitory control.** At each wave, individual-level regions-of-interest (ROI) values were extracted at coordinates corresponding to peak activations in the interference minus neutral second-level contrast for each participant. The first
eigenvariate values of the contrast images were extracted using spherical masks of 6mm surrounding MNI coordinates, thresholded at $p < .001$, family-wise error corrected. Significant regions for each wave are presented in Appendix D. To ensure theoretically meaningful metrics of inhibitory control and also maximize data reliability, ROIs that fulfilled the following criteria were used in the statistical modeling: (1) regions known from the literature to be engaged by inhibitory control and related to interference- and error-processing, and (2) that appeared at all four time points. Among all the ROIs, pre-SMA emerged in all four assessments and is one of the prominent regions that has been related to interference processing in prior research (Bush et al., 2003). Variance in activity in pre-SMA was thus chosen as a neural correlate of inhibitory control. Bivariate correlates revealed that pre-SMA activity was negatively associated with MSIT accuracy at Wave 1 and Wave 2 ($r_s = -.37 - -.27, ps < .01$), and positively correlated with MSIT reaction time at Wave 1 and Wave 2 ($r_s = .21 - .30, ps < .01$). Thus, higher neural activity in pre-SMA indicated poorer adolescent IC performance.

**Family SES.** Parents reported their annual income using an ordinal scale (1 = None; 2 = less than $1,000; 3 = $1,000 - $2,999; 4 = $3,000 - $4,999; 5 = $5,000 - $7,499; 6 = $7,500 - $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 $49,999 a year. For each family, income was estimated as the mid-point of the category parents chosen. As mentioned in the previous section, 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 then calculated, using the same method as the early adolescent sample (see Chapter II), by dividing the family income by the federal poverty threshold for a family of that size. Father and mother each reported their years of education and a parental education score was calculated as the average of father and mother education level. Family income-to-needs ratio and parental education were moderately correlated \( (r = .49, p < .001) \). A family SES index was calculated by standardizing, averaging and standardizing again the scores of family income and parental education, with higher scores indicating higher family SES.

**Parental Warmth and Attachment Security.** Adolescents rated perceived positive supportive parenting from mother and father using the Inventory of Parent and Peer Attachment (IPPA, Raja, McGee, & 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. Adolescents answered each item separately for their mother and father. Sample questions include “My mother/father respects my feelings” and “My mother/father accepts me as I am”. High internal consistency scores (Cronbach’s \( \alpha = .82 \) - .92) were found for the total score across all four waves. An overall warmth score was calculated as the average of the 12 items, with higher scores indicating higher levels of perceived warmth and security. Within each wave, adolescent-report maternal warmth and paternal warmth were moderately correlated \( (rs = .43 \text{ to } .56, ps < .001) \). Therefore, a global parental warmth score was computed by averaging youth-report maternal warmth and paternal warmth at each wave.
**Parental Negativity.** Adolescents were also asked to rate negative aspects of their relationship with parents using Parent-Child Relationship Scale (PCR, Hetherington & Clingempeel, 1992). Adolescents answered 7 items separately for mother and father on a 5-point Likert type scale, ranging from 1 (extremely) to 5 (not at all). A total score was computed by reverse-scoring items and averaging responses to create an overall parent-child negativity score. On this scale a higher score indicates higher parent-child negativity. Sample items include: “How much does your mother/father yell at you after she/he has had a bad day?”, and “How much does you criticize your mother/father?”. This scale has shown good reliability across four waves (Cronbach’s alpha = .82 - .87). A global parental rejection score was created by averaging child reported maternal rejection and paternal rejection at each wave. Within each wave, adolescent-report maternal negativity and paternal negativity were moderately correlated ($r_s = .34$ to $.48$, $ps < .001$). Thus, a global parental negativity score was computed by averaging youth-report maternal negativity and paternal negativity at each wave.

**Parental Monitoring.** Adolescents also reported different aspects of parental monitoring using the Parental Monitoring Scale (Stattin & Kerr, 2000). This scale includes 25 items in a total score, capturing subscales pertaining to parental knowledge (9 items), child disclosure (5 items), parent solicitation (5 items), and parental control (6 items). Sample questions include, “Do your parents normally know where you go and what you do after school?”, “How often do your parents talk with your friends when they come over to your house?”, and “Does your child keep a lot of secrets from you about what he/she does during his/her free time?”. A parental monitoring score was calculated as the average of the 25 items at each wave, with higher score representing higher
parental monitoring. This overall monitoring score showed good reliability across four time points (Cronbach’s alpha = .90 - .94).

Note that parents also rated their parent-child relationship (i.e. warmth, negativity and monitoring) using the same scales. However, like the early adolescent sample study (Chapter II), adolescent-report parenting behaviors were only modestly correlated with parents’ self-reports. Thus, we focused on adolescents’ perceptions of parenting behaviors to be consistent with the early adolescent sample study , and also given than youth’s perception of parental behaviors has been found to be a more proximal and stronger correlate than parents’ self-perceptions of their parenting behaviors, with youth behavioral outcomes (Abar et al., 2015; Frampton et al., 2010; Taylor et al., 2011).

**Data Analysis Plan**

Descriptive statistics and bivariate correlations were estimated to examine the normality of distributions and the associations among the main study variables.

A latent growth modeling (LGM) approach was then used to test the research hypotheses. LGM is based on the general structural equation modeling (SEM) framework, and estimates growth parameters over time as latent factors. SEM-based models treat the latent factor as a random variable and estimate both the mean and variance for each latent variable. LGM also allows growth parameters to be modeled as outcomes, and individual differences in growth can be correlated with other variables in the model (Curran & Willoughby, 2003).

LGM was used to examine: a) developmental trajectories of behavioral and neural correlates of IC from age 14 to 17 (hypothesis 2.1); b) the direct and indirect effects of family SES on the developmental trajectories of IC via parenting behaviors (hypotheses
2.2 and 2.3); c) the main and interaction effects of family SES and parenting behaviors in predicting IC change. To this end, we implemented three sets of LGMs.

The first set of models were unconditional LGMs that estimated the growth trajectories of behavioral and neural correlates of IC (MSIT accuracy, MSIT reaction time and Medial frontal cortex activity) over four time points (from age 14 to 17). The models were configured to estimate individual initial level (intercept) and growth (linear slope and quadratic curves), as well as the overall averages and variances of these growth parameters using maximum likelihood estimation. Intercept, linear slope and quadratic curve capture the starting point, the rate of linear change and the quadratic change over time, respectively. All models initially included the intercept, linear slope and quadratic latent factors. Loadings of the four time points onto the latent intercept were fixed at one, loadings onto the latent slope were fixed at 0, 1, 2, 3, and loadings on the latent quadratic term were fixed at 0, 1, 4, 9. Mplus 7.4 software package was used to estimate the LGMs, and model fit was evaluated by chi-square value, RMSEA, and CFI. A nonsignificant chi-square value indicates an adequate model fit, so do RMSEA values below .08 and CFI values above .90 (Bentler, 1990).

The second set of models tested whether and how the developmental trajectories of behavioral and neural correlates of IC are linked with variance in family SES. We fit conditional LGMs with family SES at wave 1 as a predictor on the growth parameters of IC (estimated from waves 2 to 4; see Figure 12). A significant path coefficient from family SES at wave 1 to the latent IC intercept indicates that prior family SES predicts initial level of IC. Significant path coefficients from family SES at wave 1, to latent IC
linear and quadratic slope terms indicate that prior family SES predicts the shape and rate of developmental change in IC across waves 2 to 4.

We then tested the indirect effects of family SES at Wave 1 on the growth parameters of adolescent IC via parenting practices following the recommendations suggested by Preacher and Hayes (2008). The indirect model included paths (a) from family SES at Wave 1 to parenting behaviors at Wave 2, (b) from parenting behaviors at Wave 2 to the intercept and slope of adolescent IC, and (c) from family SES at Wave 1 to the intercept and slope of adolescent IC. Indirect effects were calculated using the IND command in MPlus. Bias-corrected bootstrap confidence intervals (CIs) were calculated for the indirect effects using 10,000 bootstrapping samples.

Finally, to examine whether the association between family SES at wave 1 and the developmental trajectories of adolescent IC would be moderated by parental behaviors, we fit conditional LGMs and added family SES at Wave 1, parenting behaviors at Wave 2 and the interaction term of family SES-by-parenting behavior as predictors (see Figure 13). In the first step of the model, all growth parameters were regressed onto the independent variables of family SES and parenting behaviors. In the second step, an interaction term representing the product of family SES and parenting behavior was added. In total 9 conditional LGMs were tested (3 outcomes × 3 moderators). Each significant interaction was probed using simple slopes analysis, in which the association between SES and the growth parameter(s) was estimated at low (-1 SD) and high (+1 SD) levels of the parenting behavior variable.
Results

Descriptive and Correlational Analyses

The descriptive statistics and bivariate correlations among the main study variables are presented in Table 2. Higher family SES at Wave 1 was linked with higher parental warmth at Wave 4. Parental warmth, rejection and monitoring showed rank-order stability across four waves. Average MSIT accuracy (difference score between Interference condition and neutral condition) increased from -.10 to -.02 and average MSIT reaction time (correct trials in Interference condition minus neutral condition) decreased from .48 to .34 seconds across the four waves; both patterns suggest that adolescent behavioral IC improves from 14 to 17 years. In addition, pre-SMA activity gradually decreased from Wave 1 to Wave 4. MSIT accuracy, reaction time and neural activities all presented moderate to high rank-order stability across the four time points. MSIT task performance was significantly correlated with pre-SMA activity at wave 1 and wave 2; though no longer significant in waves 3 and 4, the correlation was in the same direction as in waves 1 and 2.

Parenting behaviors generally were modestly associated with MSIT task performance and neural activity. Higher parental warmth and monitoring linked to higher MSIT accuracy and lower MSIT reaction time. On the contrary, higher parental rejection was associated with lower MSIT accuracy and longer MSIT response time. Lower neural activity was associated with lower parental rejection and higher parental warmth and monitoring.
Hypothesis 2.1

**Behavioral IC.** Each individual’s data for MSIT accuracy and reaction time, along with the sample mean, are presented in Figure 14 and 15. As shown in the figures, MSIT accuracy gradually increased and MSIT reaction time gradually decrease across the four time points. To formally test the developmental trajectory of MSIT performance, we started by fitting univariate LGMs with intercept, linear slope and quadratic term from Wave 1 to Wave 4 for MSIT accuracy and reaction time, respectively. Both models fit the data well ($\chi^2(3) = 2.60, p = .46$, CFI = 1.00, and RMSEA = .00 for MSIT accuracy; $\chi^2(3) = 1.37, p = .71$, CFI = 1.00, and RMSEA = .00 for MSIT reaction time).

The means and variances of intercepts, slopes and quadratic terms for these two models are presented in Table 3. The intercept for both MSIT accuracy and response time showed significant variance. Moreover, MSIT accuracy increased from Wave 1 to Wave 4 with significant variances. MSIT response time decreased from Wave 1 to Wave 4, with significant variance. In both models, the mean and variance for the quadratic terms were not significant, which indicated that MSIT accuracy and reaction time followed a linear growth pattern from Wave 1 to Wave 4.

Note that in subsequent analyses (for hypotheses 2.2, and 2.3) developmental trajectories of MSIT performance from Wave 2 to Wave 4 were analyzed instead, so that SES measured at wave 1 could precede the growth parameters (estimated across waves 2 to 4). Thus, as a preliminary step at this stage of analyses, additional univariate LGMs with intercept and linear slope were fitted for MSIT accuracy and MSIT reaction time from Wave 2 to Wave 4, and both models showed good fit ($\chi^2(1) = .89, p = .34$, CFI = 1.00, and RMSEA = .00 for Accuracy; $\chi^2(1) = 1.37, p = .24$, CFI = 1.00, and RMSEA
The results are listed in Table 3 and were very similar to those reported above for the growth parameters estimated from wave 1 to wave 4.

**Neural Correlates of IC.** Growth trajectories of pre-SMA activity at individual and mean level over four time points are presented in Figure 16. Like behavioral indicators of IC, the neural correlates of IC also showed a linear change over the four time points. We fit univariate LGM with intercept, linear slope and quadratic term to formally test the trajectory of pre-SMA activity from Wave 1 to Wave 4 ($\chi^2(3) = 0.55, p = .97, CFI = 1.00, \text{and} \ RMSEA = .00$). As shown in Table 3, the mean and variance for the quadratic terms were not significant, which like the behavioral indicators of IC, indicates a linear change pattern across the four time points.

As with the behavioral accuracy and reaction time models, prior to testing subsequent hypotheses the univariate LGMs for pre-SMA were rerun from Wave 2 to Wave 4. The model fit the data well ($\chi^2(1) = 1.13, p = .29, CFI = .99, \text{and} \ RMSEA = .03$), with significant mean level for intercept and slope and significant variability for intercept but not slope (i.e., individuals showed similar developmental trends over the three time points).

**Hypotheses 2.2 and 2.3: Direct and Indirect Links between Family Socioeconomic Status and Inhibitory Control Development**

In the next step, we estimated the association between family SES and the development of IC by adding family SES at Wave 1 as a predictor on the latent intercept and slope variables in each LGM for waves 2 to 4. For MSIT accuracy, the model fit the data well ($\chi^2(2) = .96, p = .62, CFI = 1.00, \text{and} \ RMSEA = .00$). However, family SES at Wave 1 was not associated with the intercept ($b = .004, SE = .003, p = .21$) or slope ($b = .05$ for Reaction time).
= .00, SE = .002, p = .96) of MSIT accuracy. For MSIT reaction time, the model also fit the data well ($\chi^2(2) = 1.68, p = .43, \text{CFI} = 1.00, \text{and RMSEA} = .00$). Family SES at Wave 1 was not associated with the intercept ($b = -.006, SE = .006, p = .32$) or slope ($b = -.002, SE = .003, p = .50$) of MSIT reaction time. For pre-SMA activity, the model also fit the data well ($\chi^2(2) = 2.74, p = .25, \text{CFI} = 1.00, \text{and RMSEA} = .00$). Family SES at Wave 1 was not associated with the intercept ($b = -.004, SE = .003, p = .87$) or slope ($b = .002, SE = .02, p = .92$) of pre-SMA activity. In summary, results indicated that family SES at Wave 1 was not directly associated with the intercept or the slope of IC development, and hypothesis 2.2 was not supported.

To test hypothesis 2.3, indirect predictive effects of family SES on adolescent IC development via parenting behavior were also tested. There was no evidence of any significant indirect, mediated effects through any of the parenting behavior scores. Detailed results are presented in the Appendix E.

**Parenting Behaviors as Moderators**

In the final set of analyses, the moderating effect of parenting behavior on the association between early family SES and the development of adolescent IC was tested. To this end, the latent growth parameters were regressed on family SES at Wave 1, parenting behaviors at Wave 2 and the two-way interaction term of family SES-by-parenting behaviors in adolescent MSIT accuracy, reaction time and neural activities models, respectively. Results are shown in Table 4.

**Parental warmth as the moderator.** The model fit the data well for MSIT accuracy ($\chi^2(3) = 3.20, p = .36, \text{CFI} = .99, \text{and RMSEA} = .02$). However, neither the
main effects nor the interaction effect of family SES and parental warmth was significant in predicting the intercept or the slope of MSIT accuracy.

Regarding MSIT reaction time, the model fit the data well ($\chi^2 (3) = 2.21, p = .70, CFI = 1.00, \text{ and } \text{RMSEA} = .00$). A significant main effect of parental warmth on the intercept and slope of MSIT reaction time was observed. A significant interaction effect of family SES-by-parental warmth was also found in predicting the intercept of MSIT reaction time. We used simple slopes in post-hoc probing of the interaction, with family SES regressed back on the intercept of MSIT reaction time at various standard deviation (SD) thresholds above and below the mean of parental warmth. Results indicated that higher family SES was associated with lower MSIT reaction time at lower levels of parental warmth (at -1SD: $b = -.018, SE = .008, p = .03$). In contrast, the link between family SES and the intercept of MSIT reaction time was near zero and nonsignificant at higher levels of parental warmth (at +1SD: $b = .006, SE = .008, p = .45$).

Regarding neural activity in pre-SMA, the model fit was excellent ($\chi^2 (3) = 3.72, p = .44, CFI = 1.00, \text{ and } \text{RMSEA} = .00$). None of the main effects was significant in predicting the intercept and slope of pre-SMA activity. Significant interactions between family SES and parental warmth on the intercept and slope of pre-SMA activity were observed. Simple slope analysis was performed in post-hoc probing of the interaction. The association between family SES and pre-SMA activity was not significant in either level (high, or low) of parental warmth, so the interaction term could not be interpreted. This problem could have been due to restricted variance in the latent intercept score. Therefore, the simple slope analysis was performed again with family SES, but this time regressed back on the raw score of Wave 2 pre-SMA activity (rather than its latent
intercept score), at standard deviation (SD) thresholds above and below the mean of parental warmth. Results indicated that higher family SES was associated with lower pre-SMA activity at lower levels of parental warmth (-1SD: $b = -0.10$, $SE = 0.04$, $p = 0.01$). In contrast, the link between family SES and the raw scores of pre-SMA activity was near zero and nonsignificant at higher level of parental warmth (+1SD: $b = 0.06$, $SE = 0.04$, $p = 0.11$).

**Parental rejection as the moderator.** For MSIT accuracy and reaction time, even though the models fit the data well ($\chi^2(3) = 1.91$, $p = 0.59$, CFI = 1.00, and RMSEA = 0.00 for MSIT accuracy and $\chi^2(3) = 1.42$, $p = 0.84$, CFI = 1.00, and RMSEA = 0.00 for MSIT reaction time), neither the main effects nor the interaction effects of family SES and parental rejection were significant in predicting the intercepts or slopes of MSIT accuracy or reaction time scores. Detailed results are presented in Table 4.

Regarding pre-SMA activity, the model had an excellent fit ($\chi^2(3) = 4.54$, $p = 0.34$, CFI = 0.98, and RMSEA = 0.03). Significant interaction effects of family SES-by-parental rejection on the intercept and slope of pre-SMA activity were observed. To interpret the interaction, we performed simple slopes analysis with family SES regressed back on the raw scores of Wave 2 pre-SMA activity at various standard deviation (SD) thresholds above and below the mean of parental rejection. Results indicated that higher family SES was marginally associated with lower pre-SMA activity at higher levels of parental rejection (+1SD: $b = -0.08$, $SE = 0.04$, $p = 0.06$). In contrast, the link between family SES and the intercept of pre-SMA activity was near zero and nonsignificant at lower levels of parental rejection (-1SD: $b = 0.05$, $SE = 0.04$, $p = 0.25$).
Parental monitoring as the moderator. For MSIT accuracy and reaction time, the models fit the data well ($\chi^2(3) = .53$, $p = .91$, CFI = 1.00, and RMSEA = .00 for MSIT accuracy and $\chi^2(3) = 3.73$, $p = .44$, CFI = 1.00, and RMSEA = .00 for MSIT reaction time). However, also as shown in Table 4, neither the main effects nor the interaction effect of family SES and parental monitoring were significant in predicting the intercept or slope of MSIT accuracy or reaction time.

Regarding pre-SMA activity, the model fit the data well ($\chi^2(4) = 6.35$, $p = .17$, CFI = .95, and RMSEA = .06). Significant interaction effects of family SES-by-parental monitoring were found in predicting the intercept of pre-SMA activity. To interpret the interaction, we performed simple slopes analysis with family SES regressed back on the raw score of Wave 2 pre-SMA activity at various standard deviation (SD) thresholds above and below the mean of parental monitoring. Simple slope analyses revealed that higher family SES was associated with lower pre-SMA activity at lower levels of parental monitoring ($-1SD: b = -.10$, $SE = .04$, $p = .02$). In contrast, the association between family SES and the intercept of pre-SMA activity was near zero and nonsignificant at higher level of parental monitoring ($+1SD: b = .05$, $SE = .04$, $p = .18$).

In a post-hoc analysis, the moderation models with significant interaction effects were re-run but with the income and parental education indicators of SES separated out, in order to determine whether the effects were driven by specific SES variables. In summary, results showed that parental warmth moderated the link between family income and the intercept of pre-SMA activity and the link between parental education and the intercept of pre-SMA activity. Parental monitoring moderated only the link between family income and the intercept of pre-SMA activity, not the association
between parental education and pre-SMA activity. Parental rejection did not moderate the association between pre-SMA activity with either family income or parental education.

**Discussion**

Utilizing a four-wave longitudinal sample from 14- to 17-years of age, the current study aimed to characterize the developmental trajectories of IC task performance and its neural correlates during middle adolescence. This study also aimed to investigate the associations among family SES, parenting behaviors and behavioral and neural correlates of IC development, and the mechanism underlying the above associations by testing hypothesized pathways presented in Figure 1 (paths ‘b’ and ‘c’ for mediation mechanism and ‘d’ for moderation mechanism).

The first hypothesis was that IC would show gradual improvement from 14 to 17 years. LGMs were fitted to examine the growth trajectories of IC task performance indicators and pre-SMA activity, and results revealed that MSIT accuracy and reaction time showed linear change from Wave 2 to Wave 4. There also was significant variability in both intercept and slope. The findings generally support hypothesis 2.1 and are in line with the findings from prior studies that also have shown significant improvement of IC during middle to late adolescence (Liu et al., 2016; Ordaz et al., 2013). For pre-SMA activity, there was significant variance in the intercept and mean level decreases in slope over time (although there was not significant variance in the slope component). Results indicated that individuals showed significant differences in pre-SMA activity at middle adolescence (at initial assessment), and these individual differences persisted across four assessments. These results echo findings from prior studies suggesting a pattern of decreasing pre-SMA activity (found in a cross-sectional sample of 8-to19-year-olds; Liu
et al., 2016), and decreasing DLPFC activity from 9 to 26 years of age (found in a longitudinal sample of adolescents; Ordaz et al., 2013).

For the second and third hypotheses, the predictive effects of family SES at Wave 1 and parental behaviors at Wave 2 on the growth trajectories of IC development were examined in a series of conditional LGMs. Specifically, we examined the direct effect of family SES at Wave 1 on the growth trajectories of IC (behavioral performance and neural activity), as well as the indirect effect of family SES at Wave 1 on the growth trajectories of IC (behavioral performance and neural activity) via parenting behaviors at Wave 2. The current analyses found no evidence for direct or indirect effects of family SES on the development of IC task performance or neural activation changes, via various parenting behavior constructs. This null finding appears to be different from previous research showing significant association between early family SES and IC development in childhood through early adolescence (Farah et al., 2006; Noble et al., 2005; Noble et al., 2007) or showing that parenting mediates the association between family SES and EF (including IC; Blair et al., 2011; Sarsour et al., 2011).

For the statistical moderation model, evidence was found that the link between family SES at Wave 1 and growth trajectories of IC varied as a function of parenting behaviors at Wave 2. Specifically, the results revealed that higher family SES was associated with a higher MSIT accuracy intercept, but only in families with low levels of parental warmth; the link between family SES and the intercept of MSIT accuracy was near zero and nonsignificant at higher levels of parental warmth. A similar pattern was found for neural correlates of IC. Higher family SES was associated with lower pre-SMA activity intercept, only at lower levels of parental warmth and monitoring, and higher
levels of parental rejection. In contrast, the link between family SES and pre-SMA activity intercept was near zero and nonsignificant at higher levels of parental warmth and monitoring, and lower levels of parental rejection. The findings are supportive of prior findings that the interaction of parental warmth and family SES influence EF development in young children (Rochette & Bernier, 2014), and that parenting moderates the link between neighborhood-level and family-level SES and brain development (Whittle et al., 2017).

According to the results, the association between family SES and adolescent IC development was not mediated by parenting behaviors. Rather, the intercept of behavioral IC development was more strongly related to family SES only in the face of low parental warmth. This finding is in line with the resilience theory, which suggests that the negative effects of risks on child outcomes may be moderated by environmental, social and individual factors (Zimmerman et al., 2013). Parenting behavior that is warm and supportive appears to buffer youth from negative cognitive and behavioral outcomes that are associated with growing up in a lower SES context. In contrast, less warm parenting may increase adolescents’ vulnerability to the negative effects associated with low family SES, in terms of IC as an outcome. Our findings expand upon prior research by demonstrating the association between family SES and the intercept of statistical interaction effects of family SES and the neural correlates of IC vary as a function of parenting behaviors (Spielberg et al., 2015). For adolescents from less supportive and more rejecting parenting environments, the intercept of IC-related neural development may be particularly vulnerable to the negative effects associated with low family SES. In contrast, parenting behaviors that are supportive in emotional warmth and supervision
(i.e., monitoring) appear to be a buffer and protect youth from negative effects of low family SES on brain development—at least with regard to IC as measured using the MSIT. It is plausible that in the families with low parental warmth and high parental rejection, family stress strains adolescent’s stress response system, which in turn impacts the neural functioning related to IC development in prefrontal cortex, which also has a high concentration of glucocorticoid receptors (Arnsten, 2009; Blair et al., 2005; Evans & Schamberg, 2009). On the contrary, parental warmth and affection may mitigate the negative effects of family stress on adolescent’s stress response system by creating a sensitive and supportive environment.

However, it is worth noting that the present study showed strong evidence for the interactive effects of family SES and parenting behaviors on the intercept of the behavioral and neural indicators of IC development. Their statistical effects on the slopes of developmental trajectories were modest and nonsignificant. For behavioral IC, its slope was not predicted by the interactive effects of family SES and parenting behaviors, indicating that the SES disparities in IC were stable across time and did not increase or decrease. For neural correlates of IC, the interactive effects of family SES and parenting behaviors on the slope of pre-SMA activity were significant. However, the interactive effects were not further explored given the nonsignificant variance found in the latent slope of pre-SMA activity.

Results of the current study should be considered in light of the following limitations. First, the current study only tested the developmental trajectory of one ROI that is related to interference processing and appears at all four time points. Prior literature suggested that age-related improvements in IC are related to the development of
component-specific brain systems, as well as their integration (Hwang, Velanova, & Luna, 2010; Luna et al., 2015). However, the current investigation could not determine whether and how multiple ROIs work together to influence IC development during adolescence. Future studies would benefit from a more integrative “whole brain” perspective when examining the brain development associated with behavioral IC. Prior neural connectivity literature has revealed dynamic brain network remodeling throughout development that may support the improvement in behavioral IC observed in adolescence (Hwang et al., 2010). Longitudinal studies are warranted to better understand these kinds of network dynamics and to investigate developmental changes in neural connectivity across distributed brain systems. Second, although longitudinal design was used in the current study with family SES measured at Wave 1 and parenting and youth IC measured at Wave 2 to 4, the nature of correlational data prevents us from inferring causality. Moreover, the current design does not rule out the possibility of passive gene-environment correlations, that is, better youth IC may be due to the same genes that contribute to higher parental education, higher family income and better caregiving behaviors (Bridgett, Burt, Edwards, & Deater-Deckard, 2015).
CHAPTER IV

GENERAL DISCUSSION

IC is a key cognitive capacity that is slowly developing through adolescence and is critical for regulating impulses and risk-taking behaviors at all stages of the lifespan (Braams et al., 2015; Duell et al., 2018; Durston et al., 2003; Kim-Spoon et al., 2016; Lahat et al., 2012; Nigg, 2017). The emergence and development of IC in childhood has been widely studied, and dramatic improvement of IC has been found during the preschool years (Carlson & Moses, 2001; Garon et al., 2008; Moriguchi, & Hiraki, 2013; Simmonds et al., 2008). However, much remains unknown about the developmental change of IC across adolescence, and most studies to date have employed cross-sectional designs. Individual differences in adolescent IC have been found in those cross-sectional studies, but the mechanisms underlying the development of individual differences is largely unknown.

Social contexts, such as family SES and parenting behaviors, are important contextual factors influencing individual differences in IC development during childhood (Farah et al., 2006; Morries et al., 2007; Noble et al., 2005; Roskam et al., 2014). There is an increasing number of studies investigating the links between family SES, parenting behaviors and IC development in childhood and the findings suggest two mechanisms. First, family SES indirectly influences IC development through its effect on parenting behaviors (i.e., parenting is a mediator) (Blair et al., 2011; Luby et al., 2013; Sarsour et al., 2011). Second, family SES also impacts IC development in distinct ways, depending on parenting behaviors in the home (i.e., parenting is a moderator) (Rochette & Bernier, 2014; Weisleder et al., 2016; Whittle et al., 2017). Due to the persistent links of SES and
parenting with child IC development, it is worthwhile to also examine their roles in the development of adolescent IC. However, little is known about whether and how these mechanisms apply in adolescence.

The current investigation added to a growing literature seeking to describe the development of adolescent IC and elucidate the potential mechanisms that may influence individual differences in IC development. We addressed these research gaps using two longitudinal study samples that spanning the period from late childhood to middle adolescence. Distinct analytic models were employed to address specific research hypotheses in each study. All of the analyses were directed toward extending knowledge of the development of IC during adolescence and of the processes underlying the associations between family SES, parenting behaviors and IC.

**Development of Inhibitory Control during Adolescence**

The first goal of the current study (tested as hypothesis 1 in both study samples) was to delineate the development of behavioral and neural correlates (only in the second study sample) of IC across late childhood to middle adolescence. The development of IC task performance during adolescence was tested using distinct IC tasks in the two samples (i.e. Color-word Stroop task in the early adolescent sample, and MSIT in the middle adolescent sample). To summarize, the results from two samples indicated that IC task performance improved throughout early- and mid-adolescence, and the activity of neural regions that are closely related to IC task performance decreased across mid-adolescence. Together, these behavioral and neural variables showed longitudinal changes indicative of gradual improvements in IC in adolescence. This finding is in line with existing cross-sectional behavioral studies showing that IC task performance
improves through adolescence and does not reach its plateau until early adulthood (Best & Miller, 2010; Duell et al., 2018; Huizinga et al., 2006).

In the early adolescent study sample, results indicated that both reaction accuracy and reaction time during the Stroop task improved significantly from 10 to 13 years of age, during the transition to adolescence. In the middle adolescent study sample, growth curves of MSIT task performance were estimated using LGMs; they showed that both MSIT accuracy and reaction time showed linear change across middle adolescence and the growth parameters (both intercept and slope) varied significantly between individuals. To our knowledge, only one prior study has examined the longitudinal developmental trajectory of IC task performance in adolescence (Ordaz et al., 2013). Our findings are consistent with this study, revealing that age-related improvements in performance continue through adolescence, and show significant individual variability in starting points as well as rates of improvement (i.e., slopes), in most indicators of IC that were assessed. Compared to Ordaz et al., the current investigation focused on developmental trajectories of behavioral IC in a large sample of adolescents all measured for four times and with a variety of contextual factors, which provided a unique opportunity to deepen the research literature on the neural correlates and potential mechanisms of change in IC development during adolescence.

Within the neuroscience literature, although brain regions that are related to adolescent IC have been demonstrated in an accumulating body of cross-sectional studies, there has been no clear conclusion regarding the age-related changed in BOLD response during IC across adolescence. Prior studies have found both linear increases and decreases in neural activity with development, as well as some evidence of non-linear
changes in relevant brain regions (Bush et al., 2003; Liu et al., 2016; Luna et al. 2010; Ordaz et al., 2013; Velanova, et al., 2008).

Our results revealed that pre-SMA, which supports interference control, showed linear decreases in activity across mid-adolescence. The slope estimate did not show significant variability, suggesting individuals show essentially parallel trajectories. That is, the rank order of individuals in their neural activity earlier in mid-adolescence, was stable; participants who showed the greatest activity in pre-SMA at 14-years of age, continued to be the individuals with the highest levels of neural activity four years later. This finding aligns with results from prior developmental neuroimaging study suggesting that other ROIs that are closely related to IC also showed nonsignificant variability in slope as development proceeds across adolescence (Ordaz et al., 2013).

When considered as a whole, the current study’s findings appear to indicate that neural activity may become more efficient with development, as less brain activity is required to achieve better behavioral performance. The literature suggests that the greater efficiency observed in later adolescence could be due to dramatic structural and functional neurodevelopment occurring across puberty (Fuhrmann et al., 2015). For instance, synaptic pruning in the frontal cortex enables improvement of computational abilities in local circuits by trimming the synapses that are not commonly used. Meanwhile, myelination processes may promote the integration of different neural systems and contribute to greater synchronization of neural activity (Luna et al., 2010; Nave & Werner, 2014). Both the pruning and myelination processes may help reduce demands on local processing and result in lower BOLD responses during IC task performance, reflecting reduced task demand on prefrontal regions (Ghuman et al. 2008;
Luna et al., 2015). This idea is supported further by prior literature on adults, which has suggested that adults show lower activity in dorsolateral prefrontal regions compared to children, with an increasing engagement of other brain regions such as occipital and parietal areas. As brain maturation continues into and through adolescence, communication between functionally diverse regions becomes more efficient, which enables more attentional and sensory regions—in addition to prefrontal executive function regions—being recruited to assist with inhibitory process (Velanova et al., 2008; Spielberg, Miller, Heller, & Banich, 2015). Another plausible explanation is that as age increases, adolescents have a greater tendency toward regulating their own behaviors due to socialization, and gain more practice with cognitive control; together, these help to shape the structural and functional changes of prefrontal regions related to inhibitory processes (Schore, 1996).

However, there also are studies pointing to higher neural activity in the frontal lobes of adults compared to children or adolescents (Adleman et al. 2002; Andrews-Hanna et al., 2011; Bunge et al. 2002; Rubia et al. 2000; Rubia et al. 2006), or nonlinear developmental changes for different prefrontal regions (Liu et al., 2016). It should be noted that direct comparison across studies is complicated given the differences in IC tasks, types of cognitive control measured, and fMRI designs and analysis methods (Andrews-Hanna et al., 2011).

**Family Socioeconomic Status, Parenting Behaviors and Adolescent Inhibitory Control Development**

The second main goal of the current investigation (testing hypotheses 2-3) was to examine the association among family SES, parenting behaviors and the development of
IC during adolescence. Even though the direct association between family SES and adolescent IC at any given age was not significant in either study sample, evidence was found for both statistical mediation (Figure 1, paths ‘b’ and ‘c’) and moderation (Figure 1, path ‘d’) by parenting behaviors, of the link between family SES and adolescent IC development. In the early adolescent study sample, significant indirect effects were found for family SES at age 9 on IC at age 13 via parenting behaviors at age 10; lower family SES at age 9 was related to less parental warmth and greater parental rejection at age 10, which in turn was associated with lower IC task performance at age 13. This finding is consistent with the family stress model and several prior studies, suggesting that parenting behavior is an important social factor that links child IC development with more distal family context factors such as family income and parental education level (Blair et al., 2011; Holochwost et al., 2016; Sarsour et al., 2011). Lower family SES, which is often characterized by higher economic stress and lower social prestige, may affect child and adolescent IC development by influencing parenting quality (Fatima et al., 2016; Hackman et al., 2015; Sosic-Vasic et al., 2017). Economic stress and chaotic environments associated with lower family SES may overwhelm parents and affect the emotional, verbal and behavioral responsiveness of parents to their children. In these highly stressed families, parents may not be able to provide time and effort to reinforce desired behaviors and encourage IC development in their children and adolescents.

One advantage of the early adolescent study sample is that roughly equal number of White, African American and Hispanic families were recruited, which allows for statistically separating any potential SES effects from race/ethnicity effects, by controlling for the effects of ethnicity while testing the effects of family SES on IC
Results revealed that when statistically controlling for the potential confound between SES and family race/ethnicity, the above results were still obtained. Specifically, the indirect effects of family SES on the development of adolescent IC via parenting practices were independent of race/ethnicity variation, even though ethnicity was associated with SES (i.e. White families had significantly higher incomes and parent education levels than African American families, whose SES was significantly higher than the Hispanic families). The current finding is in line with results found for other EF capacities in younger children (Hackman et al., 2015), and extends prior literature by utilizing balanced proportions of members of various racial/ethnic groups and tease apart the specific SES effects from the effects of ethnic background in adolescence (Hackman & Farah, 2009).

Another possible mechanism for the family SES-parenting-IC development link is through passive gene-environment correlation, that is, child’s inherited genotype may be related to their rearing environment. Specifically, the genes that contribute to better child IC may be inherited from parents with better IC. The genes also contribute to higher parental education, higher family income and better caregiving behaviors (Bridgett et al., 2015). However, the current study design is not able to completely rule out the possibility of passive gene-environment correlation, since parents provide both genes and rearing environments. Future studies using adoption designs or cross-fostering animal study designs are needed to test the effects of family SES, parenting on youth IC development while controlling for passive gene-environment correlation.

Results from the middle adolescent study sample suggested that the link between family SES at 14-years of age and IC development from 15 to 17 years of age—and the
neural functioning measure--was statistically moderated by several parenting dimensions. Specifically, a consistent association between family SES at age 14 and the initial level of IC neural activity (age 15) was found only for adolescents who were exposed to lower parental warmth, lower parental monitoring and higher parental rejection. In contrast, family SES was not related to the initial level of IC neural activity among adolescents whose parents were warm, supportive and non-rejecting. One pathway linking family SES to the neural correlates of IC is adolescent’s stress response system. It is plausible that family stress strains adolescent’s stress response system, which in turn impacts the neural functioning related to IC development in prefrontal cortex, which also has a high concentration of glucocorticoid receptors (Arnsten, 2009; Blair et al., 2005; Evans & Schamberg, 2009). This pathway may be especially salient for adolescents who do not receive warm, supportive parenting or who are exposed to high levels of rejection. In contrast, warm and supportive parenting and guidance from parents may mitigate family stress and buffer adolescents from the negative effects of socioeconomic stress on their stress response system.

Results from the early and middle adolescent study samples did not contradict each other. Indeed, the two studies’ sets of results complement each other, and highlight the complexity of the mechanism underlying individual differences in IC development. The current investigation also highlights the significance of parenting behaviors in understanding the link between family SES and behavioral and neural function of IC during adolescence. The findings are added to a growing literature proposing that the mechanisms reflected in the tested statistical 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; Holochwost et al., 2016; Sarsour et al., 2011). Therefore, it is critical to conduct analyses at multiple levels to better understand the multi-faceted and complex nature of IC. Future longitudinal studies measuring behavioral performance and BOLD responses using different IC 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 IC.

Despite evidence suggesting that various parenting behaviors may play different roles in children's and adolescents’ development, relatively few investigations have examined parental warmth, parental rejection and parental monitoring within a single study. The results generally supported the important roles of parental warmth and parental rejection in mediating the link between family SES and IC task performance. Furthermore, warmth, rejection and monitoring all moderated the link between family SES and the development of neural functioning related to IC.

In both study samples, the effects of family SES (a composite score of family income and parental education) was tested, and then the models were reran with the income and parental education indicators of SES separated out, in order to determine whether the effects were due to specific SES variables. Results suggested that the mediation effects (family SES - parenting - adolescent IC) in the early adolescent sample and the moderation effects (family SES - by – parenting on adolescent IC) in the middle adolescent sample were not driven by one indicator. Rather, it is the global family socioeconomic status that influences adolescent IC development. The current results highlighted the importance of measuring family SES using multiple inter-related
indicators. For a measurement perspective, a SES composite score based on family income and parental education are more consistent and reliable, and generally have stronger predictive validity (Rushton, Brainerd, & Pressley, 1983).

**Limitations and Future Directions**

The current investigation’s results should be considered in light of several limitations. First, different IC tasks and different measures of parenting practices were employed in the two study samples, which prevented directly comparing the results across the two studies and delineating the developmental changes of IC from late childhood to middle adolescence directly by combining the two samples. Different results from the two study samples could be due to distinct developmental stages, or differences in measures and tasks.

Second, in both samples, adolescent-reported parenting behaviors showed only small to modest correlations with parents’ self-reported parenting practices. The discrepancy may indicate developmental separation between parents and adolescents. It could also indicate problematic parent–child communication (De Los Reyes & Kazdin, 2005; Ehrlich, Cassidy, & Dykas, 2011; Guion et al. 2009). The low correlation also raises questions about reliability of youth self-report, and it did not allow for testing of more reliable multi-informant composite scores for parenting behaviors.

Third, in the current investigation, we focused on different parenting behaviors as important proximal factors in explaining the link between family SES and adolescent IC. Effects of other social factors, such as peer relationship, sibling interactions, physical home environment, learning materials, and neighborhood socioeconomic disadvantage are also worth exploring. Moreover, adolescents spend more time outside of family and
peer relations become very salient during adolescence (Brown & Bakken, 2011). Peer relationship has been found as a very important contextual factor influencing adolescent’s emotion, behavior and decision-making. For instance, peer acceptance has been found as a valuable source of social support for individuals, and peer problems may contribute to lower EF, especially in the late childhood and early adolescence (Holmes et al., 2016).

Fourth, family SES in the current studies were measured using family income-to-needs ratio and parental education. Family income was reported as an interval variable with non-regular spacing and was then transformed into a continuous variable through rough approximation, which may obscure the nonlinear nature of the construct. Future research may benefit from directly measuring income using exact value. It might also benefit from adopting a broader concept of family SES and measuring not only family income and parental education, but also subjective notions of status, for instance, individual’s perception of their status relative to others in their community, or satisfaction/worry of family financial situation.

Finally, as mentioned above, our models are not able to eliminate the possibility of passive gene-environment correlation, since parents provide both genes and rearing environments. It is possible that the genes that contribute to better parent IC may also contribute to higher parental education, higher family income and better caregiving behaviors. Meanwhile, their child may inherit the genes and show better IC (Bridgett et al., 2015). Future studies using adoption designs or cross-fostering animal study designs are needed to test the effects of family SES, parenting on youth IC development while controlling for passive gene-environment correlation.
Future study would also benefit from formally testing potential gender effects on IC development during adolescence, given that prior literature suggested gender-specific pathways in inhibition development in adults (Liu, Zubieta, & Heitzeg, 2012). Though not proposed in the current study, gender effect was tested as a covariate in all the models in both study samples as supplemental analyses. In the first study sample, four paths from gender to IC indicators and parenting behaviors at both waves were added into the mediation models, and none of the paths was significant. In the second study sample, three paths from gender to parenting behavior, and intercept and slope of the IC indicators, and none of the paths was significant either. The results suggested no gender effect on IC development during adolescent. It is worthwhile for future studies with larger sample size to formally test the gender effects on adolescent IC development using multiple group comparison in structural equation modeling.

**Strengths**

Despite the limitations, the current investigation contributes to the extant literature in several key ways. First, the two longitudinal samples employed in the current investigation span late childhood to middle adolescence, which provides a unique opportunity to examine the development of adolescent IC across a much wider age range and investigate a broader set of research questions. Second, the large multiple-wave longitudinal samples used in the early adolescence and middle adolescence studies provided enough statistical power to systematically investigate the developmental trajectories and statistical predictors of IC development. Third, the current investigation is the first to test the additive and interactive statistical effects of family SES and parenting behaviors on the development of behavioral IC and neural functioning related
to IC in adolescence. Findings suggest that family SES, which was measured prior to parenting behaviors and IC performance were measured, showed persistent associations with adolescents’ IC development one to three/four years later. The results also provide strong evidence that parenting practices play important roles during adolescence, even though it is during this time in development that youth are known to spend more time outside the family (Brown & Bakken, 2011). These findings could potentially inform future prevention and intervention work to improve effective parenting practices and enhance adolescents’ cognitive control, especially for adolescents from socioeconomically disadvantaged families. Fourth, three different dimensions of parenting practices (i.e. parental warmth, parental rejection and parental monitoring) were tested in the current investigation. Though correlated with each other, these three dimensions showed significant statistical mediating effects on the association between family SES and youth behavioral IC development and significant moderating effects on the association between family SES and neural correlates of IC. Furthermore, unlike many of the prior studies that focused solely on maternal behaviors and youth IC, the current investigation included adolescents’ perceptions of both mothers’ and fathers’ parenting—providing a broader index of parenting behaviors in the home. Finally, in the early adolescent study sample, oversampling of African Americans and Hispanics allowed consideration of potential racial and ethnic group differences into the model, providing a novel opportunity to distinguish its potential effects from family SES. Results revealed that it is family income and parental education, rather than ethnicity, that interacted with parenting behaviors to influence the development of behavioral IC during early adolescence.
Conclusion

Results from two longitudinal study samples spanning 9 to 17 years of age echoed the findings from prior cross-sectional studies. IC task performance continued to improve linearly from early to middle adolescence. Neural activity in pre-SMA brain regions decreased across middle adolescence, suggesting that neural processing during IC task performance becomes more efficient across adolescence. The current study also broadens understanding of how family SES and various parenting behaviors can shape the development of behavioral performance and neural functioning related to IC during adolescence. Instead of directly predicting IC, family SES was indirectly related to adolescent IC behavioral performance improvements through its statistical association with several parenting behaviors. Moreover, the direct link between family SES and neural functioning of IC was statistically moderated by parenting, with effects being largest for youth with the least supportive and most rejecting parenting environments.

The results highlight the critical importance of conducting analyses at multiple levels of IC and evaluating different contextual factors in order to better understand the complex nature of IC development. Identifying the specific parenting behaviors as important mechanisms in the link between family SES and adolescent IC development may inform prevention and intervention programs for improving adolescent IC and self-regulation across a wide range of family SES. Our findings suggest that it is not just family SES or parenting that lead to IC development. The mechanism underlying the association among family SES, parenting and youth cognitive capacities is rather complex. Given the importance of family SES and parenting for youth development, it may be important for intervention and prevention work to consider both contextual factors. Policies that aim to
reduce family poverty may have meaningful effects on influencing parenting practices and improving youths’ brain functioning and cognitive development. Moreover, home-based interventions that help improving parenting behaviors may also provide cognitive and social benefits for youth facing socioeconomic adversity.
Table 1.
Descriptive statistics and bivariate correlations of the main study variables in the early adolescent sample

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Note. Gender: 0 = Male; 1 = Female; W2 = Wave 2, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6, RT = Reaction time.

** p < .01, * p < .05
Table 2.
Descriptive statistics and bivariate correlations of the main study variables in the middle adolescent study sample

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\[ .77 \]

\[ .56 \]

\[ .60 \]

Note. Gender: 0 = Male; 1 = Female; W1 = Wave 1, W2 = Wave 2, W3 = Wave 3, W4 = Wave 4.

**p < .01, *p < .05, †p < .10.
Table 2 (cont.)
Descriptive statistics and bivariate correlations of the main study variables in the middle adolescent study sample

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Note. W1 = Wave 1, W2 = Wave 2, W3 = Wave 3, W4 = Wave 4, MSIT = Multi-source interference task, RT = Reaction time.
** p < .01, * p < .05.
Table 2 (cont.)
Descriptive statistics and bivariate correlations of the main study variables in the middle adolescent study sample

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<td>.06</td>
<td>.26**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>m6_11_49</td>
<td>-.09</td>
<td>-.08</td>
<td>-.19</td>
<td>-.27**</td>
<td>.02</td>
<td>-.03</td>
<td>.21*</td>
<td>.19*</td>
<td>.15</td>
<td>.10</td>
<td>.36**</td>
<td>.42**</td>
<td>1</td>
</tr>
<tr>
<td>27</td>
<td>m6_14_46</td>
<td>-.01</td>
<td>-.23*</td>
<td>-.22*</td>
<td>-.15</td>
<td>.08</td>
<td>-.14</td>
<td>.20*</td>
<td>.11</td>
<td>.07</td>
<td>.05</td>
<td>.35**</td>
<td>.29**</td>
<td>.35**</td>
</tr>
</tbody>
</table>

*Note.* W1 = Wave 1, W2 = Wave 2, W3 = Wave 3, W4 = Wave 4, MSIT = Multi source interference task, RT = Reaction time.

**p < .01, * p < .05.
Table 3.
Latent growth modeling results for behavioral inhibitory control in the middle adolescent sample

<table>
<thead>
<tr>
<th></th>
<th>IC Accuracy W1 - W4</th>
<th>IC RT W1 - W4</th>
<th>Pre-SMA W1 – W4</th>
<th>IC Accuracy W2 - W4</th>
<th>IC RT W2 - W4</th>
<th>Pre-SMA W2 – W4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>-.098***</td>
<td>.479***</td>
<td>.82***</td>
<td>-.044***</td>
<td>.405***</td>
<td>.51***</td>
</tr>
<tr>
<td>Slope</td>
<td>.053***</td>
<td>-.075***</td>
<td>-.21</td>
<td>.016***</td>
<td>-.034***</td>
<td>-.04*</td>
</tr>
<tr>
<td>Quadratic</td>
<td>-.002</td>
<td>.000</td>
<td>-.11</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Variance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>.003**</td>
<td>.004**</td>
<td>.17***</td>
<td>.001**</td>
<td>.004**</td>
<td>-.05**</td>
</tr>
<tr>
<td>Slope</td>
<td>.001*</td>
<td>.001*</td>
<td>.95</td>
<td>.001*</td>
<td>.001*</td>
<td>-.01</td>
</tr>
<tr>
<td>Quadratic</td>
<td>.002</td>
<td>.000</td>
<td>.54</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Covariance</td>
<td>-.002**</td>
<td>-.60*</td>
<td>-.21*</td>
<td>.001*</td>
<td>-.001*</td>
<td>-.01</td>
</tr>
</tbody>
</table>

Note. W1 = Wave 1, W2 = Wave 2, W4 = Wave 4, MSIT = Multi source interference task, RT = Reaction time.
*** p < .001, ** p < .01, * p < .05.
Table 4.
Effect of family SES on the development of IC moderated by parenting behaviors in the middle adolescent sample

<table>
<thead>
<tr>
<th></th>
<th>Accuracy</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>RT</th>
<th></th>
<th></th>
<th></th>
<th>pre-SMA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intercept</td>
<td>Slope</td>
<td>Intercept</td>
<td>Slope</td>
<td>Intercept</td>
<td>Slope</td>
<td>Intercept</td>
<td>Slope</td>
<td>Intercept</td>
<td>Slope</td>
</tr>
<tr>
<td>SES</td>
<td>.003</td>
<td>.003</td>
<td>.00</td>
<td>.002</td>
<td>-.006</td>
<td>.006</td>
<td>-.002</td>
<td>.003</td>
<td>-.009</td>
<td>.027</td>
</tr>
<tr>
<td>SES X Parental warmth</td>
<td>.007</td>
<td>.003</td>
<td>-.003</td>
<td>.002</td>
<td>-.011*</td>
<td>.006</td>
<td>.005+</td>
<td>.003</td>
<td>.005</td>
<td>.027</td>
</tr>
<tr>
<td>SES X Parental hostility</td>
<td>-.002</td>
<td>.003</td>
<td>.001</td>
<td>.002</td>
<td>.004</td>
<td>.006</td>
<td>.001</td>
<td>.003</td>
<td>.02</td>
<td>.028</td>
</tr>
<tr>
<td>SES X Parental monitoring</td>
<td>.003</td>
<td>.003</td>
<td>.000</td>
<td>.002</td>
<td>-.009</td>
<td>.005</td>
<td>.001</td>
<td>.003</td>
<td>-.013</td>
<td>.028</td>
</tr>
</tbody>
</table>

Note. SES = Socioeconomic Status, RT = reaction time
*** p < .001, ** p < .01, * p < .05
Figure 1. Heuristic models for the mediation and moderation mechanisms.
Note. The Upper model is the heuristic model of the mediation mechanism. Lower model is the heuristic model of the moderation mechanism.
Figure 2. Color-word Stroop task.

Note. Color-word Stroop task requires participants to press the button that matches the color in which the word was printed (i.e., its “ink”), and ignoring the meaning of the word.
Figure 3. Conceptual model for the mediation path model in the early adolescent sample.

Note: SES = Socioeconomic status, IC = Inhibitory control, W2 = Wave 2, W3 = Wave 3, W5 = Wave 5; W6 = Wave 6.
Figure 4. Conceptual model for the moderation models in the early adolescent sample.

Note: SES = Socioeconomic status, IC = Inhibitory control, W2 = Wave 2, W3 = Wave 3, W6 = Wave 6.
Figure 5. Standardized coefficients for Path analysis of family SES at wave 2, parental warmth at wave 3 and wave 5, and accuracy at wave 3 and wave 6 in the early adolescent sample.

Note: SES = Socioeconomic status, W2 = Wave 2, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6

** p < .01, * p < .05, † p < .10
Figure 6. Standardized coefficients for Path analysis of family SES at wave 2, parental warmth at wave 3 and wave 5, and reaction time at wave 3 and wave 6 in the early adolescent sample.

Note: SES = Socioeconomic status, W2 = Wave 2, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6

** p < .01, * p < .05, † p < .10
Figure 7. Standardized coefficients for Path analysis of family SES at wave 2, parental rejection at wave 3 and wave 5, and accuracy at wave 3 and wave 6 in the early adolescent sample.

Note: SES = Socioeconomic status, W2 = Wave 2, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6

**p < .01, *p < .05, †p < .10
Figure 8. Standardized coefficients for Path analysis of family SES at wave 2, parental rejection at wave 3 and wave 5, and reaction time at wave 3 and wave 6 in the early adolescent sample.

Note: SES = Socioeconomic status, W2 = Wave 2, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6

** p < .01, * p < .05, † p < .10
Figure 9. Standardized coefficients for Path analysis of family SES at wave 2, parental monitoring at wave 3 and wave 5, and accuracy at wave 3 and wave 6 in the early adolescent sample.

Note: SES = Socioeconomic status, W2 = Wave 2, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6

** p < .01, * p < .05, † p < .10
Figure 10. Standardized coefficients for Path analysis of family SES at wave 2, parental monitoring at wave 3 and wave 5, and reaction time at wave 3 and wave 6 in the early adolescent sample.

Note: SES = Socioeconomic status, W2 = Wave 2, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6

** $p < .01$, * $p < .05$, † $p < .10$
Figure 11. The Multi-source interference task.

“What is the different number?”
**Figure 12.** Conceptual model for the mediation models in the middle adolescent sample.  
*Note:* SES = Socioeconomic status, IC = Inhibitory control, W1 = Wave 1, W2 = Wave 2, W3 = Wave 3, W4 = Wave 4.
Figure 13. Conceptual model for the mediation model in the middle adolescent sample.  
*Note:* SES = Socioeconomic status, IC = Inhibitory control, W1 = Wave 1, W2 = Wave 2,  
Figure 14. Growth trends of MSIT accuracy for individual and sample-wide mean levels over the four time points.
Figure 15. Growth trends of MSIT reaction time for individual and sample-wide mean levels over the four time points.
Figure 16. Growth trends of pre-SMA activities for individual and sample-wide mean levels over the four time-points.
APPENDIX A

PARENTAL AGE AND EDUCATION ACROSS ETHNICITY IN EARLY ADOLESCENT SAMPLE

As shown in the table below, ANOVAs indicated that parental age and education showed significant ethnic differences. Post-hoc analyses revealed that European American parents were significantly older and more highly educated than were African American and Hispanic American parents, and African American parents were significantly older in age and more highly educated than were Hispanic American parents.

Table A
Descriptive statistics and ANOVA of parental age and education across ethnicity.

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Mother age M(SD)</th>
<th>Father age M(SD)</th>
<th>Mother education M(SD)</th>
<th>Father education M(SD)</th>
<th>Stroop Accuracy W3 M(SD)</th>
<th>Stroop Accuracy W6 M(SD)</th>
<th>Stroop RT W3 M(SD)</th>
<th>Stroop RT W6 M(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>European American</td>
<td>41.94 (6.61)</td>
<td>43.45 (5.96)</td>
<td>17.00 (2.80)</td>
<td>17.19 (2.90)</td>
<td>.90 (.07)</td>
<td>.95 (.07)</td>
<td>939.2</td>
<td>803.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(104.5)</td>
<td>(96.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>37.80 (8.37)</td>
<td>39.84 (8.44)</td>
<td>13.81 (2.06)</td>
<td>13.45 (2.26)</td>
<td>.86 (.07)</td>
<td>.94 (.06)</td>
<td>972.1</td>
<td>879.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(109.5)</td>
<td>(120.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic American</td>
<td>34.39 (6.05)</td>
<td>37.24 (7.26)</td>
<td>9.99 (4.45)</td>
<td>10.36 (4.21)</td>
<td>.87 (.06)</td>
<td>.92 (.08)</td>
<td>945.8</td>
<td>871.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(104.9)</td>
<td>(96.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F(df)</td>
<td>26.55***</td>
<td>15.73***</td>
<td>94.00***</td>
<td>74.05***</td>
<td>2.31</td>
<td>2.26</td>
<td>1.84</td>
<td>9.64***</td>
</tr>
</tbody>
</table>

*Note. RT = Reaction Time, W3 = Wave 3, W6 = Wave 6; *** p < .001.*
APPENDIX B

PATH ANALYSES RESULTS FOR SES INDICATORS IN EARLY ADOLESCENT SAMPLE

Path analyses were conducted to further examine the associations among SES indicators, parenting and youth IC, with the income (Table B.1) and parental education (Table B.2) indicators of SES separated out, in order to determine whether the effects were due to specific SES variables. Unstandardized coefficient for each path in the models were presented below.

Table B.1
Unstandardized coefficients for the longitudinal associations among family income, parenting behaviors and IC.

<table>
<thead>
<tr>
<th></th>
<th>Parental warmth</th>
<th>Parental Rejection</th>
<th>Parental monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IC accuracy b(SE)</td>
<td>IC RT b(SE)</td>
<td>IC accuracy b(SE)</td>
</tr>
<tr>
<td><strong>Direct effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income W2 → Parenting W3</td>
<td>.18(.06)**</td>
<td>.18(.06)**</td>
<td>-.17(.06)**</td>
</tr>
<tr>
<td>Income W2 → IC W3</td>
<td>.09(.07)</td>
<td>-.09(.07)</td>
<td>.09(.07)</td>
</tr>
<tr>
<td>Income W2 → Parenting W5</td>
<td>.05(.06)</td>
<td>.07(.07)</td>
<td>.03(.06)</td>
</tr>
<tr>
<td>Income W2 → IC W6</td>
<td>.12(.05)*</td>
<td>-.22(.07)**</td>
<td>.12(.06)*</td>
</tr>
<tr>
<td>Parenting W3 → Parenting W5</td>
<td>.37(.07)**</td>
<td>.36(.07)**</td>
<td>.54(.07)**</td>
</tr>
<tr>
<td>IC W3 → IC W6</td>
<td>.16(.06)**</td>
<td>.38(.07)**</td>
<td>.17(.05)**</td>
</tr>
<tr>
<td>Parenting W3 → IC W6</td>
<td>.10(.06)</td>
<td>-.03(.08)</td>
<td>-.11(.06)†</td>
</tr>
<tr>
<td>IC W3 → Parenting W5</td>
<td>.11(.07)</td>
<td>.05(.07)</td>
<td>-.09(.08)</td>
</tr>
<tr>
<td><strong>Indirect effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income W2 → Parenting W3 → IC W6</td>
<td>.18(.01)*</td>
<td>.01(.02)</td>
<td>.02(.01)</td>
</tr>
<tr>
<td>Income W2 → IC W3 → Parenting W5</td>
<td>.01(.01)</td>
<td>-.01(.01)</td>
<td>-.01(.01)</td>
</tr>
</tbody>
</table>

*Note. IC = Inhibitory control, RT = reaction time, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6
*p < .05, **p < .01, ***p < .001, †p < .10.
Table B.2
Unstandardized coefficients for the longitudinal associations among parental education, parenting behaviors and IC.

<table>
<thead>
<tr>
<th></th>
<th>Parental warmth</th>
<th>Parental Rejection</th>
<th>Parental monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IC accuracy</td>
<td>IC RT</td>
<td>IC accuracy</td>
</tr>
<tr>
<td>Direct effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education W2 → Parenting W3</td>
<td>.20(.06)**</td>
<td>.20(.06)**</td>
<td>-.12(.06)*</td>
</tr>
<tr>
<td>Education W2 → IC W3</td>
<td>.03(.07)</td>
<td>.04(.07)</td>
<td>.03(.07)</td>
</tr>
<tr>
<td>Education W2 → Parenting W5</td>
<td>.05(.07)</td>
<td>.06(.06)</td>
<td>.07(.06)</td>
</tr>
<tr>
<td>Education W2 → IC W6</td>
<td>.03(.05)</td>
<td>-.09(.07)</td>
<td>.03(.05)</td>
</tr>
<tr>
<td>Parenting W3 → Parenting W5</td>
<td>.36(.07)***</td>
<td>.36(.07)***</td>
<td>.34(.07)***</td>
</tr>
<tr>
<td>IC W3 → IC W6</td>
<td>.14(.06)*</td>
<td>.42(.07)***</td>
<td>.14(.06)*</td>
</tr>
<tr>
<td>Parenting W3 → IC W6</td>
<td>.09(.06)</td>
<td>-.04(.08)</td>
<td>-.10(.06)†</td>
</tr>
<tr>
<td>IC W3 → Parenting W5</td>
<td>.11(.06)</td>
<td>.03(.06)</td>
<td>-.05(.05)</td>
</tr>
</tbody>
</table>

Indirect effects
<table>
<thead>
<tr>
<th></th>
<th>Parental warmth</th>
<th>Parental Rejection</th>
<th>Parental monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IC accuracy</td>
<td>IC RT</td>
<td>IC accuracy</td>
</tr>
<tr>
<td>Education W2 → Parenting W3 → IC W6</td>
<td>.02(.01)</td>
<td>-.01(.02)</td>
<td>.01(.01)</td>
</tr>
<tr>
<td>Education W2 → IC W3 → Parenting W5</td>
<td>.01(.01)</td>
<td>.00(.01)</td>
<td>.00(.01)</td>
</tr>
</tbody>
</table>

Note. IC = Inhibitory control, RT = reaction time, W3 = Wave 3, W5 = Wave 5, W6 = Wave 6
* p < .05, ** p < .01, *** p < .001, † p < .10.
Appendix C

The main and interaction effects of family SES and parenting on youth IC in early adolescent sample

Regression analysis were conducted to test the main and interactive effects of family SES at wave 2 and parenting behaviors at wave 3 (i.e. parental warmth, rejection and monitoring) on adolescent Stroop accuracy and reaction time at wave 3, wave 6 and the changes between two waves (using residualized change scores by regressing Stroop accuracy/reaction time at wave 6 on Stroop accuracy/reaction time at wave 3). Detailed results are presented in the table below.

Table C
Cross-Sectional and Longitudinal Analyses for the main and interaction effects of family SES and parenting behaviors on youth IC.

<table>
<thead>
<tr>
<th></th>
<th>Accuracy W3</th>
<th>Accuracy W6</th>
<th>Δ Accuracy</th>
<th>RT W3</th>
<th>RT W6</th>
<th>Δ RT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b(SE)</td>
<td>b(SE)</td>
<td>b(SE)</td>
<td>b(SE)</td>
<td>b(SE)</td>
<td>b(SE)</td>
</tr>
<tr>
<td>SES W2</td>
<td>.05(.07)</td>
<td>.06(.06)</td>
<td>.01(.01)</td>
<td>.03(.07)</td>
<td>-.14(.08)^†</td>
<td>-.17(.08)^*</td>
</tr>
<tr>
<td>Parental warmth W3</td>
<td>.01(.08)</td>
<td>.08(.06)</td>
<td>.01(.01)</td>
<td>.06(.09)</td>
<td>-.04(.09)</td>
<td>.00(.09)</td>
</tr>
<tr>
<td>SES X Parental warmth</td>
<td>.09(.06)</td>
<td>-.02(.04)</td>
<td>-.01(.00)</td>
<td>-.06(.11)</td>
<td>-.03(.06)</td>
<td>-.06(.06)</td>
</tr>
<tr>
<td>F (3, 212)</td>
<td>1.00</td>
<td>1.65</td>
<td>2.58</td>
<td>.27</td>
<td>1.28</td>
<td>1.74</td>
</tr>
<tr>
<td>SES W2</td>
<td>.03(.07)</td>
<td>.06(.05)</td>
<td>.01(.01)</td>
<td>.02(.07)</td>
<td>-.12(.08)^†</td>
<td>-.14(.08)^†</td>
</tr>
<tr>
<td>Parental rejection W3</td>
<td>-.02(.09)</td>
<td>-.10(.07)</td>
<td>-.01(.01)</td>
<td>.04(.09)</td>
<td>.15(.09)</td>
<td>.15(.09)</td>
</tr>
<tr>
<td>SES X Parental rejection</td>
<td>-.03(.06)</td>
<td>.03(.05)</td>
<td>.00(.00)</td>
<td>.03(.06)</td>
<td>.06(.07)</td>
<td>.09(.06)</td>
</tr>
<tr>
<td>F (3, 212)</td>
<td>.13</td>
<td>2.23</td>
<td>2.83</td>
<td>.11</td>
<td>2.08</td>
<td>2.39</td>
</tr>
<tr>
<td>SES W2</td>
<td>.03(.07)</td>
<td>.08(.05)</td>
<td>.01(.01)^†</td>
<td>-.01(.07)</td>
<td>-.13(.08)^†</td>
<td>-.14(.08)^†</td>
</tr>
<tr>
<td>Parental monitoring W3</td>
<td>-.05(.07)</td>
<td>-.02(.06)</td>
<td>.00(.01)</td>
<td>.06(.07)</td>
<td>.09(.08)</td>
<td>.07(.08)</td>
</tr>
<tr>
<td>SES X Parental monitoring</td>
<td>-.07(.08)</td>
<td>-.06(.07)</td>
<td>-.01(.01)</td>
<td>.12(.08)</td>
<td>.09(.09)</td>
<td>.08(.09)</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
<td>----------</td>
<td>----------</td>
<td>----------</td>
</tr>
<tr>
<td>$F (3, 212)$</td>
<td>.44</td>
<td>1.24</td>
<td>1.41</td>
<td>.87</td>
<td>1.92</td>
<td>1.76</td>
</tr>
</tbody>
</table>

*Note.* SES = Socioeconomic status, RT = reaction time, W2 = Wave 2, W3 = Wave 3, W6 = Wave 6, Δ Accuracy = Change of accuracy from Wave 3 to Wave 6, Δ RT = Change of reaction time from Wave 3 to Wave 6.

* p < .05, ** p < .01, *** p < .001, † p < .10.
### APPENDIX D

**INTERFERENCE EFFECT IN THE MSIT FOR WAVE 1 - 4**

Table D.1
Areas of significant activation for the contrast of Interference minus Neutral blocks of the Multi-Source Interference Task at Wave 1

**Wave 1 MSIT Interference - Neutral**

<table>
<thead>
<tr>
<th>Cluster #</th>
<th>Region</th>
<th>Size</th>
<th>x</th>
<th>y</th>
<th>z</th>
<th>T</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>L Pre-Supplementary Motor Area</td>
<td>3082</td>
<td>-6</td>
<td>14</td>
<td>49</td>
<td>21.79</td>
</tr>
<tr>
<td></td>
<td>L Middle Frontal Gyrus</td>
<td></td>
<td>-27</td>
<td>-7</td>
<td>55</td>
<td>19.77</td>
</tr>
<tr>
<td></td>
<td>R Middle Frontal Gyrus</td>
<td></td>
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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 \( p < .001 \).
Table D.2
Areas of significant activation for the contrast of Interference minus Neutral blocks of the Multi-Source Interference Task at Wave 2

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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 $p < .001$. 


Table D.3
Areas of significant activation for the contrast of Interference minus Neutral blocks of the Multi-Source Interference Task at Wave 3

Wave 3 MSIT Interference - Neutral

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*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 \( p < .001 \).
Table D.4
Areas of significant activation for the contrast of Interference minus Neutral blocks of the Multi-Source Interference Task at Wave 4

Wave 4 MSIT Interference - Neutral

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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 $p < .001$. 

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APPENDIX E

INDIRECT EFFECTS OF FAMILY SES ON IC VIA PARENTING IN THE MIDDLE ADOLESCENT SAMPLE

The indirect effects of family income and parental education on the development of IC via more proximal family factors, such as parenting behaviors were examined. Three parenting behaviors (i.e. parental warmth, hostility and monitoring) were tested separately and the results were presented in the table below.

Table E
Indirect effects of family SES on adolescent IC development via different parenting behaviors

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<td>.001</td>
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Note. SES = Socioeconomic status, RT = reaction time
*p < .05, **p < .01, ***p < .001, †p < .10
BIBLIOGRAPHY


Giorgio, A., Watkins, K. E., Chadwick, M., James, S., Winmill, L., Douaud, G., ... & James, A. C. (2010). Longitudinal changes in grey and white matter during adolescence. *Neuroimage,* 49(1), 94-103. doi:10.1016/j.neuroimage.2009.08.003


doi:10.1017/S0954579412000405


doi:10.1080/15295192.2011.585552


doi:10.1007/s10826-019-01411-x

doi:10.1177/0022219407311748


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143


147


