ABSTRACT

Title of Dissertation:	DEVELOPMENT OF MOTIVATIONAL INFLUENCES ON MONITORING AND CONTROL RECRUITMENT IN THE CONTEXT OF PROACTIVE AND REACTIVE CONTROL IN ADOLESCENT MALES
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Adolescence and the onset of puberty is a time period of physiological and behavioral changes that include a heightened reward sensitivity, but underdeveloped cognitive control. Cognitive control involves monitoring for salient stimuli and recruiting control to adapt behavior advantageously to reach a specific goal and is supported by the three domains of executive functioning (EF): inhibitory control, set-shifting, and working memory. Proactive control is engaged after an informative cue in preparation for an upcoming stimulus, while reactive control can be employed when preparation is not possible and you need to respond to a stimulus. Oscillations in the theta frequency (4-8Hz) during both cue presentation and stimulus presentation are implicated in proactive and reactive control processes. While reward has been shown to upregulate proactive control in adults, little work has assessed how reward

influences theta oscillations during both proactive and reactive control throughout adolescence and pubertal development. Further, no work has sought to understand how EF abilities bolster reward-related changes in proactive or reactive control. Here, 68 adolescent males ($Mean_{age}$=13.61, SD_{age}=2.52) aged 9 – 17 years old completed a rewarded cued flanker paradigm while electroencephalogram (EEG) was collected. They also completed tasks from the NIH toolbox that tap the three EF domains. Behaviorally, reward hindered performance on proactive trials, particularly in mid-puberty, while enhancing performance on reactive trials. Reward was associated with increases in cue-locked theta power, but with overall reductions in cue-locked theta ICPS. Stim-locked theta power increased on reactive trials with increasing age, while stim-locked theta ICPS peaked in mid-adolescence for rewarded trials. Increased cue theta power was associated with worse performance on proactive trials. On proactive trials, adolescents with low levels of inhibitory control experience more reward-related interference, while reward-related interference was mitigated by better set-shifting abilities only in younger and older adolescents. In conclusion, reward differentially impacts proactive and reactive control throughout adolescent development and EF influences the impact of reward on proactive control throughout adolescence.

DEVELOPMENT OF MOTIVATIONAL INFLUENCES ON MONITORING AND CONTROL RECRUITMENT IN THE CONTEXT OF PROACTIVE AND REACTIVE CONTROL IN ADOLESCENT MALES

by

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List of Abbreviations

TF = time frequency

ICPS = inter-channel phase synchrony

CSD = current source density

FE = flanker effect

RT = reaction time

EF = executive function

WM = working memory

IC = inhibitory control

SS = set-shifting

Chapter 1: Introduction

The ability to monitor one's environment and exert control over one's actions in order to exhibit goal-driven behavior is a hallmark of human cognition. Specifically, this ability is called cognitive control, a neurocognitive process by which we both *monitor* for salient stimuli and recruit subsequent *control* to advantageously adjust our behavior to reach said goal. Medial-lateral brain areas, such as the anterior cingulate cortex (ACC), are implicated in the monitoring of salient events, with increased activity in these areas acting as an "alarm signal" for important surrounding stimuli. Next, recruiting prefrontal areas has been associated with instantiating control over behavior in order to enact the appropriate response to said stimuli (Cavanagh & Frank, 2014). Extensive work using electroencephalography (EEG) in adults has characterized neural oscillations associated with these two underlying components of cognitive control, *monitoring* and *control recruitment*. Specifically, theta oscillations (i.e., power in 4-8 Hz), thought to be generated in the ACC, are associated with monitoring and detecting stimuli and are typically increased to salient events, such as conflict, novelty, or losses (Cavanagh, Zambrano-Vazquez, & Allen, 2012). Alternatively, phase synchrony of theta oscillations in medial areas with lateral frontal areas, is thought to reflect control recruitment (Cavanagh, Cohen, & Allen, 2009). Theta oscillations, measured with both theta power and theta phase synchrony, are thought to reflect organizing activity across disparate regions to implement cognitive control (Cavanagh & Frank, 2014).

Cognitive control can be strategically deployed on differing timescales: **proactively**, using informative cues to prepare for upcoming stimuli, or **reactively**,

responding reflexively upon the presentation of a stimulus in the absence of any cue. Proactive control is planful and preparatory, while reactive control is more impulsive or reflexive. Leveraging the fine temporal resolution of EEG, we can measure both *monitoring* and *control recruitment* at each of these timescales. Specifically, theta oscillatory dynamics to an informative cue can describe proactive control, while theta oscillatory dynamics after a non-cued stimulus can assess reactive control. Theta oscillations to both cue and stimulus events provide more sensitive measures of the neural mechanisms associated with both proactive and reactive control (Buzzell et al., 2019)

Our ability to utilize cognitive control improves throughout childhood and adolescence (Durston et al., 2002; Luna, 2009; Ordaz, Foran, Velanova, & Luna, 2013). Both proactive control and reactive control can be employed at an early age, but older children and adolescents are better at engaging proactive control (Elke & Wiebe, 2017; Munakata, Snyder, & Chatham, 2012). However, we know little about the development of monitoring (i.e., theta power) and control recruitment (i.e., theta phase synchrony) in the context of proactive and reactive control.

Importantly, the aim of employing cognitive control is to reach a specific goal and this goal must have some sort of motivating factor. One example of such a motivating factor is reward. Adolescence, a time period marked by underdeveloped cognitive control (Beatriz Luna, Padmanabhan, & O'Hearn, 2010), is also associated with an increase in reward sensitivity (Shulman et al., 2016). This imbalance between reward and cognitive control systems is implicated in the increase in risk-taking behaviors during the adolescent period (Clark et al., 2017). In particular, boys exhibit increased reward

sensitivity and risky behaviors in adolescence. Previous work has shown that, though adolescents typically exhibit deficits in cognitive control compared to adults (Ordaz, Davis, & Luna, 2010; Williams, et al., 1999), adolescents can upregulate their performance in the presence of reward to reach adult levels (Geier & Luna, 2009). In adults, reward specifically enhances proactive control (Chiew & Braver, 2016; Padmala & Pessoa, 2011). Yet, there is little work that investigates how reward influences both proactive and reactive control throughout development or if the influence of reward on cognitive control is specific to either monitoring or control recruitment.

Executive functions (EF), basic cognitive processes that include set shifting, inhibitory control, and working memory, also improve throughout development (Best & Miller, 2010; Huizinga, Dolan, & van der Molen, 2006). Inhibitory control is positively associated with reactive control, and prior work in our lab has shown that working memory has a positive relation with proactive control (Troller-Renfree, 2018) in children as young as five. Further, inhibitory control abilities relate to ERP measures of reactive control (i.e., N2; Lamm, Zelazo, & Lewis, 2006). In regards to proactive control, ERP measures of proactive control (i.e., P3b) mediate the relations between working memory and task performance, above and beyond age alone (Troller-Renfree, 2018). However, no work to date has examined reward-related neural measures of monitoring vs control recruitment that may mediate the relation between EF abilities and reward-related task performance in the context of proactive and reactive control across childhood and adolescence.

Given these critical gaps in the literature, the current study aims to, first, characterize the developmental differences in the influence of reward and control strategy

(proactive vs reactive) on two distinct mechanisms of cognitive control (monitoring and control recruitment) in a sample of pre-adolescent and adolescent males aged 9 – 17 years old. Second, the study examines relations between executive functions and reward-related proactive and reactive control throughout development. The analytic approach and developmental perspective of this dissertation will allow for a nuanced parsing of how reward affects proactive and reactive control in males across development and which executive functions bolster these processes, providing vital information related to optimal timing and potential targets for intervention for risky decision-making.

Chapter 2: Background

2.1 Cognitive Control

Cognitive control is the ability to both *monitor* for situations that require control, as well as subsequently *recruit control* in a situation-appropriate manner. Extensive prior work has characterized neural measures derived from EEG associated with both monitoring and control recruitment. In order to react to some salient event, including situations like increased conflict, committing an error, experiencing novelty, or getting feedback, the brain must monitor for these situations and detect that event. Utilizing the excellent temporal resolution of EEG, the monitoring and detection of these salient events is observed in negative-going Event-Related Potentials (ERPs), including the N2 (conflict and novelty detection), the ERN (error monitoring; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991), and the FRN (feedback; Holroyd & Coles, 2002). Though these ERPs are elicited in separate situations, they are all primarily generated by increases in theta band (4-8Hz) power in fronto-central areas (Cavanagh et al., 2012; Ullsperger, Fischer, Nigbur, & Endrass, 2014). Indeed, theta power over fronto-central electrodes is larger for high-conflict compared to low conflict trials, for example for no-go compared to go trials and for incongruent compared to congruent flanker stimuli (Nigbur, Ivanova, & Stürmer, 2011); theta power is larger for errors compared to correct trials on a flanker task in both adults (Bernat, Williams, & Gehring, 2005; Cavanagh, Cohen, & Allen, 2009; Hall, Bernat, & Patrick, 2007) and children (Albrecht et al., 2014; Buzzell et al., 2019). Finally, theta power is also larger for losses compared to gains in a gambling task in both adults (Bernat et al., 2015; Foti et al., 2015) and children (Bowers, Buzzell, Bernat, Fox, & Barker, 2018). Thus, salient stimuli are associated with increases

in theta power, which signals the need to take our brains off "autopilot" so that we can then adjust our behavior appropriately.

After the detection of salient events (e.g., feedback, errors, conflict) that signal a need to adjust behavior, control needs to be instantiated in order to perform the necessary behavioral adjustments. Control instantiation is typically associated with activation in lateral PFC (Miller, 2000) and increased connectivity to the PFC and within the PFC (Cole, Yarkoni, Repovš, Anticevic, & Braver, 2012). The mechanism behind the recruitment of control is thought to be the synchronization of neural activity (i.e., phase) between medial and lateral PFC regions, specifically in the theta frequency (Cavanagh, Cohen, & Allen, 2009; Kerns et al., 2004; van Driel, Ridderinkhof, & Cohen, 2012). Increased phase synchrony between regions in EEG is typically called inter-channel phase synchrony (ICPS). Indeed, theta ICPS is increased after many of the same salient stimuli that increase theta power, including errors (Buzzell et al., 2019; Cavanagh et al., 2009; van Driel et al., 2012), high conflict stimuli (Aviyente, Tootell, & Bernat, 2017; Choi, Jung, Kim, & Kim, 2010), and loss feedback (Watts, Tootell, Fix, Aviyente, & Bernat, 2018). Moreover, increased theta ICPS after errors predicts increased accuracy on the next trial (Buzzell et al., 2019; Cavanagh et al., 2009), suggesting that control has been engaged to adjust behavior. Monitoring, increased theta power, and control recruitment, increased phase synchrony in theta between medial-lateral sites, are important neural processes that both comprise cognitive control.

Dual Mechanisms of Cognitive Control

These neural processes, monitoring and control recruitment, can also be employed on two different timescales: reactive versus proactive cognitive control. The Dual

Mechanisms of Control Theory (Braver, 2012) postulates that *reactive control* involves processing information in a stimulus-driven and reflexive way; whereas, *proactive control* involves more future-oriented and planful strategies. For example, when driving on the highway, one can engage proactive control to prepare for an exit by utilizing highway signs that indicate that the exit is approaching on the right in two miles. On the other hand, individuals may need to employ reactive control if the driver of that car had missed those preparatory signs, but then sees that their exit is immediately on the right. Reactive control stems from the need to resolve interference or conflict, for example, between two stimuli or when needing to withhold a prepotent response (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Nigbur, Ivanova, & Stürmer, 2011). However, proactive control mechanisms utilize informative cues to prepare and reduce this interference for easier resolution of conflict and, thus, better performance. While these strategies are independent of one another, both can be engaged depending on task demands and costs associated with each strategy.

Monitoring and control recruitment, as indexed by increases in theta power and theta ICPS respectively, occur during both proactive and reactive control. During proactive control, informative cues that allow for preparation for an upcoming stimulus produce an increased theta power response in adults (Cavanagh et al., 2012; Cooper, Darriba, Karayanidis, & Barceló, 2016) and in adolescents (Mazaheri et al., 2014), providing a signal that increased control is needed to prepare for the subsequent stimulus. Increases in lateral prefrontal cortex (PFC) connectivity are also seen after informative cues that facilitate proactive control (Cooper et al., 2015). These findings suggest that informative cues upregulate monitoring (via increases in theta power) and proactive

control recruitment (via increases in medial-lateral phase synchrony), so that subjects can prepare to correctly respond. Moreover, proactive control is thought to be engaged after errors to improve performance on the next trial (Buzzell et al., 2019; Cavanagh et al., 2009). Errors, as compared to correct responses, have consistently elicited increases in theta power, showing increases in error monitoring (Cavanagh et al., 2009, 2012; Nigbur, Cohen, Ridderinkhof, & Stürmer, 2012; van Driel et al., 2012; Van Noordt, Campopiano, & Segalowitz, 2016). Additionally, increased theta connectivity after errors is associated with increased performance on the next trial, providing evidence for another situation in which proactive control is engaged to enhance performance (Buzzell et al., 2019). Both monitoring for a salient event, whether that event is an informative cue that enhances preparation for an upcoming stimulus or an internal signal that performance needs to be improved, and subsequent control are employed during proactive control.

In the absence of an informative cue, one must rely on reactive control. The appearance of the stimulus engages monitoring and subsequent reactive control recruitment in order to appropriately respond to the stimulus without preparation (Buzzell et al., 2019; Cooper et al., 2015). During reactive control, stimuli with increased levels of conflict (e.g., incongruent trials, nogo trials, stop trials on a stop-signal) elicit increased theta power (Hanslmayr et al., 2008; Harper, Malone, & Bernat, 2014; Lavallee, Meemken, Herrmann, & Huster, 2014). There is also evidence that theta ICPS between two regions after stimulus presentation is reflective of reactive control instantiation. For example, increased medial-lateral phase synchrony to the stimulus is associated with correct responses, reflecting the use of reactive control to respond appropriately. On the other hand, reduced medial-lateral theta phase synchrony to the stimulus control is seen

before error responses (Buzzell et al., 2019). While numerous studies have examined theta dynamics under various conditions and with different manipulations (e.g., Cavanagh, Cohen, & Allen, 2009b; Cavanagh & Frank, 2014; Gulbinaite, van Rijn, & Cohen,2014; Van Driel, Ridderinkhof, & Cohen, 2012), no study has examined both cue-locked and stimulus-locked theta dynamics during a paradigm that taps both proactive and reactive control. Parsing monitoring and control recruitment during proactive and reactive control allows for the investigation of the subprocesses of cognitive control to more fully understand neural mechanisms associated with dual mechanisms of cognitive control and could provide novel targets for intervention.

EEG as a Tool to Study Monitoring and Control Recruitment Throughout Development

Importantly, studies have investigated changes in cognitive control neural activity using Event-Related Potentials (ERPs) derived from EEG (Hoyniak, 2017; Lo, 2018). Traditional analyses of ERPs compute amplitude by averaging neural activity to individual trials, which isolates neural activity time-locked to an event of interest and assumes that the component of interest is temporally synchronous across trials (Luck, 2005). However, ERPs do have drawbacks that limit the ability to measure both *monitoring* and *control recruitment*. First, the averaging approach causes three issues. One, separate frequency bands differentially relate to constructs involved in executive functioning, broadly. As previously mentioned, theta is associated with monitoring for salient events like novelty, conflict, or errors (Cavanagh et al., 2012), processes integral to cognitive control. Other frequencies are implicated in processes like sustained attention (i.e., alpha power suppression; van Driel et al., 2012) and reward processing (i.e., delta power; Cavanagh, 2015). Averaging over these frequencies loses the fine-grained

information held in different frequency bands. Second, power in distinct frequency bands can show different patterns locked to your event of interest (e.g., increases in theta power, but suppression of delta power after feedback; Bernat, Nelson, & Baskin-Sommers, 2015), but can overlap temporally. Though theta power is thought to primarily contribute to ERPs like the N2 and ERN, work has shown that delta power contributes to these ERPs as well (Bernat, Nelson, Holroyd, Gehring, & Patrick, 2008; Harper et al., 2014). An increase in theta power and simultaneous decrease in delta power can diminish expected ERP amplitudes. Third, the latency of these ERPs can be variable and change throughout development (Lo, 2018). Indeed, age predicts latency of the N2 above and beyond other predictors (Lamm, Zelazo, & Lewis, 2006b), showing that ERP latencies change with age. Averaging over peaks at variable latencies will attenuate ERP amplitudes.

Thus, the approach of averaging ERP waveforms can remove the subtleties of cognitive function associated with different frequency bands and can blunt waveform amplitudes due to opposite effects in overlapping frequencies and latency jitter throughout development. As such, utilizing time-frequency (TF) measures can provide more information, including latency, amplitude, and coherence, across different frequency bands, allowing for more insight into complex cognitive control processes (Cooper et al., 2016) throughout development. Indeed, while the FRN, an ERP elicited by feedback, showed no association with age, age-related effects were seen in both the theta and delta bands, frequencies that contribute to the FRN, once separated using time-frequency PCA (Bowers et al., 2018). Thus, time-frequency methods revealed

developmental changes within both the theta and delta bands that were unable to be detected using ERPs alone.

Another drawback of standard approaches to computing ERPs is that ERPs are not measures of connectivity, limiting investigations of control recruitment. Functional connectivity measures from fMRI can assess connectivity between brain regions, but the poor temporal resolution of fMRI cannot separate monitoring from control recruitment. Therefore, the adult cognitive control literature widely uses medial-lateral prefrontal phase synchrony in EEG as a measure of connectivity to index rapid control recruitment during tasks (Cavanagh et al., 2009b; Cohen & Donner, 2013; Kerns et al., 2004; van Driel et al., 2012). However, this technique has not been widely applied in developmental studies, missing effects related to network connectivity, which has been shown in the fMRI literature to play an important role in cognitive control development. Indeed, a recent investigation of EEG dynamics during a flanker task in adolescents highlights that medial-lateral ICPS in the theta frequency was related to post-error behavioral adjustments, while theta power and inter-trial phase synchrony were not (Buzzell et al., 2019). Leveraging these more advanced TF and phase synchrony measures may elucidate more complex patterns of cognitive control functioning that are not observed with just ERPs alone.

Development of Cognitive Control

Cognitive control improves throughout childhood and adolescence as individuals improve their abilities to resolve conflict (e.g., withhold inappropriate responses or filter attention to avoid distraction). This improvement is evidenced by decreases in error rates and reaction times with increasing age (Bunge, Dudukovic, Thomason, Vaidya, &

Gabrieli, 2002; Luna et al., 2001; Ordaz, Foran, Velanova, & Luna, 2013; Troller-Renfree et al., 2019).

fMRI studies have investigated how brain activation is different in children, adolescents, and adults when completing cognitive control tasks. Children show activation in control-related regions at a young age, demonstrating that these processes are functioning relatively early in development (Luna et al., 2001; Padmanabhan, Geier, Ordaz, Teslovish & Luna, 2011). Monitoring and control recruitment processes engage a fronto-parietal network that includes the anterior cingulate cortex (ACC) in the medial pre-frontal cortex (mPFC), the supplementary motor area (SMA), the inferior frontal gyrus (IFG), and the lateral PFC (lPFC; Carter et al., 1998; MacDonald, Cohen, Stenger, & Carter, 2000; Ullsperger & von Cramon, 2001). Throughout development, activation in the ACC during cognitive control tasks increases (Andrews-Hanna et al., 2011; Ordaz et al., 2013). Moreover, activation in the ACC mediates relations between age and corrected-error rates on an antisaccade task (Ordaz et al., 2013), suggesting that the ACC maturation plays an important role in the development of cognitive control. The ACC is thought to play a role in monitoring and is the proposed generator for theta oscillations (Hanslmayr et al., 2008; Nigbur et al., 2011).

In addition to changes in the ACC, the activation of PFC during cognitive control tasks also undergoes substantial changes throughout development. In a flanker task, children displayed opposite interference-related prefrontal activation compared to adults and completely failed to activate a region in the right vlPFC (Bunge et al., 2002), showing immaturities in control-related regions. Moreover, cross-sectional studies have provided evidence that successful response inhibition is associated with increased

activation in prefrontal and parietal regions in children and adolescents compared to adults (Chevalier, Jackson, Revueltas Roux, Moriguchi, & Auyeung, 2019; Durston et al., 2002; Luna et al., 2001a). The interpretation of these results is that the decreased activation seen in prefrontal areas in adults reflects more efficient processing during inhibitory control tasks, whereas children and adolescents need more prefrontal activity to complete cognitive control tasks. In longitudinal studies, similar patterns of activation changes in the PFC are seen throughout development. In an accelerated cohort longitudinal design of participants aged 9 – 26, though motor area activation did not change with age in an antisaccade task, right dlPFC activation decreased with age (Ordaz et al., 2013), again suggesting that younger children activate frontal areas during a cognitive control task, but that frontal areas become more efficient as age increases. Though we see changes in activation in distinct regions of the brain during cognitive control throughout development, recent work has emphasized the importance of network integration metrics, such as functional connectivity and graph theoretic measures, to assess how these distinct regions, which all seem to play a role in cognitive control, interact throughout development (Luna, 2009; Somerville & Casey, 2010). Importantly, control networks transition from a focus on local structures to strengthened connections with more distal regions of the brain and this transition predicts improved cognitive control (Fair, Cohen, Power, Dosenbach, & Church, 2009; Stevens, Skudlarski, Pearlson, & Calhoun, 2009).

Currently, the majority of the work investigating the development of cognitive control has been done with fMRI. However, fMRI cannot parse the subprocesses of cognitive control described above (i.e., monitoring and control recruitment) due to its

poor temporal resolution. In order to understand the development of *monitoring* and *control recruitment*, EEG, with its excellent temporal resolution, must be used. EEG is an especially useful tool for investigating neural changes throughout childhood and adolescence, as children more readily cooperate with EEG nets compared to fMRI scanners, thus providing more usable data. Utilizing measures of theta oscillatory activity in developmental samples could provide evidence for developmental changes in more nuanced measures of cognitive control.

Development of Proactive and Reactive Control

Reiterating the thesis of the Dual Mechanisms of Cognitive Control Theory (Braver, 2012), cognitive control can be employed either proactively or reactively, and the ability to engage these strategies also changes throughout development. Reactive control is online at a young age, as children are able to respond to stimuli and complete basic tasks, such as a Go/Nogo task or stroop tasks (Grammer, Carrasco, Gehring, & Morrison, 2014; Prevor & Diamond, 2005) at age 2-3. Reactive control seems to improve linearly throughout childhood and adolescence (Durston et al., 2002; Luna et al., 2001). Depending on the task, children and adolescents reach adult levels of performance in early adolescence (e.g. Go/Nogo tasks; Huizinga, Dolan, & van der Molen, 2006) or later adolescence (e.g. antisaccade tasks; Ordaz et al., 2013). Similarly, proactive control seems to be present early in childhood, as young as 4-5 years old. (Munakata et al., 2012). Using a task-switching paradigm, 4-5 year olds and 7-8 year olds both show P3 responses on switch cues, suggesting that all ages are preparing for the upcoming switch behavior, but 7-8 year olds show larger P3 amplitudes (Elke & Wiebe, 2017), suggesting that older children are better at planning to adjust their behavior. Even at long preparation

intervals, children as young as 8 are able to maintain preparation for a trial during an antisaccade task (Ordaz, Davis, & Luna, 2010).

However, the ability to employ reactive or proactive control advantageously based on task demands changes with age. Indeed, in a task that engages both proactive and reactive control, five-year olds respond more reactively, but 8-9 year olds use a proactive strategy (Chevalier, Martis, Curran, & Munakata, 2015; Troller-Renfree, 2018; Chatham, Frank, & Munakata, 2009). When during a cue/gift task, 5 year olds did show an enhanced P3 response when a proactive control strategy was encouraged, but only 10 year olds showed an enhanced P3 response and enhanced pupillometric response when proactive control strategy was just possible (Chevalier et al., 2015).

Throughout adolescence and into adulthood, the ability to engage proactive control continues to improve. Compared to adults, adolescents had a blunted contingent negative variation (CNV), an ERP waveform that is associated with preparation before a cue (Killikelly & Szűcs, 2013). Improvements in proactive control throughout ages 10-25 were also associated with increased frontal activation and, critically, increased striatal-frontal connectivity when there is an increased probability of a stop-signal during a stop-signal task (Vink et al., 2014a), implying increased control recruitment when proactively preparing to inhibit a response. It is evident that neural activity associated with the engagement of proactive control develops throughout childhood and adulthood; however, we know little about the developmental changes in the subprocesses of cognitive control (e.g., theta dynamics) associated with sustaining proactive control after informative cues or with exercising reactive control when preparation is not possible.

2.2 Motivational Influences on Cognitive Control

Goal-directedness is a hallmark of behavior in a majority of organisms from planaria to humans. Cognitive control, how we advantageously adjust our behavior to reach goals, would not have a reason to operate if there was no motivating factor to reach said goal. Motivation is generally defined as external or internal incentives that change biological function to produce a noticeable change in behavior (Yee & Braver, 2018). Throughout development, what is considered an incentive and how incentives affect behavior can change. Incentives can take many forms, such as avoiding punishment or seeking praise, but one incentive of particular interest during the adolescent time period, is obtaining reward. Adolescence is a time period marked by heightened reward sensitivity and increased sensation seeking (Cauffman et al., 2010). While this reward-driven behavior can be adaptive for adolescents to explore their environment in preparation for adulthood, dysfunctional reward processing can also lead to potentially life-threatening situations, such as drug use and other risky behaviors (Spear, 2000), or affective disorders, such as depression (Forbes et al., 2010). Before discussing the effects of reward on cognitive control in adults and adolescents, the influence of different types of reward on adolescent reward processing are examined.

What is Rewarding for Adolescents vs Adults?

Two examples of particularly motivating incentives are social reward and monetary reward. Typically, research uses monetary reward during behavioral tasks as a simple incentive to perform well or as a reinforcer of correct behavior. However, in different developmental periods of life, adults and adolescents may find some incentives more motivating than others. For example, social reward could be more salient for

adolescents, who are in a period of their life in which peer interaction is increasingly frequent and important (Spear, 2000). On the other hand, monetary incentives may be more salient to adults, who generally have more expenses and debt, compared to adolescents. Subsequently, it is important to evaluate what kind of reward is the best motivator in these age ranges.

When comparing social and monetary reward in adults, ERP components in anticipation of these rewards, like the cue-P3 and CNV, are not different in morphology/topography or in latency. Further, consummatory ERPs upon the receipt of rewards, like the RewP and feedback-P3, were not different between monetary and social rewards. Both incentives sped up reaction times in comparison to a neutral condition, but cues for monetary reward elicited faster reaction times than cues for social reward (Ait Oumeziane, Schryer-Praga, & Foti, 2017).

Though children and adolescents have reported more motivation for social reward compared to monetary reward, both types of reward sped up reaction times in children, adolescents, and adults (Wang, Liu, & Shi, 2017). Moreover, neural responses in the STS/TPJ to both monetary and social reward showed no differences in adolescents (Flores et al., 2018), even when relating to a self-report measure of social closeness, providing evidence that social reward and monetary reward function similarly in adolescents.

These studies point to the fact that monetary and social rewards elicit similar neural responses in both adults and adolescents, alleviating concerns that money may not be as motivating as social reward in adolescence. However, it is important to also explore any differences between adolescents and adults in how subjectively incentivized they are

by money. When choosing their own compensation for study participation, studies have shown that there are no age-related differences in 10 to 20 year olds in whether they chose a gift card or cash or in what type of gift card was chosen (Paulsen, Hallquist, Geier, & Luna, 2015). Moreover, there were no age-related effects on subjective valuable of gift card or cash choice (Geier & Luna, 2012; Paulsen et al., 2015). Consequently, though multiple incentives can be rewarding, it seems that money is a sufficiently motivating factor from late childhood until adulthood.

Moreover, there are differences in reward sensitivity measured via self-report and behavioral paradigms between genders. Males report being more sensitive to rewards on self-report questionnaires (Li, Huang, Lin, & Sun, 2007; Torrubia, Avila, Molto, & Caseras, 2001; van Hemel-Ruiter, de Jong, Ostafin, & Wiers, 2015). Task-based evidence suggests that males are more affected by the magnitude of rewards and react more quickly than women to high reward conditions (Spreckelmeyer et al., 2009). Further, doses of testosterone given to young women shift responses on an IOWA gambling task to favor rewards and become more disadvantageous. These gender differences are also present in adolescence. Adolescent males make more risky decisions and show greater activity in the nucleus accumbens upon receipt of reward (Alarcón, Cservenka, & Nagel, 2017). Thus, there are important gender differences in reward sensitivity that need to be carefully considered.

Pubertal Influences on Reward-Related and Control-Related Circuitry

Puberty is an important event that occurs during adolescence, which impacts behavioral and neurological development. Pubertal development is sex-specific, in that males and females undergo different changes in hormone concentrations at different

trajectories. Puberty involves physiological changes in hormone levels that prepare the human body to reach reproductive status. Puberty includes both adrenarche, the increased growth of body hair, elevated body odor, and onset of skin acne, and gonadarche, the maturation of sexual organs. Males typically experience increases in testosterone (Butler et al., 1989; Matchock, Dorn, & Susman, 2007), while levels of estrogens and estradiols increase in females (Apter, 1980; Lee, Xenakis, Winer, & Matsenbaugh, 1976). Both the onset (8-14 years old) and completion (12-17 years old) of puberty occur sooner in females as compared to the onset (10-14 years) and completion (14-18 years) of puberty in males (Lee, 1980). Though males tend to experience a later onset of puberty, they tend to have a faster progression through puberty (Marceau, Ram, Houts, Grimm, & Susman, 2011).

Adolescent development and pubertal status have been associated with changes in neural circuitry implicated in both reward and control networks. For reward-related neural activity, adolescence is associated with changes in the dopaminergic system, a neurotransmitter involved in reward sensitivity and sensation-seeking. In rats, dopaminergic neurons show increases in firing rates that peak in mid-adolescence before declining into early adulthood (McCutcheon & Marinelli, 2009). Similarly, expression levels of dopamine receptors in both the striatum and prefrontal cortex peak during adolescence before declining into adulthood (Andersen, Thompson, Rutstein, Hostetter, & Teicher, 2000; Gelbard, Teicher, Faedda, & Baldessarini, 1989). In adolescent humans, elevated activity in the striatum has been associated with anticipating and receiving reward (Galván, 2013), while rewards were also associated with reduced striatum – prefrontal connectivity (Teslovich et al., 2014). Adolescents in mid/late puberty have less

striatal activation and more prefrontal activation compared to pre/early pubertal adolescents and do not differ from young adults. In adolescent males specifically, higher testosterone levels are associated with greater caudate activity while anticipating rewards (Forbes et al., 2010).

Adolescence and the onset of puberty is also related to important changes in prefrontal cortex, an important component of cognitive control networks. After birth, both grey matter and white matter undergo important changes. As age increases, synapses in the brain undergo pruning. Pruning eliminates under-used synapses to optimize neuronal transmission. Primate work shows that the number of synapses in the prefrontal cortex peaks in adolescence before declining into adulthood (Bourgeois, Goldman-Rakic, & Rakic, 1994; Huttenlocher, 1979). In humans, higher association cortices (e.g., OFC, dlPFC) show decreases in gray matter volume through adolescence, implying that areas in control-related networks are being refined through pruning (Gogtay et al., 2004). This fine-tuning could impact the enhanced integration of control-related networks throughout adolescence. Second, myelination, or the white matter that acts as a protective covering around axons, increases linearly with age. Myelination speeds the transmission of action potentials down axons by increasing the capacitance of the axon and decreasing ion leakage, thus increasing efficiency of transmissions. Importantly myelination also increases in frontal, temporal, and parietal cortices (Liston et al., 2006), areas essential for cognitive functioning, from childhood to adulthood. Of note, although puberty most likely plays some role in the development of these networks, little work has thoroughly examined pubertal status. One study failed to provide evidence of a link between pubertal stage and cognitive control development, as there was no association between puberty,

either clinician ratings or hormone levels, with improvements in cognitive control performance (Ordaz, Fritz, Forbes, & Luna, 2018).

Reward and Cognitive Control in Adults

Typically, the potential for a reward enhances behavioral performance, both accuracy and reaction time, in simple cognitive control tasks that assess response inhibition. For example, in adults, potential reward mitigates both behavioral and neural markers of conflict during reactive control (Boehler, Hopf, Stoppel, & Krebs, 2012; Boehler, Schevernels, Hopf, Stoppel, & Krebs, 2014; Padmala & Pessoa, 2011; Yamaguchi & Nishimura, 2019). In a stop signal task, reward facilitated stopping (Boehler et al., 2012; Schevernels et al., 2015) and increased stopping success rates (Boehler et al., 2012). Additionally, potential reward decreases both interference (incongruent vs neutral trials) and response facilitation (congruent vs neutral), suggesting that motivation enhances attentional filtering by allowing the individual to focus on the relevant stimulus and ignore distracting information (Chiew & Braver, 2016; Padmala & Pessoa, 2011). The enhancement of attentional filtering for potential reward is further evidenced by an enhanced N1 to rewarded stop cues in a stop signal task (Schevernels et al., 2015) and to rewarded go trials in a go/nogo task (Langford, Schevernels, & Boehler, 2016).

Other work has assessed the effects of reward on tasks that tap both proactive and reactive control in adults. In adults, reward and information interact to produce low error rates and decreased reaction times when cues are informative about the upcoming stimulus and denote reward (Chiew & Braver, 2013, 2016; Soutschek, Strobach, & Schubert, 2014). Particularly, in the AX-CPT paradigm, participants are told to watch for

the A (cue)-X(probe) letter pair and to press one button when the A appears (e.g., 1), but a different button when the X appears (e.g., 2). If the stimuli are not the A-X letter pair (e.g., A-Y, B-X, or B-Y trials) then they should press 1 twice. However, a majority of trials are AX letter pairs, promoting a strong tendency to prepare to press 2 when an A cue appears, so error rates are increased on rare A-Y trials. Yet, when a B cue appears, participants know that they should press 1 and do not need to engage proactive control. In a rewarded AXCPT task, incentive conditions almost entirely shift the control strategy to proactive control as seen by increased accuracy on A-X trials and decreased accuracy on A-Y trials (Chiew & Braver, 2013). In a working memory task, lPFC activity was sustained during trials with reward cues, suggesting that proactive control was actively maintaining the task set on these trials, a pattern not apparent in nonrewarding trials (Jimura, Locke, & Braver, 2010). As such, motivation can act on selective parts of cognition, such as attentional filtering or proactive control, so that behavior is optimized to receive the potential reward.

Reward and Cognitive Control throughout Development

While work in adults has explored the influence of reward on proactive control strategy, to date, work examining the interaction of reward and cognitive control in adolescents has focused on tasks without a preparatory cue. Both children (Padmanabhan, Geier, Ordaz, Teslovich, & Luna, 2011) and adolescents modulate their performance when there is a potential for reward by becoming more accurate and speeding up reaction times (Chung et al., 2011; Geier, Terwilliger, Teslovich, Velanova, & Luna, 2010; Zhai et al., 2015). Adolescents recruit the salience network, associated with cognitive control,

moreso for rewarded trials than nonrewarded trials while preparing an antisaccade response (Hallquist, Geier, & Luna, 2018). Moreover, with higher magnitude reward, adolescents error rates decreased on an antisaccade task, but adult performance was not modulated by incentive type or magnitude (Geier & Luna, 2012).

In an effort to provide a neurological explanation for reward-driven behavior in adolescents, cognitive neuroscientists have examined the interplay between reward-related brain networks and cognitive control-related brain networks. Indeed, these two different networks, which both play an important role in motivating and controlling an individual's behavior, have unique developmental trajectories. Reward-related neural activity peaks in adolescence (Silverman, Jedd, & Luciana, 2015; Van Leijenhorst et al., 2010), while cognitive control-related activation linearly increases from childhood to adulthood (Shulman et al., 2016; Somerville & Casey, 2010; Steinberg, 2008). Indeed, adolescents display *increased* striatal activity, not observed in children or adults, during rewarded trials compared to neutral trials in an antisaccade task (Geier et al., 2010; Padmanabhan et al., 2011). This increased reward-related activity manifests when preparing the response to earn the reward and upon receipt of the reward (Ernst et al., 2005; Galvan et al., 2006a). Critically, these increases in striatal activity predict real-world risk-taking behaviors in adolescence (Galvan, Hare, Voss, Glover, & Casey, 2007). This result aligns with the ∩- shape associated with heightened reward sensitivity throughout development. However, control-related regions are still becoming more efficient throughout adolescence (Luna et al., 2001; Ordaz et al., 2013). These divergent developmental trajectories result in an imbalance in reward-related and cognitive control networks during adolescence. Furthermore, just as control-related network integration,

rather than activation in disparate regions, is critical for cognitive control development, recent work has emphasized the importance of corticostriatal *connectivity* in the development of motivation-cognition interactions (Somerville & Casey, 2010). Corticostriatal connectivity between reward-related striatal areas and control-related cortical areas, is weaker in adolescents compared to adults, especially under high stakes (Insel, Kastman, Glenn, & Somerville, 2017). Striatal activity supports cognitive control earlier in development, but hampers performance in adulthood (Paulsen et al., 2015). Again, connectivity between regions is tantamount to reward interacting with cognitive control processes to influence behavior, highlighting the importance of connectivity measures.

Substance use research also extensively examines the interaction of reward and cognitive control. Overactive sensitivity to rewards (e.g., drugs) and decreases in cognitive control and control-related network activation are also hallmarks of substance use disorder (Volkow, Fowler, & Wang, 2003; Volkow, Fowler, et al., 2010; Volkow, Wang, Fowler, & Tomasi, 2012). Substance users, including users of cannabis, cocaine, and alcohol, typically exhibit increased striatal activity and corticostriatal connectivity after cues of their drug of choice (Filbey & Dunlop, 2014; Wilcox, Teshiba, Merideth, Ling, & Mayer, 2011; Wrase et al., 2007). Moreover, substance users even display increased connectivity between reward areas and cortical areas at rest (Ma et al., 2010) and early increases in corticostriatal activity at rest is predictive of earlier substance use onset (Motzkin, Baskin-Sommers, Newman, Kiehl, & Koenigs, 2014). In contrast to increases in activation in reward areas and increases in connectivity between reward and cortical areas in substance users, substance users display deficits in cognitive control

(Charles-Walsh, Upton, & Hester, 2016) and reduced connectivity within prefrontal areas (Ma et al., 2010; Schmidt et al., 2014). Further, specific interactions between reward sensitivity and cognitive control predict onset of adolescent substance use. Specifically, adolescents with high behavioral activation systems, marked by approach behaviors, but low cognitive control show earlier onset of adolescent substance use, while this relation does not exist for adolescents with high behavioral activation systems, but high cognitive control (Kim-Spoon et al., 2016). This profile of increased reward sensitivity but underdeveloped, or dysfunctional, cognitive control seen in both adolescence and substance use underscores the importance of understanding the nuance of these interactions in both proactive and reactive contexts. Reward cognitive control interactions play an important role in both adolescent risk-taking, spanning many domains, but also continues to describe substance users into adulthood.

Taken together, this research emphasizes that reward influences cognitive control and motivational factors play an important role in goal-directed behavior. Though work in adults implicates reward as a strong influencer of control strategy use, little work has addressed this reward-control strategy interaction throughout development. The interaction between reward and control is especially important in adolescence, a period of heightened reward sensitivity, but immature control, and in males, who display more reward-seeking and risk-taking. Further, no work to date has used more advanced time-frequency measures to characterize monitoring and control recruitment during active preparation vs reactive responses in rewarded contexts. Thus, the first aim of my dissertation will be to characterize the developmental differences in the influence of reward and control strategy (proactive vs reactive) on two distinct mechanisms of

cognitive control (monitoring and control recruitment) in a sample of pre-adolescent and adolescent males aged 9 – 17 years old. I anticipate that in the presence of informative cues, reward will enhance proactive control recruitment, especially in mid-adolescence. In the absence of informative cues, reward will upregulate the recruitment of reactive control, particularly in mid-adolescence.

Because heightened reward sensitivity and decreased cognitive control are both implicated in substance use and risky behavior (Hawes et al., 2017), especially during adolescence, it is vital to understand how reward influences these nuanced cognitive control mechanisms throughout development. Dysfunction in these mechanisms may provide insight into adolescent risk-taking and substance use.

2.3 Executive Functions (EF)

Executive functions (EF) are a set of cognitive processes that are essential for proper cognitive control. EF abilities include planning, organizing, and controlling behavior. A large variety of cognitive tasks are considered to tap executive function, but literature typically shows moderate correlations between them (Lehto, 1996; Welsh, Pennington, & Groisser, 1991) and exploratory factor analyses tend to create separable factors when a battery of cognitive tasks is administered (Miyake et al., 2000; Visu-Petra, Cheie, Benga, & Miclea, 2012). It is important to consider these distinct domains of EF to provide a more nuanced understanding of executive functions. Indeed, Miyake and colleagues (2000) found that EF could be separated into three distinct, yet correlated, factors: inhibitory control, updating (working memory), and set-shifting. Inhibitory control, synonymous with reactive control explained above, is the ability to withhold inappropriate prepotent responses or ignore distractors to make an appropriate response.

Working memory is the ability to temporarily store and maintain a representation necessary for a task at hand. Set-shifting is the ability to flexibly change mental representations or rules in response to changing goals.

Development of Executive Functions

These three domains also exist throughout development. For example, Huizinga, Dolan, & van der Molan (2006) tested the three domains in groups of 7-year olds, 11-year olds, 15-year olds, and 21-year olds using a battery of cognitive tasks. The factor structure of the three-domain model did not vary by age, even after accounting for processing speed. Though, it is important to note that this model differed from Miyake's model because the three inhibition tasks did not load strongly onto one factor and were each used as separate inhibition factors (Huizinga et al., 2006).

Additionally, each domain exhibits similar developmental trajectories to each other. Working memory develops almost linearly throughout childhood and adolescence (Brocki & Bohlin, 2004) before reaching adult levels in older adolescence, around 15-19 years old (Huizinga et al., 2006; Luciana & Nelson, 1998; Luna, Garver, Urban, Lazar, & Sweeney, 2004). Inhibitory control seems to develop and reach adult levels of performance more quickly, around age 12 (Brocki & Bohlin, 2004; Durston et al., 2002; Huizinga et al., 2006; Beatriz Luna et al., 2004). Similarly, set-shifting is less efficient at younger ages compared to older ages, again reaching adult levels around age 12 (Crone, Bunge, van der Molen, & Ridderinkhof, 2006; Huizinga et al., 2006).

Relations between EF and Reward and Control Strategy

Executive functions and reward systems both rely on the dopaminergic system. First, dopamine has a central role in the craving of pleasurable stimuli, like reward, and contributes to consummatory pleasure (Robinson & Berridge, 1993). Second, according to the Dopamine Gating Hypothesis (Braver & Cohen, 2000), dopaminergic inputs to the PFC act as a gating mechanism to update task representations and guard against interference from distracting stimuli. These two aspects of dopamine gating, updating representations and goal-directed attention, are integral to working memory and cognitive control. Previous work has linked working memory to proactive control specifically. In adults, greater working memory capacity and increased fluid intelligence are associated with the use of proactive control strategies (Kane & Engle, 2002; Janowich & Cavanagh, 2019). With greater working memory capacity, task goals are better maintained and used to prepare for upcoming stimuli. The same pattern is seen in children (Munakata et al., 2012). In particular, in children ages 5 and 9, working memory predicted the use of proactive control strategy above and beyond age and the other EF domains. Moreover, an ERP component called the P3b, associated with updating, mediated the relation between working memory and proactive strategy use (Troller-Renfree, Buzzell, & Fox, in press).

Additionally, inhibitory control and set shifting have been associated with neural measures of cognitive control. Performance on the Iowa Gambling Task, reflecting rule use and set-shifting, and a Stroop task, indexing inhibitory control, predicted N2 amplitudes on a go-nogo task above and beyond age in children aged 7 – 16 (Lamm et al., 2006b). N2 amplitudes are typically associated with conflict monitoring and reactive control, suggesting that both inhibitory control and set-shifting abilities underlie reactive

control throughout all ages of development. Collectively, these results provide evidence that EF abilities are integral to cognitive control development.

Though there is a strong neuropharmological link between executive functions, rewards, and cognitive control via the dopamine system, few tasks have examined how EF abilities are associated with the influence of reward on cognitive control. In one study, fewer cognitive shifting problems were associated with better antisaccade performance on both neutral and rewarded trials. Further, adolescents with greater cognitive shifting problems needed to recruit more OFC activation to successfully inhibit antisaccade responses, but this pattern was not evident for rewarded trials (Zhai et al., 2015). This result suggests that reward upregulates control-related activation in the OFC in those with cognitive shifting problems.

These three EF skills, working memory, inhibitory control, and set shifting, subserve proactive and reactive control when these strategies are used to accomplish a goal. Reward motivates individuals and subsequently alters both neural activity and behavioral performance associated with cognitive control. However, no work to date has examined how EF supports proactive and reactive control in the presence or absence of reward. Additionally, EF abilities continue to develop throughout adolescence, raising the question of how EF influences on the effect of reward on proactive and reactive control change throughout development. Therefore, the second aim of the current study is to examine relations between executive functions and proactive and reactive control throughout development, both in the absence and in the presence of reward. I expect that individual differences in EF domains will relate to cognitive control recruitment and performance, regardless of age. Specifically, increased working memory abilities will

bolster reward-related proactive control performance as mediated by increased preparatory cue-locked theta ICPS after informative cues. Additionally, inhibitory control abilities will support reward-related reactive control in the absence of informative cues as mediated by increased stimulus-locked theta ICPS.

Chapter 3: Methods

3.1 Participants

Male participants aged 9 - 17 were recruited to participate in the study. To mitigate the possibility of heterogeneous effects due to gender differences in reward sensitivity and pubertal development, the sample was restricted to only males. Male children and adolescents were recruited using the Infant and Child Studies database at the University of Maryland. The University of Maryland Institutional Review Board approved all procedures.

According to a power analysis conducted using G*Power 3.1 (Faul, Erdfelder, Lang, & Buchner, 2007), 60 participants provide 85% power to detect a small effect size of 0.2 for a repeated measures ANOVA with a within-between interaction at $\alpha = .05$. A repeated measures ANOVA power analysis was used to estimate the sample size necessary, even though multi-level models will be used for analysis. Due to a dearth of resources to perform a power analysis for a multi-level model, G*Power was used to approximate the necessary sample size.

The sample consisted of 76 male participants. All participants reported no birth defects or current diagnoses, no visual/uncorrected visual impairment, and no allergies to salts/plastics/latex. Eight participants were excluded for various reasons including developmental delays not reported at screening (n = 2), <60% accuracy on task baseline (n = 5), and too few stimulus-locked trials after EEG cleaning (n=1). The final sample consisted of 68 neurotypical males (M_{age} = 13.61, SD = 2.52 years, Range = 9.09 – 17.84; see Figure 1 for histogram). Table 1 details information about demographics and pubertal status of the final sample.

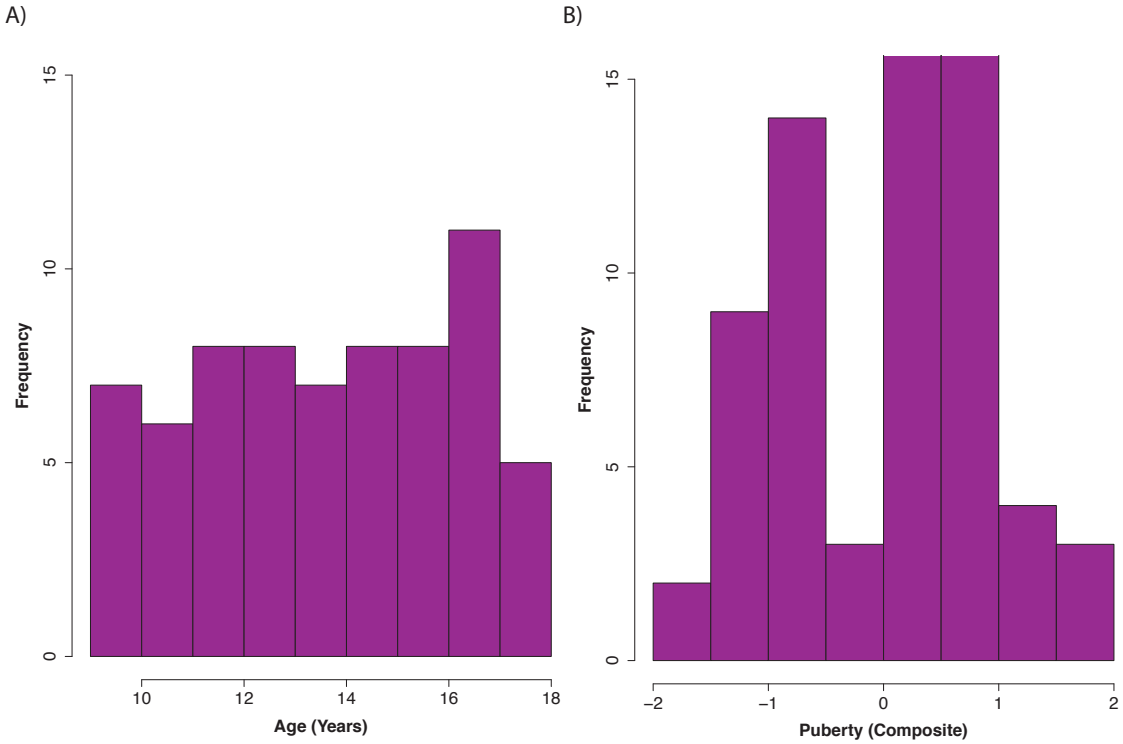


Figure 1. Histogram distribution of participants' age (in years) and puberty composite scores.

Table 1.
Sample Demographics, N = 68

Mean Age (Years)	13.61 (2.52)
Puberty Status	
Pre-Pubertal	7 (8.8%)
Early Pubertal	17 (25.0%)
Mid-Pubertal	25 (36.8%)
Late Pubertal	18 (26.5%)
Post-Pubertal	1 (1.5%)
Race/Ethnicity	
Black/African American	17 (25.0%)
Asian	5 (7.4%)
Caucasian	30 (44.1%)
Hispanic	4 (5.9%)
Biracial	12 (17.6%)
Mother's Education Level	
High School Graduate	2 (2.9%)
Associates Degree	3 (4.4%)
College Graduate	16 (23.5%)
Graduate Degree	45 (66.2%)
Other	1 (1.5%)
Unknown	1 (1.5%)
Median Annual Household Income	$135,000

3.2 Procedures

All parents of child and adolescent participants provided informed consent and all child and adolescent participants provided assent. Participants were seated about 70 cm in front of the presentation computer. Then, participants completed two blocks of a rewarded cued flanker paradigm. Participants completed a baseline block without any possibility of reward. This baseline block established their mean flanker reaction time. Next, participants completed a reward block. In this block, participants were rewarded on reward-cued trials in which they were both correct and their reaction time was less than their mean reaction time from the baseline block. All tasks were presented in EPrime 2.0.10 (Psychology Software Tools, Pittsburgh, PA). EEG was recorded throughout both

blocks using a HydroCel Geodesic net and Netstation 5.2 software (EGI, Inc; Eugene, OR). Upon completion of the task, the participant completed a neurocognitive battery (NIH Toolbox; Gershon et al., 2013) and self-report questionnaires.

3.3 Rewarded Cued Flanker Paradigm

The rewarded cued flanker paradigm (Figure 2) was adapted from Chiew & Braver (2016). In this paradigm, participants were presented with an array of arrows (e.g. > > > > >). They were instructed to press a button on a button box depending on the direction of the center arrow as quickly and as accurately as possible. In 50% of the trials, the arrows were congruent, or facing the same direction (> > > > > or < < < < <). In the other 50% of trials, the arrow was incongruent, or the middle arrow was facing a direction than the flanking arrows (> > < > > or < < > < <).

Before the arrows were presented, the participants were given cues to indicate which trial type (i.e., congruent or incongruent) was about to be presented. A box was presented in the center of the screen with a cue image on each side of the box. If a circle was presented, the upcoming trial was congruent. If a triangle was presented, the upcoming trial was incongruent. Alternatively, if a question mark was presented, then the participant was unaware of what trial type was to be presented. Trials with cues that were predictive of the type of stimulus that will be shown (i.e., circle and triangle) indexed *proactive control* because participants could prepare for the upcoming stimulus. On the other hand, trials with question mark cues indexed *reactive control*, as participants could not prepare for the upcoming stimulus and needed to reflexively react once the stimulus was presented. The mapping of the cue shape and stimulus type was explicitly stated to the participant and the participant completed a cue "quiz" before the start of the

experiment to ensure correct cue mapping. The participant must have scored 100% on this quiz to continue to the task.

In addition to the informative nature of the cue shape, the cue color informed the participant about the potential for reward. Cues (circle, triangle, question mark) could either be blue or orange. In one version, blue cues (circle, triangle, question mark) informed the participant that they could potentially receive a reward of $0.10 on that trial if they were both accurate and fast enough, while orange cues meant they could not receive reward. In the second version, the orange cues denoted potential reward, but blue cues indicated no reward. These versions were counterbalanced across participants. Blue and orange were chosen as cue colors to mitigate concerns about using green, a color commonly associated with money and reward, which was used in the original task (Chiew & Braver, 2016).

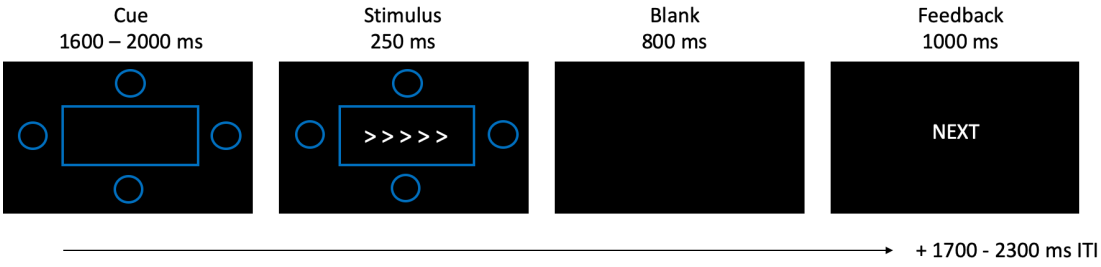
Participants completed a practice block, a baseline block, and a reward block. In the practice and baseline blocks, both blue and orange cues were presented, but the participant was not told about the meaning of the colors. First, the participants practiced and did not advance to the baseline block until they reached 60% accuracy. This criterion was established to ensure that no learning was still taking place during the baseline block. In the baseline block, a mean RT of correct trials was calculated and used as a reaction time cutoff in the reward block. Next, in the reward block, the participants were notified of the potential for reward (for their assigned reward color – either blue or orange depending on counterbalance) and that they must respond to the flanker array correctly before the reaction time cutoff in order to collect the reward. The participant did not know their actual reaction time cutoff; they just knew that they needed to respond

quickly. Using a baseline block can lead to worry about practice effects; however, previous work has shown similar effects when using pre-task baseline vs post-task baseline (Savine & Braver, 2012). The baseline block consisted of 96 trials and the reward block consisted of 288 trials. Of the 68 participants, 33 participants completed the task with blue as reward, and 35 participants completed the task with orange as reward.

The participants were also given trial-level feedback based on their responses. In the baseline block, if the response was incorrect (error of commission), the feedback read "WRONG" in red font. If the trial was skipped (error of omission), the feedback read "SLOW" in red font. If the participant made the correct response, a neutral feedback that reads "NEXT" in white font was presented. In the reward block, "WRONG" feedback was still presented for errors of commission and "SLOW" was presented for errors of omission for both rewarded and non-rewarded trials. For the non-rewarded trials, "NEXT" was presented when the response was correct below the RT cutoff or correct above the RT cutoff. For rewarded trials, "$$$$" was presented in green when the response was correct and below the RT cutoff. Otherwise, "NEXT" was presented when the response was correct, but above the RT cutoff. "$$$$" denoted that the participant had earned $0.10. Participants were told how much money they had earned during their breaks.

In both blocks, the timing of the trials was the consistent. The cue was presented for 1600 – 2000 ms, followed by the flanker array for 250ms. The arrows disappeared after 250ms, but the participant could still respond during a blank screen for the next 800ms. Then, the feedback appeared for 1000ms, followed by an ITI of 1700 – 2300ms.

Task performance was assessed with accuracy and reaction time measurements. First, data were cleaned by removing any anticipatory responses (<150ms RT) and any trials that were deemed outliers – having an RT two standard deviations above the mean. Accuracy was scored as percent errors and RT was scored as mean RT. Studies of flanker tasks show that typical behavior is to slow down and be less accurate on incongruent trials, > > < > > or < < > < <, compared to congruent trials, > > > > > or < < < < < (Eriksen & Eriksen, 1974). This phenomenon is known as the flanker effect and is indicative of larger *interference* from the flanking arrows on incongruent trials. Thus, the dependent measure for the following analyses was the flanker effect (FE), which was calculated as a difference score between incongruent and congruent trials for the measure of interest, and will be referred to as interference. After calculating interference scores, outliers +/- 3 SD above the mean were removed for accuracy and RT by condition (reward proactive, reward reactive, nonreward proactive, nonreward reactive). For accuracy interference, one outlier was removed in the nonreward reactive condition. For RT interference, one outlier was removed in the nonreward proactive condition. For neither accuracy interference or RT interference, no condition had skewness outside the acceptable range [-1, 1] or excess kurtosis outside the acceptable range [-2, 2] (West, Finch, & Curran, 1995).



	Reward		NonReward	
	Cue	**Stimulus**	**Cue**	**Stimulus**
Proactive	○	Congruent	○	Congruent
	△	Incongruent	△	Incongruent
Reactive	?	Either Congruent or Incongruent	?	Either Congruent or Incongruent

*Color was not meaningful in baseline block.
**Color was counterbalanced in reward block.

Figure 2. Cued-Reward Flanker Paradigm.

3.4 Questionnaires

Task Effort Survey

After both the baseline block and the reward block, participants answered five questions about their performance. For the first question, the participant rated "How motivated were you?" on a scale from 0 (not motivated) to 10 (extremely motivated). For the following four questions, the participant used a scale from 0 (not hard) to 10 (extremely hard). The four questions were: "How hard did you try to be correct after blue shapes?", "How hard did you try to be correct after orange shapes?", "How hard did you try to go fast after blue shapes?", and "How hard did you try to go fast after orange shapes?". After the reward block, participants also answered the question "How valuable is $10 to you?" This question was a measure used to index individual (i.e., age-related) differences in subjective value of money.

Reward Responsiveness

The Reward Responsiveness subscale of the BIS/BAS questionnaire (Carver & White, 1994) was completed by the participants. This scale consisted of five questions rated on a scale of 1=very true for me, 2 = somewhat true for me, 3 = somewhat false for me, or 4 = very false for me. Answers to these questions were averaged and prorated for any missing answers to create a reward responsiveness score. We then explored the relation between age and age^2 or puberty and puberty^2 with reward responsiveness in order to see if the curvilinear relation between development and reward sensitivity mirrors previous research (Shulman et al., 2016; Steinberg et al., 2008).

Puberty

The Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988) is a 5-item, well-validated measure designed to assess pubertal status via self-report in children and adolescents. The male version of the PDS given to the participants was scored by averaging answers to 5 of the questions: growth in height (i.e., have you begun a growth spurt), pubic/body hair, skin changes (i.e., pimples), voice deepening, and growth of facial hair. The respondents had four choices for each question: not yet started, barely started, is definitely underway, and seems completed. Answers to these questions were averaged and prorated for any missing answers to create a PDS puberty score. All children answered at least three of the five questions. The PDS has displayed strong convergent validity with clinician ratings of puberty (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987) and with sex hormone concentrations (Shirtcliff, Dahl, & Pollak, 2009).

The Morris & Udry scale was the second puberty measure collected (Morris & Udry, 1980). This measure included 8 multiple choice questions that examined growth of leg hair, growth of chest hair, growth of facial hair, vocal changes, and skin changes. Answers to these questions were averaged and prorated for any missing answers to create the Morris & Udry score. One participant did not complete the Morris & Udry scale; no other participant missed more than one question. This scale has been used in multiple studies to assess puberty (Ning et al., 2008; Somerville et al., 2018) and shows good validity with physician ratings (Morris & Udry, 1980).

Picture-Based Interview about Puberty (PBIP): Additionally, two picture-based questions that illustrated four stages of male body development as based on the Tanner scales (Tanner, 1975) were completed by the participant (Shirtcliff et al., 2009). The two picture-based questions asked the respondent to choose the stage of growth that was closest to their own for, first, the male genitals and, second, pubic hair. Questions were explained to the participants, then the researcher left the room and allowed the participant to answer privately. Scores on the two questions were averaged together. Two adolescents did not complete the PBIP; all others completed both questions.

PDS scores were significantly correlated with both PBIP scores, $r(64) = 0.72$, $p < .001$, and with the Morris & Udry scores, $r(65) = 0.80$, $p < .001$. The PBIP scores and the Morris & Udry scores were also highly correlated, $r(64) = 0.66$, $p < .001$. Due to the high correlations amongst the three measures, a composite was formed. Each puberty measure was z-scored. The z-scores were then averaged to form the composite. Each analysis examined, first, age-related changes, and, second, puberty-related changes.

3.5 Electroencephalography (EEG)

EEG Acquisition and Preprocessing

During the rewarded cued flanker paradigm, continuous EEG was recorded from a 128-channel HydroCel Geodesic Sensor Net. The data were collected using a NetAmp 400 amplifier and Netstation 5.2 software (Electrical Geodesic, Inc, Eugene, OR) sampled at 500 Hz and referenced to Cz online. All electrode impedances were below 50 kΩ prior to data collection. EEG analysis was conducted off-line using MATLAB 2014b (MathWorks, Inc., Natick, MA), the EEGLab Toolbox (Delorme & Makeig, 2004), and custom MATLAB scripts. Data was high-pass filtered at 0.3 Hz and low-pass filtered at 49 Hz. The FASTER plugin for EEGLab (Nolan, Whelan, & Reilly, 2010) identified bad channels ($Mean_{\text{Number of Bad Channels}} = 5.32$, SD = 2.69, Range = 1 – 14). No participant had greater than 12% bad channels. To identify artifacts in the data, independent components analysis (ICA) was performed on a copy of the dataset that was filtered with a 1 Hz high-pass filter. Prior to ICA decomposition, the copied data was epoched into arbitrary 1s epochs for the purpose of detecting and removing portions of the EEG data contaminated with significant artifact. An initial rejection of noisy EEG data was performed using a combined voltage threshold rejection of ±1000 μV to remove disconnected channels and a spectral threshold rejection using a 30 dB threshold within the 20–40 Hz band to remove EMG-like activity (EEGLAB pop_rejspec function; Delorme & Makeig, 2004). If artifact rejection rejected >20% of epochs for a given channel, this channel was removed from both the 1 Hz high-pass dataset and the 0.1 Hz high-pass ERP dataset. ICA weights from the ICA run on the copied (1Hz) dataset were then copied back to the continuous 0.3 Hz high-passed data. The adjusted-ADJUST Matlab scripts (Leach et al.,

in press; Mognon, Jovicich, Bruzzone, & Buiatti, 2011) identified artifactual independent components, which were then removed from the data. The data was then epoched from -1000ms before to 2000ms after both the cue and the stimulus. A rejection threshold of +/- 125 μV based on ocular electrodes (electrode numbers on 128 ch. geodesic net: 8, 25 127, 126) was utilized to identify and reject any ocular artifacts that may have been missed during previous processing steps. After rejection of epochs containing residual ocular artifacts, epochs containing channels with voltage +/- 125 μV were interpolated at the channel level unless more than 10% of channels exceeded this threshold within a given epoch, in which case the epoch was rejected instead. Channels that exceeded the ± 125 μV threshold for greater than 20% of epochs were instead removed from the dataset. Finally, any missing or removed channels were interpolated using a spherical spline interpolation and data was re-referenced to the average of all electrodes. The epoched data was filtered with a surface Laplacian filter in order to minimize volume conduction over the scalp by filtering out spatially broad features of the data (Cohen, 2014). This procedure improves both spatial and functional specificity of brain activity (Kamarajan, Pandey, Chorlian, & Porjesz, 2015; Tenke & Kayser, 2012). A surface Laplacian transformation highlights high-spatial-frequency at a small cluster of electrodes but lessens low-spatial-frequency activity apparent at most or all electrodes (Cohen, 2014).

Time Frequency Analyses

In order to have a complete picture of proactive and reactive control, both **cue-locked activity** and **stimulus-locked activity** were analyzed. Proactive control was thought to be engaged during the period of stimulus preparation after an informative cue revealed pertinent information about the upcoming stimulus. Indeed, cue-locked neural

activity has been shown to be increased after an informative cue, reflecting increased proactive control (Cooper et al., 2016). However, if the cue was not informative, only reactive control could be engaged after the stimulus appears in preparation for the response. In the cases of uninformative cues, stimulus-locked neural activity would be increased, indicative of reactive control. Thus, cue-locked theta power and ICPS were calculated to investigate differences in proactive stimulus preparation based on reward and control strategy. Second, stimulus-locked (to flanker arrows) theta power and ICPS were used to assess differences in reactive response preparation based on reward and control strategy (see Figure 3).

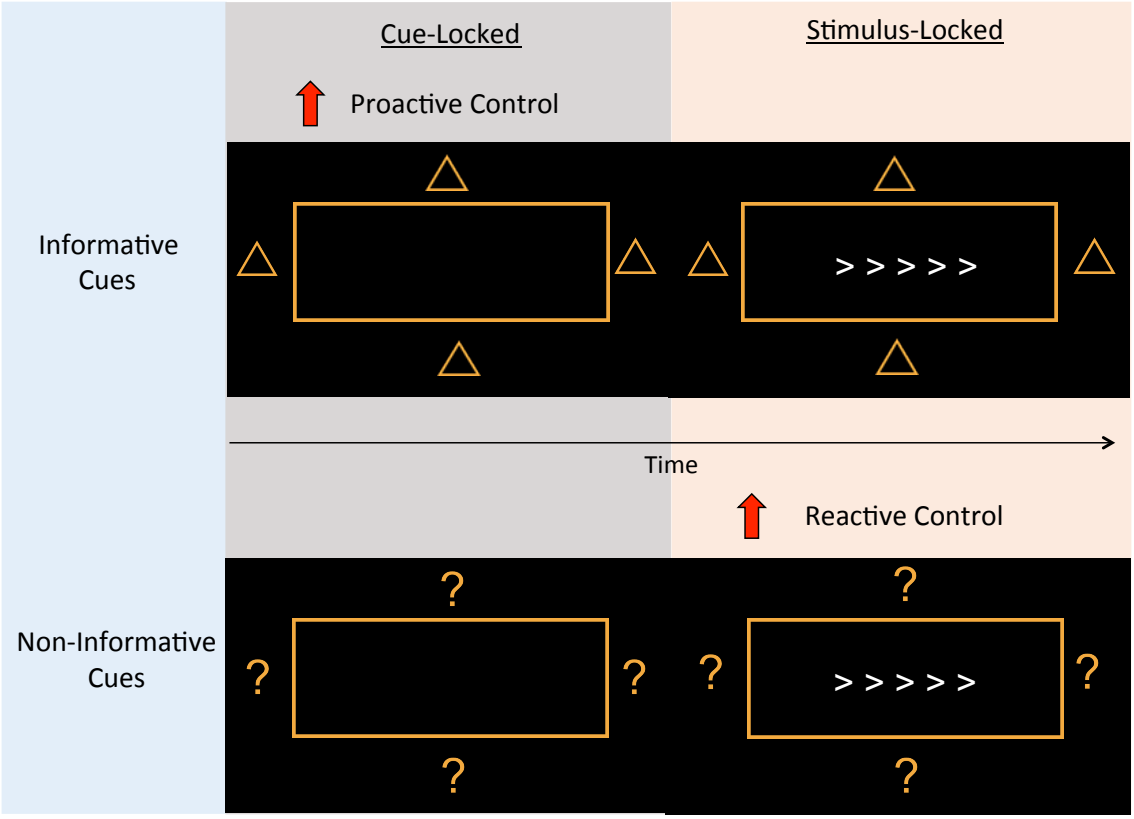


Figure 3. Example trial depicting the epochs of interest.

Multiple time frequency measures were computed from the pre-processed EEG data in order to index monitoring and control instantiation. Time frequency total power reflected monitoring activity. Additionally, time frequency inter-channel phase synchrony (ICPS) from medial electrodes to frontal electrodes was calculated to index control instantiation.

Theta power: Theta power, reflective of monitoring, in each epoch of interest (cue-locked and stimulus-locked) was computed using the EEGLAB newtimef function. Event related spectral perturbation (ERSP) was calculated for the epoched data. ERSP provides a two-dimensional (time by frequency) estimate of average changes in spectral power (in dB) relative to a baseline period (Delorme & Makeig, 2004). To compute ERSP, each CSD converted epoch was convolved with Morlet wavelets, which estimated spectral power in the frequency range 5–40 Hz (in 120 linearly spaced steps). To optimize the time-frequency resolution, wavelet cycles were set at 3 cycles at the lowest frequency (5 Hz) increasing to 12 cycles at the highest frequency (40 Hz). ERSPs were computed for all channels and separately for the four cue-locked conditions (proactive reward, reactive reward, proactive nonreward, reactive nonreward) and for the eight stim-locked conditions (proactive reward incongruent and congruent, reactive reward incongruent and congruent, proactive nonreward incongruent and congruent, reactive nonreward incongruent and congruent). ERSPs were calculated for each epoch relative to a baseline period of -400 to -100ms before the onset of either the cue or the stimulus. Epochs were then averaged across a frontocentral cluster (E12, E5, E6, E13, E112, E7, E106; see Figure 4).

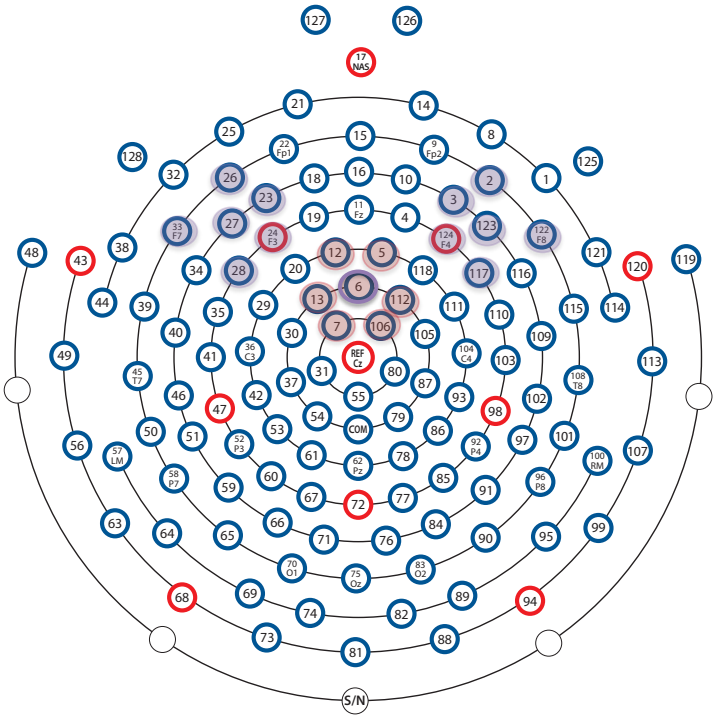


Figure 4. Clusters used for theta analyses. Both cue-locked and stim-locked theta power were analyzed from a frontocentral cluster of electrodes (red). Both cue-locked and stim-locked ICPS were analyzed from an E6 seed (circled in purple) to a right frontolateral cluster and a left frontolateral cluster (purple). Note that E6 was included in the frontocentral cluster for theta power and served as the seed for theta ICPS.

The primary ERSPs of interest were theta (4-8 Hz) activity after the cue and after the stimulus at this frontocentral cluster. To choose the time window of the region of interest, cue-locked theta power was averaged over all conditions (Fig 5A) and was found to be maximal from 4-8Hz and 100-500ms post-cue. This ROI was exported for analysis of cue-locked theta power. Because stim-locked theta power was analyzed as a flanker effect (incongruent – congruent), that subtraction was averaged over the four conditions (reward proactive, reward reactive, nonreward proactive, nonreward reactive; Fig 5B) and theta power was found to be maximal from 4-8Hz and 300-700ms post-stimulus.

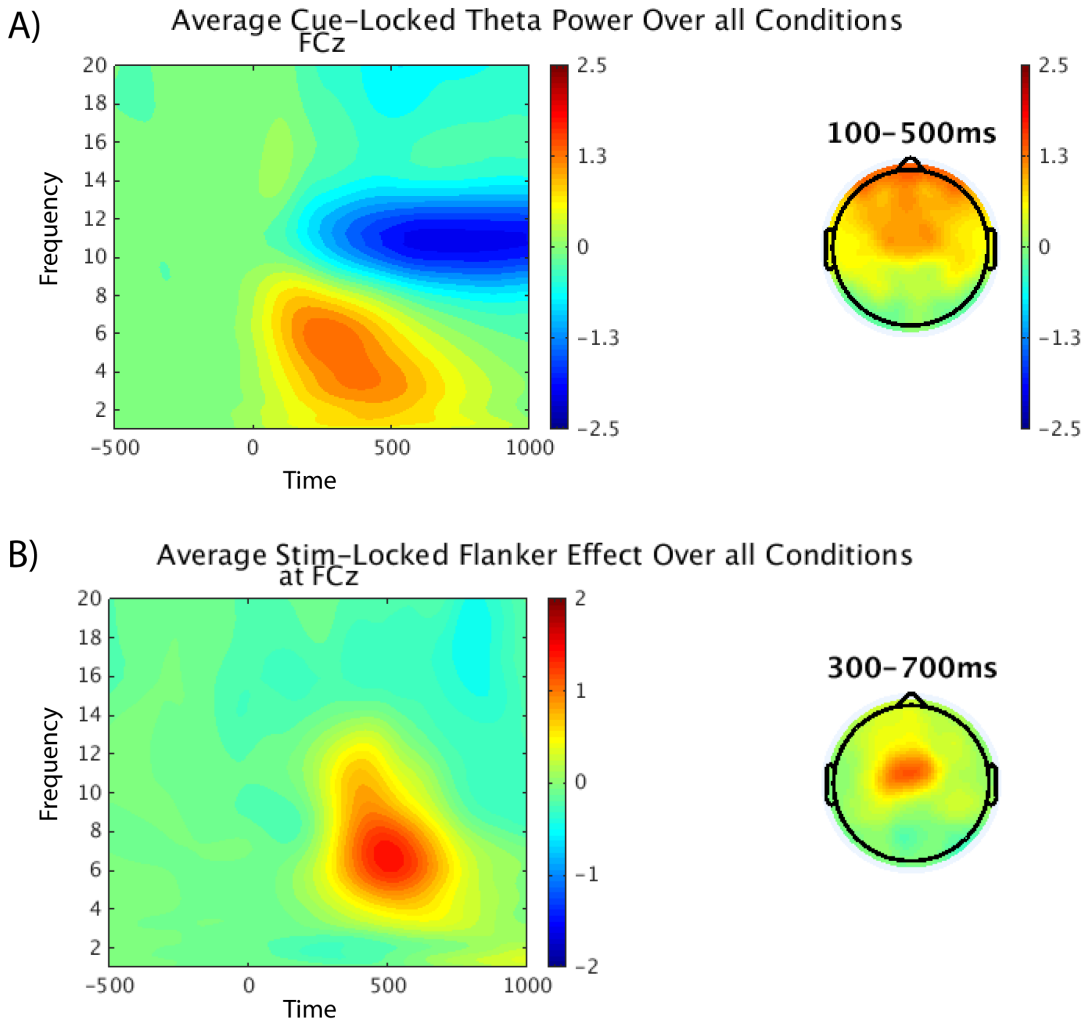


Figure 5. Cue and Stim-locked theta power averaged over all conditions. Averaging over all conditions allowed for a non-biased selection of the ROI.

Theta Inter-Channel Phase Synchrony (ICPS): Theta ICPS is a measure of the consistency of phase oscillations between two channels (or clusters of channels) over time and frequency (Cohen, 2014). Here, ICPS was calculated as follows:

$$ISPC_f = |n^{-1}\Sigma e^{i(\varphi xt - \varphi yt)}|$$

where n is the number of trials for each time and each frequency band, φ_x and φ_x are the phase angles of electrodes x and y at frequency f and time t. e^i is from Euler's formula and provided complex polar representation of phase angle difference (Cohen, 2014). Thus,

phase angles were calculated from two electrodes and then subtracted. An ICPS value closer to 1 indicated that the phase angles from two channels were completely synchronized, whereas an ICPS value close to 0 indicated random phase angle difference between two channels (Cavanagh et al., 2009). ICPS was calculated on surface Laplacian-transformed data to measure connectivity between channel clusters overlying medial and lateral frontal areas in both the left and right hemispheres (see Figure 4). ICPS was calculated for each time point of a trial and then averaged across trials using the above equation in the theta frequency (4-8Hz).

For both cue-locked and stim-locked theta power and ICPS, each condition had to have at least 8 trials to be included. Two participants each had one condition in stim-locked data that were below the 8 trial cutoff. The data for these two conditions were set to missing. For stim-locked data, flanker effects were then calculated by subtracting congruent from incongruent to create stimulus-locked interference scores. Additionally, outliers +/-3 SD above the mean were removed. For theta power, the outliers were removed within condition, while for theta ICPS, outliers were removed based on both condition and hemisphere. No outliers were removed for cue theta power. Two outliers were removed for stim theta power interference: one in the proactive nonreward condition and one in the reactive nonreward condition. For cue theta ICPS, there was one outlier in the proactive reward left hemisphere, one in right hemisphere and one in left hemisphere for reward reactive, and one in the reactive nonreward right hemisphere. There were no outliers for stim theta ICPS interference. Paired t-tests revealed that right and left lateralized cue-locked and stim-locked ICPS did not differ based on condition (p's > 0.1). Thus, right and left theta ICPS were averaged within each condition to create

a measure of medial-lateral theta ICPS. This approach was consistent with previous work that has shown that medial-lateral connectivity, as opposed to medial-occipital or medial-parietal, indexes control recruitment and is associated with improved accuracy (Buzzell et al., 2019). No EEG measure in any condition had skewness outside the acceptable range [-1, 1] or excess kurtosis outside the acceptable range of [-2, 2] (West et al., 1995).

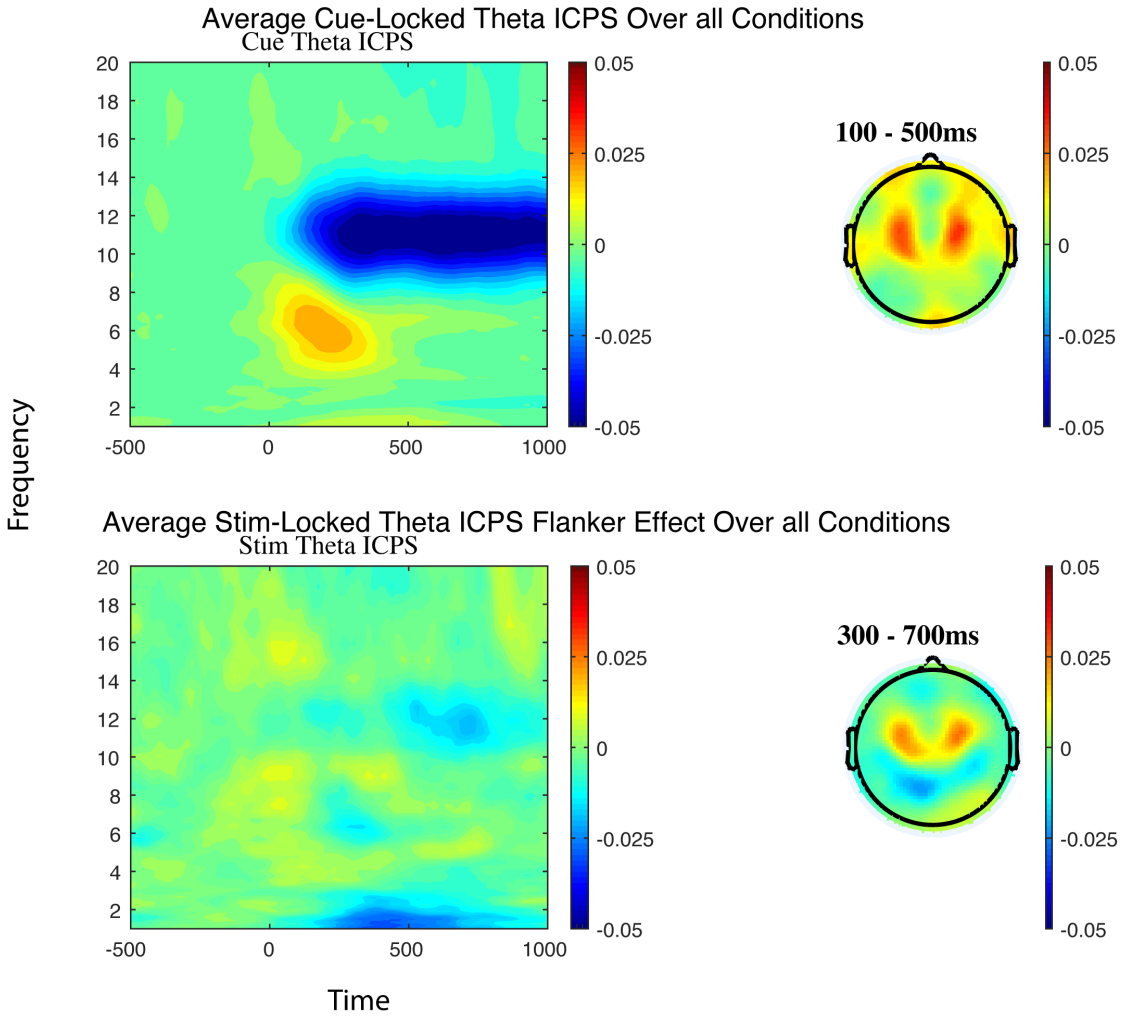


Figure 6. Cue and Stim-locked theta ICPS averaged over all conditions. ROIs were kept consistent with cue and stim-locked theta power ROIs.

3.6 NIH Toolbox

After EEG collection during the cued-reward flanker task, participants also completed three tasks of the NIH Toolbox Cognition Battery. The NIH Toolbox was developed as a standardized battery that assesses executive functions in children aged 3-17 and adults aged 18+ (Gershon et al., 2013; Heaton et al., 2014). To answer Aim #2, which investigates the influence of executive functions on cognitive control, the raw scores of NIH Toolbox tasks that tap the three main domains of executive function (Miyake et al., 2000) were used. Because we were also interested in age's impact on the relation between EF and reward-related proactive and reactive cognitive control, we used the raw scores from the NIH toolbox, rather than age-corrected or fully-corrected scores.

Working Memory

Working memory was assessed using the List Sorting Working Memory task from the NIH Toolbox. In this task, participants were presented with a series of pictures of objects (example: sheep, mouse, elephant). They had to verbally repeat the names of the objects back to the experimenter in size order, smallest to largest (example: mouse, sheep, elephant). The number of objects in the list increased on each trial and the task was ended when two trials of the same length were failed. The raw score was the total number of items correct across all trials (Slotkin et al., 2012c).

Inhibitory Control

Inhibitory control was assessed using the Flanker Inhibitory Control and Attention Test from the NIH Toolbox. This flanker task was similar to the arrays seen during the rewarded cued-paradigm task but without the cue or reward manipulations. Participants

completed 40 trials of arrow arrays by identifying the direction of the central arrow while ignoring the direction of the flanking arrows. This flanker task included both congruent and incongruent trials. The scoring algorithm incorporates both accuracy and reaction time (Slotkin et al., 2012b).

Set-Shifting

Set-shifting was assessed using the Dimensional Change Card Sort (DCCS) Task from the NIH Toolbox. Participants identified the color or shape of a stimulus. First, the dimension of interest (i.e., "color") was presented on the screen to the participant, and they had to choose the picture that matched the color of the target picture. Next, they also completed a block where shape was the dimension of interest. Finally, the testing block was a mixed block of 40 trials with a majority of "color" trials with intermittent switches to "shape." Scoring incorporated both accuracy and reaction time (Slotkin et al., 2012a).

3.7 Statistical Approach

To investigate differences in self-reported motivation, task performance, and neural measures, a series of 2-level multilevel models (MLMs) were used. Multilevel models are advantageous over traditional statistical methods, like repeated measures ANOVA, for a variety of reasons. First, MLMs can model both within-individual (e.g., repeated measures/condition differences) and between-individual effects (e.g., pubertal development scores). MLMs can also model an intercept for each participant, explaining more variance in the data, rather than forcing the entire sample to have the same intercept. Second, MLMs allow the entire sample to be used because they can estimate models for participants with some missing data; whereas, traditional ANOVAs generally

require listwise deletion (Page-Gould, 2017; Peugh, 2010). One drawback of MLM models is a lack of consensus about calculating effect sizes. Because there are two levels of analysis, across conditions and across participants, it is difficult to take both levels into account in one measure of effect size (Brysbaert & Stevens, 2018). Thus, effect sizes comparable to traditional effect sizes are not reported. In these analyses, a random intercept for each participant was estimated for each model using a variance components covariance. First, an intraclass correlation (ICC) of the given measurement was computed to justify that a multi-level model was necessary (Table 2). These values were all above the recommended ICC level (0.15 – 0.30) and suggested that MLMs are appropriate (Mathieu, Aguinis, Culpepper, & Chen, 2012).

Table 2

Intra-Class Correlations

Measure	**ICC**
Accuracy Flanker Effect	0.68
Reaction Time Flanker Effect	0.65
Cue-Locked Theta Power	0.66
Cue-Locked Theta ICPS	0.33
Stim-Locked Theta Power Flanker Effect	0.25
Stim-Locked Theta ICPS Flanker Effect	0.23

Confirming Expected Task Effects

Subjective Ratings of Motivation: Analysis of the task effort survey was done to provide evidence as to whether the participants were motivated by the potential for reward in the reward block compared to the baseline block and on reward vs non-reward trials in the reward block. First, a series of MLMs were completed to compare responses about trial types within the baseline block. Responses about how hard you tried to be

correct and fast were compared for the blue and orange trials. Trial color was effect-coded (-1 for blue and 1 for orange). Age was grand mean-centered and added as a predictor to quantify any age-related effects. Additionally, grand-mean centered age was squared and that age^2 term was entered as a predictor to explore non-linear effects. Interactions between trial color *x* age and between trial color *x* age^2 were also examined. Within the baseline block, we expected no differences for blue and orange trials, as there was no meaning assigned to the colors in the baseline block.

Second, MLMs of responses to the two questions about overall motivation ("How motivated were you to do well in this round?" and "How motivated were you to pay attention in this round?") in the baseline block vs reward block were performed. Block was an effect-coded fixed effect (-1 for baseline and 1 for reward). Age was grand mean-centered and added as a predictor to quantify any age-related effects. Additionally, grand-mean centered age was squared and that age^2 term was entered as a predictor to explore non-linear effects. Interactions between block *x* age and between block *x* age^2 were also examined. We expected higher motivation ratings for the reward block compared to the baseline block.

A third series of MLMs was used to compare ratings of how hard participants tried to be correct and fast for rewarded trials to nonrewarded trials within the reward block. Here, reward was effect coded (-1 for nonrewarded and 1 for rewarded) based on the participant's counterbalance. Age was grand mean-centered and added as a predictor to quantify any age-related effects. Additionally, grand-mean centered age was squared and that age^2 term was entered as a predictor to explore non-linear effects. Interactions between reward *x* age and between reward *x* age^2 were also examined. Within the reward

block, we expected responses to indicate that participants tried harder to be correct and fast on rewarded trials compared to nonrewarded trials. We expected that these results would confirm that the rewarded trials in the reward block significantly increase self-reported motivation. Consistent with prior work, we anticipated that there would be no age-related changes in self-reported motivation (Geier & Luna, 2012; Paulsen et al., 2015).

Of note, age must have been grand-mean centered, which was then used to compute age^2, in all analyses. If continuous age was used without centering, the age^2 term would be perfectly co-linear with age, creating multicollinearity issues. Thus, grand-mean centering, subtracting the mean value from all data points, mitigated multicollinearity and provided more trustworthy estimates. This was also true for the puberty composite scores. Effects from models using centered age were plotted with both the centered values that were used in the analysis and the continuous age values for illustrative purposes. Plots with puberty only included the puberty composite scores because the subtracted mean (-0.0035) of the z-score composite was so minimal that it did not impact the plots.

Flanker Effect: To ensure that the task was eliciting the expected flanker effect, global task effects of accuracy and reaction time were examined. Two paired t-tests were run to examine the effect of trial type (incongruent vs congruent) in the baseline block and in the reward block. I expected to see that participants were slower and less accurate on incongruent trials compared to congruent trials, regardless of block. In all subsequent analyses, the dependent measure of interest was the flanker effect (a difference score of

incongruent – congruent) for all stimulus-related measures, including accuracy, reaction time, and stimulus-locked neural measures.

Reward Responsiveness throughout Development

A multiple linear regression with age and age^2 predicting reward responsiveness was conducted. Neither age, b = -0.03, t = -1.45, p = 0.15, nor age^2, b = -0.01, t = -1.32, p = 0.19, were significant predictors of reward responsiveness. Similarly, when substituting puberty for age, neither puberty, b = -0.04, t = -.75, p = 0.46, nor puberty^2, b = -0.02, t = -.29, p = 0.77, were significant predictors of reward responsiveness. Though not statistically significant, Figure 7 displays the curvilinear pattern associated with reward responsiveness throughout adolescence.

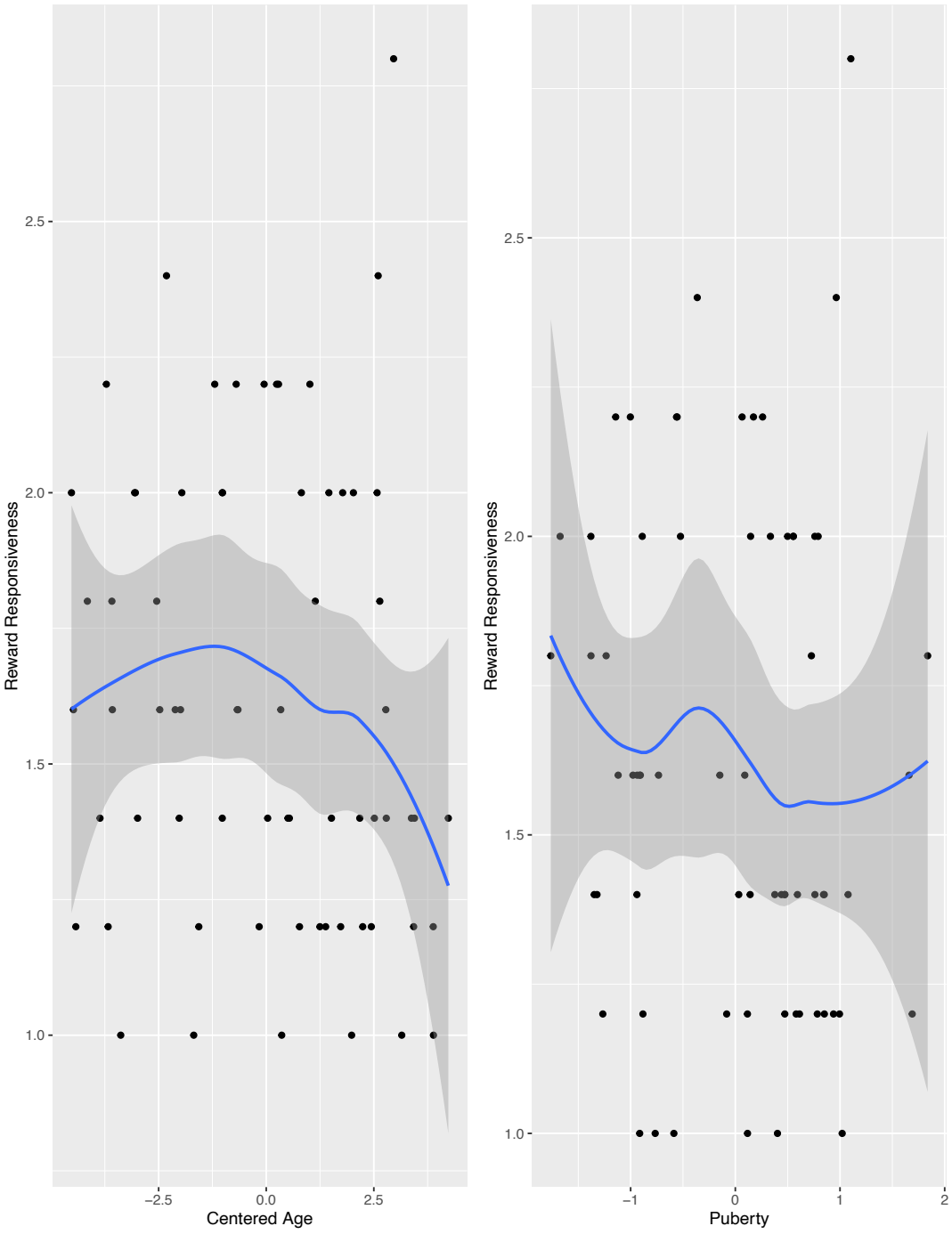


Figure 7. Patterns of reward responsiveness across adolescence.

Aim 1: Age-Related and Puberty-Related Changes in Influence of Reward on Control Strategy

Next, effects within only the reward block were examined to investigate the influence of reward and control strategy on accuracy and reaction time flanker effects throughout development. Additionally, we examined the effects of age and puberty in this developmental sample.

A series of MLMs were conducted to examine behavioral performance, cue-locked theta power and ICPS, and stim-locked theta power and ICPS during the rewarded cued flanker paradigm. For behavioral performance, the dependent variables were the accuracy (percent errors) flanker effect and the RT flanker effect. Similarly, the dependent variables for stim-locked theta were theta power flanker effect and theta ICPS flanker effect. However, for cue-locked theta, congruency was not considered because congruency was not evident to the participant for reactive question mark cues. Thus, for cue-related analyses, the dependent variables were just cue-locked theta power and ICPS values. Two MLMs were conducted for each outcome measure: one testing the effect of age and the other testing the effect of puberty. All MLMs included fixed effects of reward and control strategy. These categorical conditions were effect coded. Reactive trials were designated -1 (reference group) and proactive trials were coded as 1; Nonreward trials were coded -1 (reference group) and reward trials were coded 1. In MLMs testing age, age was grand-mean centered and used as a continuous predictor. Additionally, grand-mean centered age was squared and that age^2 term was entered as a predictor to explore non-linear effects. In MLMs testing puberty, puberty was grand-mean centered and used as a continuous predictor. Centered puberty was also squared to compute a $puberty^2$ term

that was added as a predictor to test for curvilinear relations between puberty and the outcome. In each MLM, interactions between all predictors were examined. Post-hoc tests of simple effects were used to explore significant interactions.

Brain-Behavior Relations: The next set of analyses explored relations between the four neural measures (cue-locked theta power, cue-locked theta ICPS, stim-locked theta power, and stim-locked theta ICPS) and task performance. For task performance, we specifically tested how reward-related changes in neural activity influenced reward-related *RT interference* and if age moderated those relations. Because task and developmental effects were only seen for RT interference, we focused on that behavioral measure.

To quantify reward-related measures, a difference score was calculated by subtracting the variable of interest in the nonrewarded trials from the variable of interest in the rewarded trials:

$$RewardRelated = Reward - NonReward$$

For cue-locked theta power and theta ICPS, a positive reward-related value denoted higher theta activity in the reward trials compared to the nonrewarded trials, indicating more reward-related theta activity. However, reward-related accuracy, RT, and stim-locked theta activity were calculated by subtracting flanker effects of the nonreward trials from the flanker effect of the reward trials. Thus, the interpretation of a positive reward-related value for these measures was more reward-related *interference*. More information illustrating the calculation and interpretation of these differences scores can be found in Appendix A.

A series of four hierarchical regressions were used. To simplify these analyses and avoid a triple difference score, proactive and reactive trials were investigated separately. The first two regression analyses tested the predictive power of reward-related neural activity during **proactive** trials when predicting reward-related task performance on **proactive** trials (including age and puberty, respectively). The third and fourth regression analyses tested how reward-related neural activity during **reactive** trials predicted reward-related task performance on **reactive** trials (including age and puberty, respectively).

For each hierarchical regression, the first block of predictors included the developmental measures (i.e., centered age and centered age^2 or centered puberty and centered puberty^2). The second block of predictors were the neural measures – reward-related cue theta power, reward-related cue theta ICPS, reward-related stim theta power, and reward-related stim theta ICPS – and R^2 change was assessed. The third block of predictors was interactions between each neural measure and age/puberty and between each neural measure and age^2/puberty^2 and, again, R^2 change was assessed. Outliers +/- 3SD of reward-related scores were removed prior to hierarchical regressions; thus, listwise deletion was employed for these specific analyses due to missingness from outlier removal.

Aim 2: Relations between EF and Reward's Influence on Control Strategy

Relations between EF and Task Performance: In Aim 2, we first explored the relations between EF domains, namely inhibitory control (IC), working memory (WM), and set-shifting (SS), and reward-related task performance (both accuracy and RT) throughout development. To do so, a regression path analysis was employed (see Figure

19 for path diagram). The dependent variables were reward-related proactive RT interference and reward-related reactive RT interference. The predictors were age, age^2, List Sorting Working Memory (WM), NIH Flanker Inhibitory Control (IC), and the Dimensional Change Card Sort (SS). These predictors were all mean-centered to reduce multicollinearity. Additionally, interactions between each of the EF measures and centered age and each of the EF measures and centered age^2 were entered as predictors. This path analysis was also repeated with puberty and puberty^2. Including both proactive and reactive measures in the model allowed claims to be made about specificity of the effects of EF domains on these control strategies under rewarded conditions.

Mediation of EF and Task Performance via Neural Mechanism: Next, we were interested in understanding if EF influences reward-related performance through changes in reward-related cue-locked or reward-related stim-locked theta oscillations. Thus, we conducted moderated mediations with EF *x* age predicting reward-related performance as mediated by reward-related theta.

Chapter 4: Results

Subjective Motivation

To assess differences in motivation during the task, responses on the Task Effort Survey were analyzed (Tables B1 – B4). During the baseline block, there were no differences between how hard participants tried to be correct, $t(65) = -0.04$, $p = 0.97$, or to be fast, $t(65) = -0.37$, $p = 0.71$, for blue compared to orange shapes (Fig 7A).

Comparing motivation in the baseline block to the reward block (Tables B5 – B8), participants reported that they were more motivated to do well in the reward block compared to the baseline block, $t(65) = 6.43$, $p < 0.001$ (Fig 7B). Further, there was a block *x* age interaction for how motivated the participants were to do well in the block, $t(65)= -3.73$, $p < 0.001$. Specifically, higher ratings of motivation to do well were associated with increasing age in the baseline block only, $r(66) = .42$, $p < 0.001$, while there was no association between age and motivation to do well in the reward block, $r(66)=0.05$, $p = 0.66$. These effects were qualitatively similar when substituting puberty for age. Moreover, participants were more motivated to pay attention, $t(65) = 4.66$, $p < 0.001$, in the reward block compared to the baseline block (Fig 7B). These effects were not moderated by age or puberty.

Finally, within the reward block (Tables B9 – B12), participants reported that they tried harder to be correct on the rewarded trials compared to the nonrewarded trials, $t(65) = 7.83$, $p < 0.001$ (Fig 7C). Moreover, there was a significant reward *x* age interaction, $t(65) = -2.02$, $p = 0.048$. Though follow-up correlations were not significant, they suggested that increasing age was associated with trying less hard to be correct in the reward condition, $r(66) = - 0.19$, $p = 0.12$, but with trying harder to be correct in the nonreward condition, $r(66) = 0.14$, $p = 0.24$. These effects were consistent when

substituting puberty for age. For how hard participants tried to be fast, participants tried to be faster on the rewarded trials compared to the nonrewarded trials, $t(65) = 6.70$, $p < 0.001$ (Fig 7C). This effect did not change by age or puberty. Overall, results from the task effort survey suggest that the participants were sufficiently motivated in the reward block compared to the baseline block and by the rewarded trials compared to nonrewarded trials.

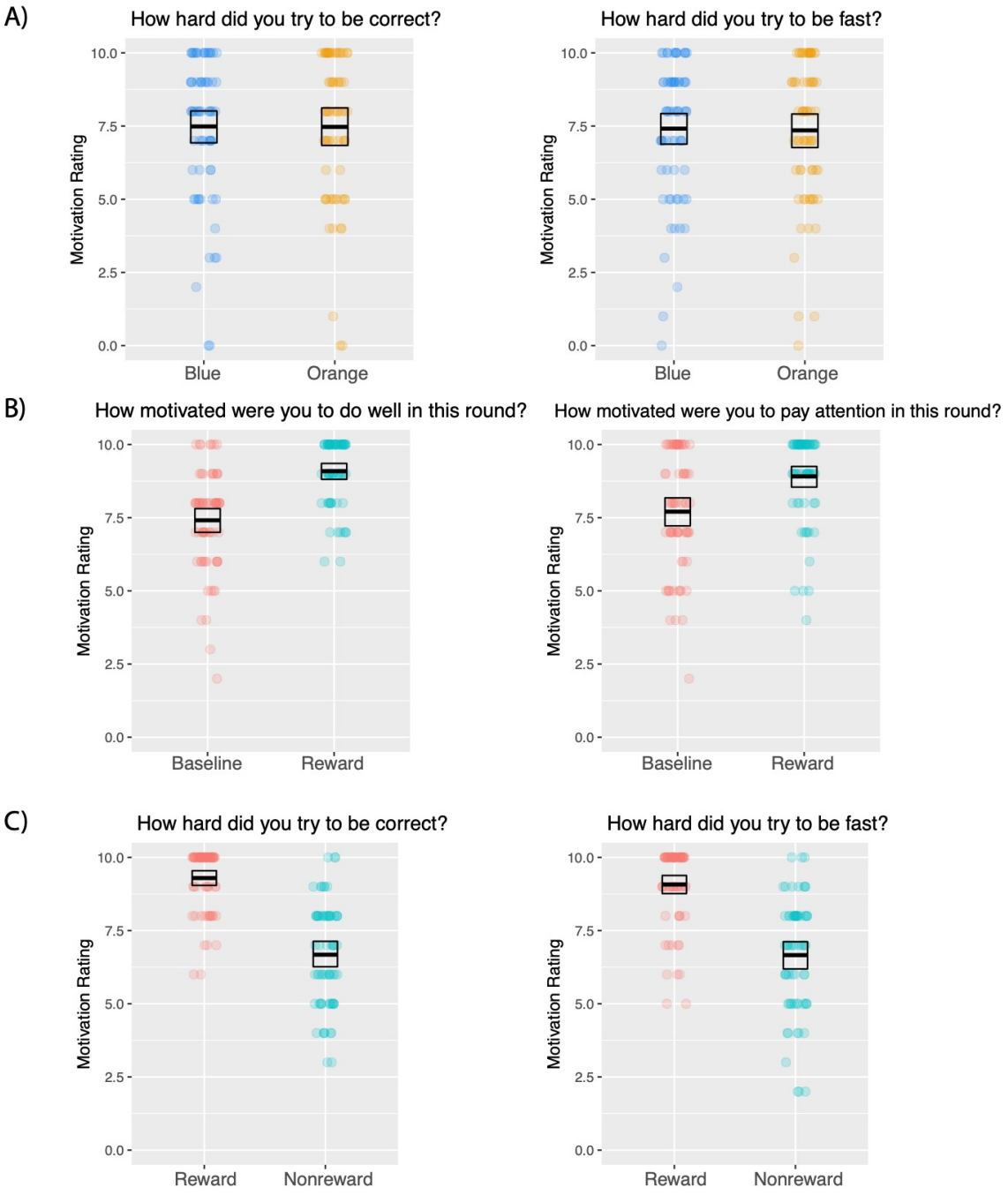


Figure 8. Results from the Task Effort Survey. A) Comparison of blue vs orange trials during the nonrewarded baseline block. B) Comparison of overall motivation in the baseline block compared to reward block. C) Comparison of rewarded trials vs nonrewarded trials during the reward block.

Correlations between Variables

The following tables display the correlations between age, puberty, behavioral, and neural measures for each condition – reward proactive, reward reactive, nonreward proactive, and nonreward reactive. Note that accuracy, RT, and stim-locked theta power and theta ICPS are interference scores (incongruent – congruent).

Table 3

Correlations among Measures for the Rewarded Proactive Condition

	1	2	3	4	5	6	7
1. Age							
2. Puberty	0.83**						
3. Accuracy Interference	- 0.13	- 0.10					
4. RT Interference	- 0.30*	- 0.14	- 0.02				
5. Cue Theta Power	0.03	- 0.01	0.29*	- 0.18			
6. Cue Theta ICPS	- 0.03	0.07	- 0.02	0.10	0.08		
7. Stim Theta Power Interference	0.18	- 0.17	0.00	- 0.12	0.39**	0.12	
8. Stim Theta ICPS Interference	- 0.17	- 0.14	- 0.07	- 0.09	0.06	- 0.17	- 0.06

† p < .10, *p<.05, **p<.01

Table 4

Correlations among Measures for the Rewarded Reactive Condition

	1	2	3	4	5	6	7
1. Age							
2. Puberty	0.83**						
3. Accuracy Interference	- 0.19	- 0.06					
4. RT Interference	- 0.17	- 0.07	0.09				
5. Cue Theta Power	- 0.10	- 0.17	0.38**	- 0.17			
6. Cue Theta ICPS	0.17	0.07	0.01	0.04	0.22†		
7. Stim Theta Power Interference	0.33*	0.22†	0.13	0.14	0.26*	0.04	
8. Stim Theta ICPS Interference	- 0.01	- 0.05	- 0.32*	- 0.04	- 0.04	0.06	- 0.11

† p < .10, *p<.05, **p<.01

Table 5

Correlations among Measures for the NonRewarded Proactive Condition

	1	2	3	4	5	6	7
1. Age							
2. Puberty	0.83**						
3. Accuracy Interference	- 0.15	0.02					
4. RT Interference	- 0.30	- 0.20	0.14				
5. Cue Theta Power	- 0.18	- 0.18	0.26*	- 0.06			
6. Cue Theta ICPS	0.07	0.04	0.01	- 0.02	0.31*		
7. Stim Theta Power Interference	0.05	- 0.10	0.00	0.00	0.15	0.02	
8. Stim Theta ICPS Interference	- 0.04	0.18	- 0.26*	- 0.02	- 0.14	- 0.14	- 0.23†

† p < .10, *p<.05, **p<.01

Table 6

Correlations among Measures for the NonRewarded Reactive Condition

	1	2	3	4	5	6	7
1. Age							
2. Puberty	0.83**						
3. Accuracy Interference	- 0.15	0.09					
4. RT Interference	- 0.36**	- 0.27*	0.00				
5. Cue Theta Power	0.02	0.01	0.16	- 0.05			
6. Cue Theta ICPS	0.18**	0.09	- 0.07	0.12	0.33*		
7. Stim Theta Power Interference	0.34	0.27*	0.14	- 0.17	0.25*	0.02	
8. Stim Theta ICPS Interference	- 0.08	0.05	- 0.14	0.09	0.18	- 0.17	- 0.11

† p < .10, *p<.05, **p<.01

Aim 1: Age-Related and Puberty-Related Changes in Influence of Reward on Control Strategy

Behavioral Results

To confirm the expected flanker effect, we first tested how participants performed on incongruent vs congruent trials. The flanker effect was present in both the baseline and the reward block. In the baseline block, participants were less accurate, $t(67) = -12.71$, $p < .001$, and slower, $t(67) = 16.938$, $p < .001$, on incongruent trials compared to congruent trials. Similarly, in the reward block, participants were less accurate, $t(68) = -17.92$, $p < .001$, and slower, $t(68) = 17.49$, $p < .001$, on incongruent trials compared to congruent trials. As such, the flanker effect, a subtraction of performance on incongruent trials minus performance on congruent trials, is used in the analyses of behavioral performance (accuracy and reaction time) and of stimulus-related theta activity.

Age-Related Changes in Influence of Reward on Control Strategy for Behavioral Performance

Next, we examined the effect of reward, control strategy, age, and age^2 on the flanker effect for behavioral performance within the reward block only. Appendix C details the complete results of the MLMs examining behavioral performance and EEG measures.

For the accuracy flanker effect, there were no significant effects of reward, control strategy, age, or age^2 (Fig 9B). There were also no significant interactions.

However, there were significant effects when predicting reaction time flanker effect, or interference. The main effect of reward was not statistically significant, $t(193.94) = -1.64$, $p = 0.10$. However, proactive trials reduced RT interference compared to reactive trials, $t(193.94) = -3.52$, $p < 0.001$. Additionally, there was a significant

interaction between age^2 and reward, $t(194.12) = -2.67$, $p = 0.008$, such that there was a curvilinear effect of age in the nonreward condition only, $t(65)=2.58$, $p =0.012$). RT interference decreases with age in the nonreward trials, dipping most in mid-adolescence (Fig9A), in line with prior evidence showing a curvilinear development of cognitive control (Luna, Marek, Larsen, Tervo-Clemmens, & Chahal, 2015).

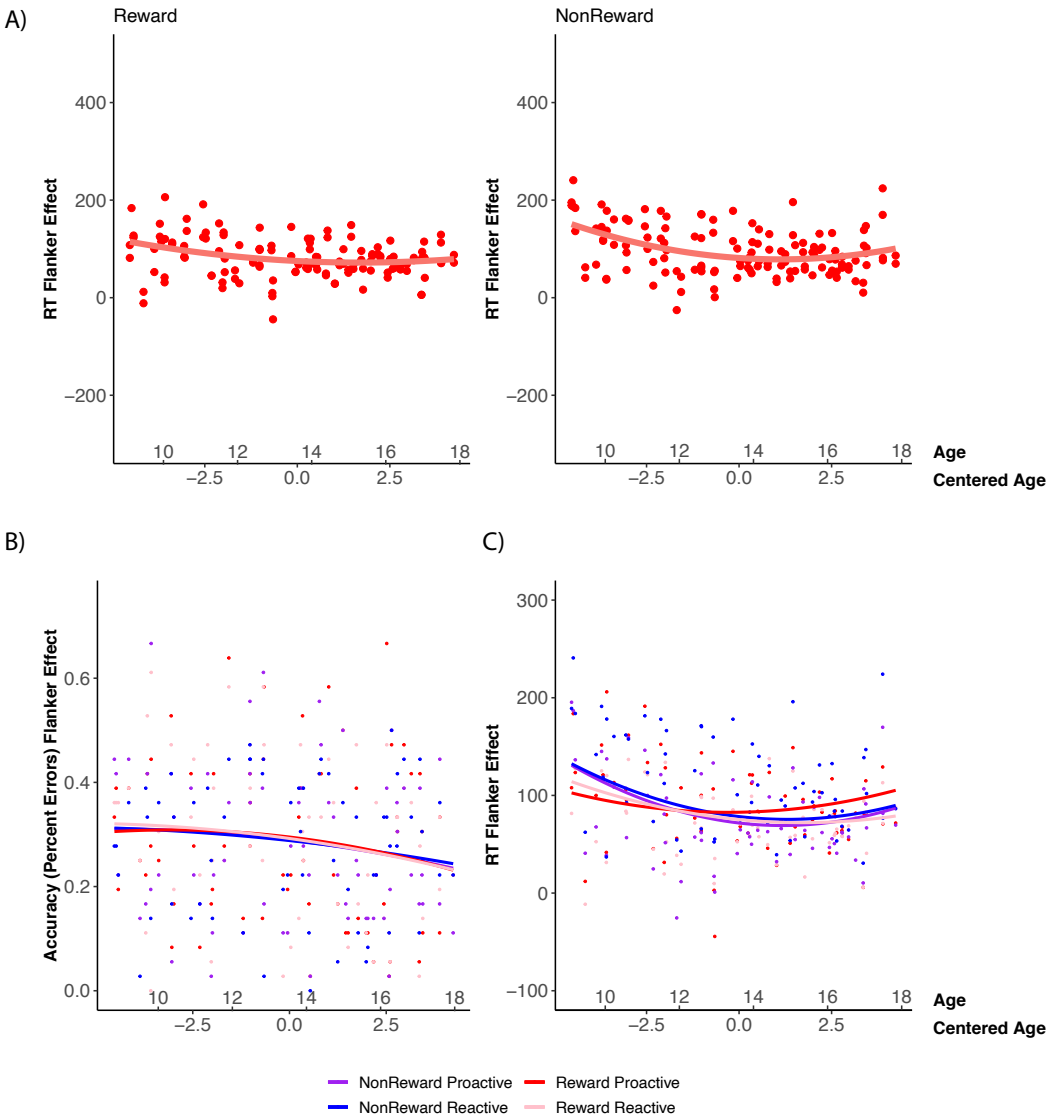


Figure 9. Age-related changes in RT interference. A) There is a curvilinear relation between age and RT interference for nonreward only. B) Relations between age and accuracy interference for each trial type. C) Relations between age and RT interference for each trial type.

There was also a significant interaction between reward and control strategy, $t(194.12) = 7.53$, $p < .001$ (Figure 10). According to a test of simple slopes (Aiken & West, 1991), in the proactive context, there was a positive effect of reward, $t(193.76) = 3.91$, $p < .001$, such that there was more interference on reward trials compared to nonreward trials. In the reactive context, there was a negative effect of reward, $t(193.80) = -20.84$ $p < .001$, such that there was less interference on reward trials compared to nonreward trials. No three-way (reward *x* control strategy *x* age) or four-way interactions (reward *x* control strategy *x* age^2) were statistically significant. Figure 9C illustrates the pattern of relations between age and flanker effect in each trial type.

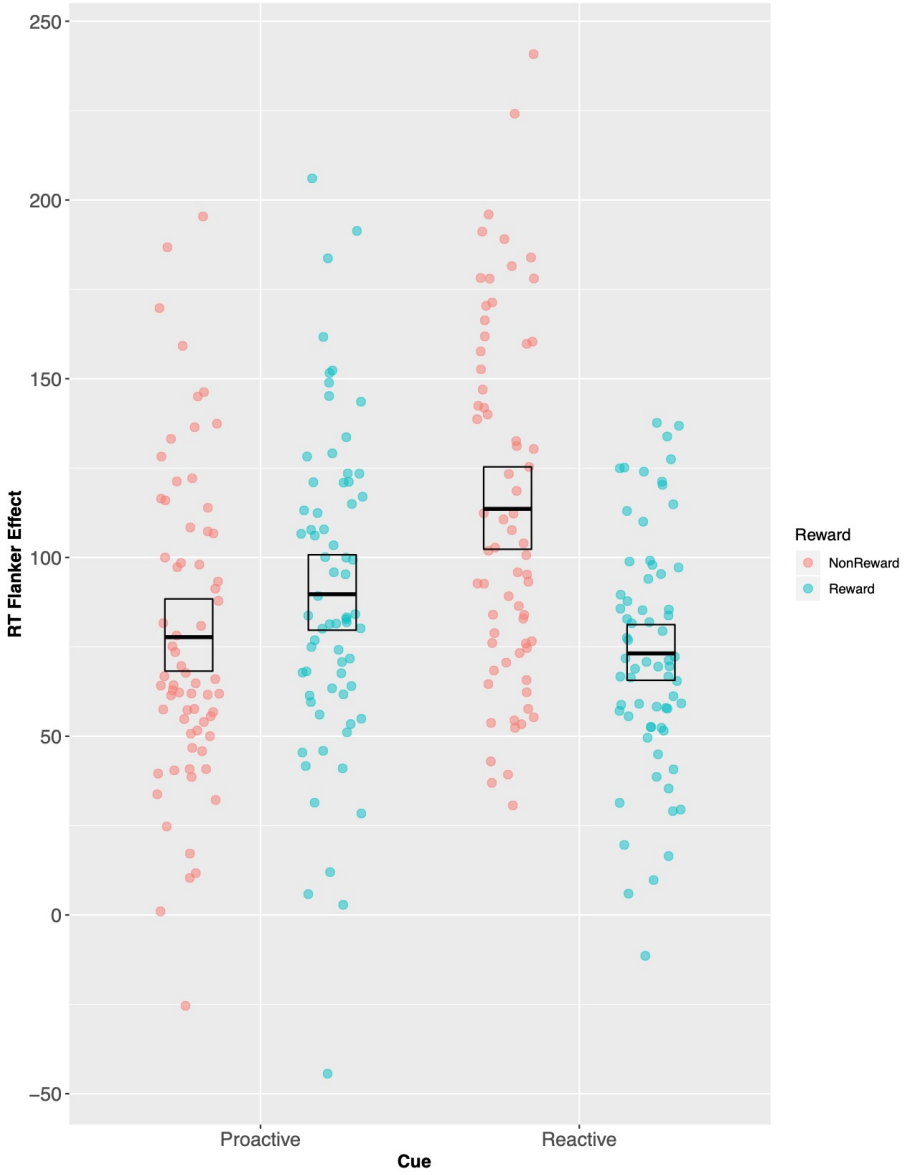


Figure 10. Interaction between reward and control strategy. In nonrewarded conditions, there was less interference on proactive trials compared to reactive trials. However, in rewarded conditions, there was more interference on proactive trials compared to reactive trials.

Puberty-Related Changes in Influence of Reward on Control Strategy for Behavioral Performance

Next, we examined the effect of reward, control strategy, puberty, and puberty^2 on the flanker effect, or interference, for behavioral performance within the reward block only. Similar to the age-related effects, there were no significant effects of reward,

control strategy, puberty, or puberty^2 on the accuracy interference but there were significant relations when predicting RT interference.

Here, there was a main effect of reward, $t(193.78)=-3.082$, $p=0.002$, such that reward is associated with faster reaction times. Consistent with the age analysis, the main effect of control strategy, $t(193.78) = -3.59$, $p < .001$, remained the same with proactive trials reducing RT interference. Reward interacted with puberty scores, $t(193.80) = 2.153$, $p = .033$, showing that RT interference decreased with puberty for reactive trials only. The interaction between reward and control strategy was also qualitatively the same, $t(193.78)= 8.61$, $p < .001$, suggesting more interference on rewarded, proactive trials. Finally, there was a significant interaction between reward, control strategy, and puberty^2, $t(193.76)$, $p = .042$. To probe this interaction, we first divided the trials by control strategy, then explored the reward *x* puberty^2 interaction. In proactive trials, there was a marginal interaction between reward and puberty^2, $t(65) = -1.96$, $p = .054$. This interaction suggests that the association between puberty and proactive RT interference is more curvilinear for reward, with RT interference peaking in mid-adolescence (Fig 11A). However, follow-up tests exploring the relation between puberty^2 and RT interference were not significant for either proactive reward or proactive nonreward. In reactive trials, the reward *x* puberty^2 interaction did not reach statistical significance, $t(65) = 0.69$, $p=0.50$. Critically, the reward *x* control strategy *x* puberty^2 interaction remained significant after controlling for age, $t(188.03) = -2.27$, $p = 0.024$, suggesting that RT interference is increased on reward proactive trials specifically in mid-puberty. Figure 11B and Figure 11C illustrate the pattern of relations between puberty and accuracy interference and between puberty and RT interference, respectively, for each trial type.

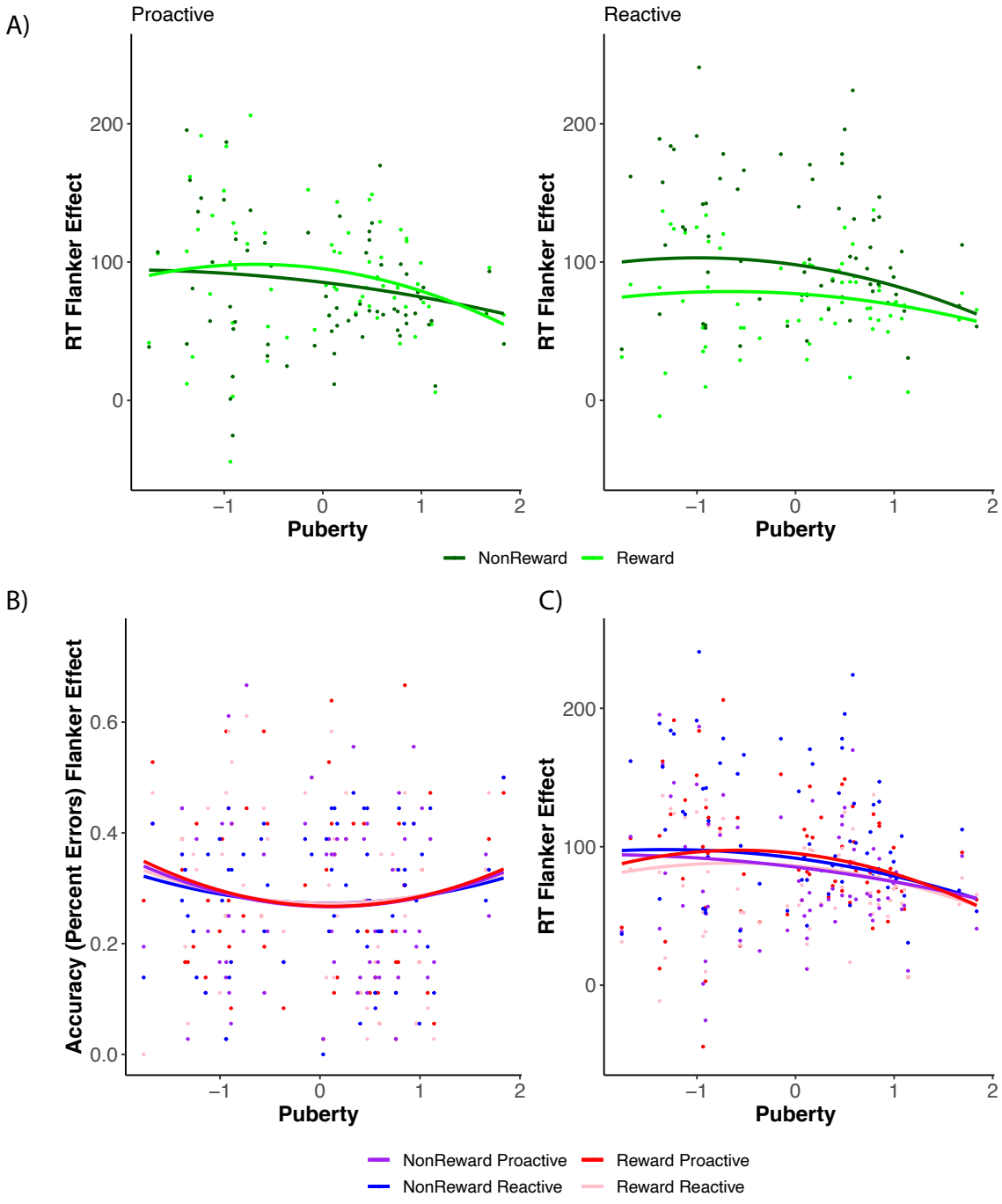


Figure 11. Puberty-related changes in RT interference. A) The interaction between cue, reward, and puberty^2 B) Relations between puberty and accuracy interference for all trial types. C) Relations between puberty and RT interference for all trial types.

Cue-Locked Theta Power

Cue-locked theta power after the presentation of the cue (either the question mark, circle, or triangle and either reward-cueing or nonreward-cueing) was analyzed in order to explore monitoring associated with proactive stimulus preparation. Figure 12 displays the time frequency surfaces and topographic plots associated with each condition.

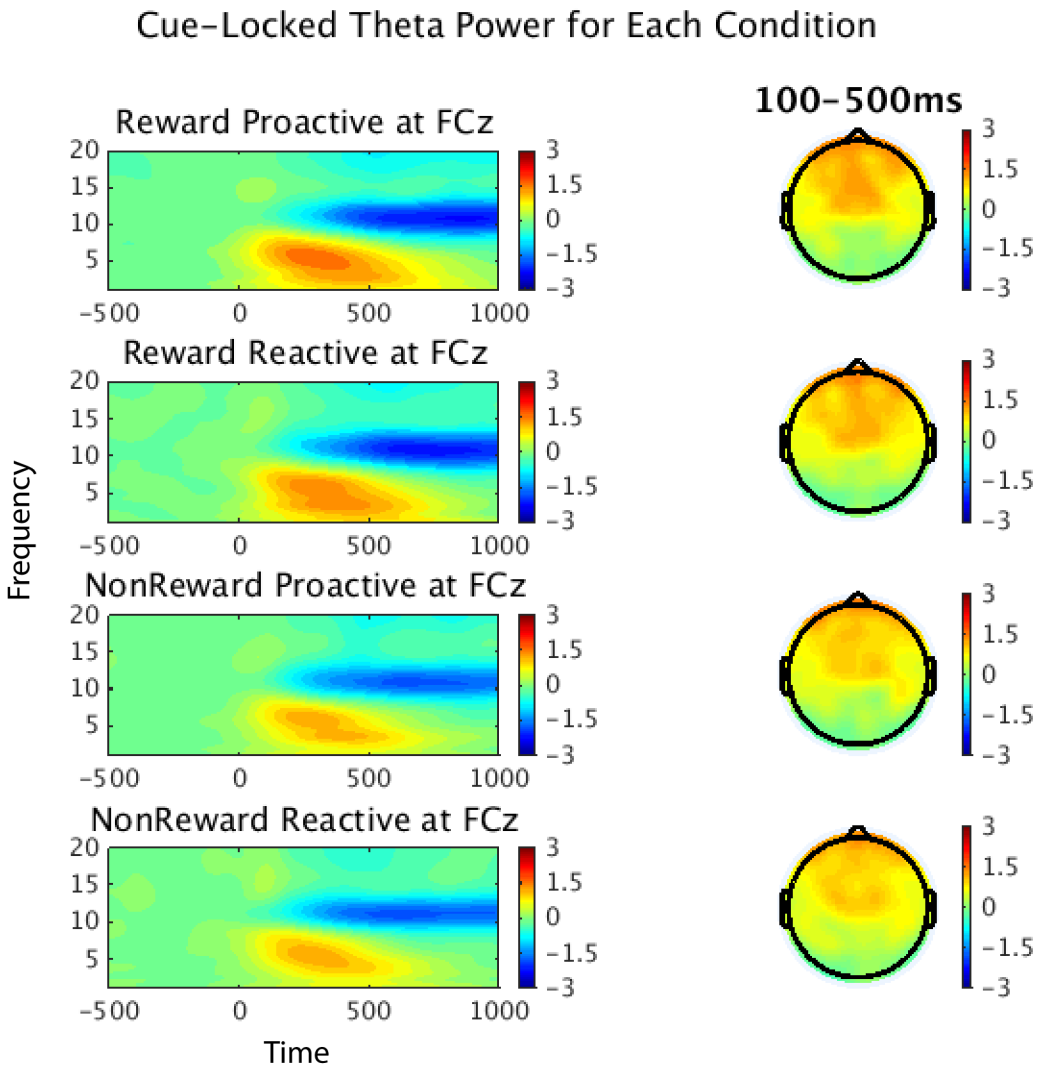


Figure 12. Time frequency surface and topographic plots for the cue-locked theta power for each condition. Topographic plots were plotted for the theta frequency at 100-500 ms. Note: these are not flanker effect plots.

Age-Related Changes in Influence of Reward on Control Strategy for Cue Theta Power

First, we examined the effects of reward, cue, age, and age^2 on cue-locked theta power. There was a main effect of reward, $t(195)=2.32$, $p = 0.021$, such that rewarded cues elicited more theta power. There was also an interaction between reward, control strategy, and age, $t(195.00) = 2.24$, $p = 0.027$. To probe this interaction, we separately examined the reward *x* age interaction in proactive vs reactive contexts. For proactive, the interaction between reward and age was significant, $t(65.98) = 2.33$, $p = 0.022$, suggesting that proactive reward cue theta power does not change with age, but proactive nonreward cue theta decreases with age. However, follow-up correlations indicated that neither correlation was significantly different from zero. In reactive contexts, the interaction between reward and puberty was not significant, $t(66) = -1.10$, $p = 0.27$.

Puberty-Related Changes in Influence of Reward on Control Strategy for Cue Theta Power

Next, we examined the effects of reward, cue, puberty, and puberty^2 on cue-locked theta power. The same main effect of reward was present, $t(195)=4.98$, $p < .001$. Additionally, there was an interaction between reward and puberty^2, $t(195)= -2.810$, $p = .005$. To probe this interaction, we examined the relation between puberty^2 and cue-locked theta power in the rewarded trials compared to nonrewarded trials. Though the interaction was significant in the omnibus model, the relation between puberty^2 and cue-locked theta failed to reach significance in either the rewarded trials, $t(65)=-1.42$, $p = 0.16$, or the nonrewarded trials, $t(65)=0.20$, $p = .84$. Though follow-up tests were not significant, the interaction suggests an inverted-u shaped relation, peaking around mid-puberty, between puberty and cue-locked theta power for rewarded trials, but a flattened

u-shaped relation between puberty and cue-locked theta power for nonrewarded trials. Finally, there was a three-way interaction between reward, control strategy, and puberty, $t(195)=2.26$, $p = .027$ (Figure 13A), similar to that seen in the age analyses. However, it is important to note that this interaction did not survive after controlling for age. Regardless, we again probed this interaction by separating proactive and reactive trials, then examining the reward *x* puberty interaction for each control strategy. In proactive contexts, there was a marginal interaction between reward and puberty, $t(66) = 1.70$, $p = 0.09$, suggesting a similar pattern seen with the age analyses. Specifically, proactive reward cue theta power does not seem to change with puberty, but proactive nonreward cue theta power decreases with puberty. In reactive contexts, the interaction between reward and puberty was also approaching significance, $t(66) = 1.64$, $p = 0.10$. Here, the interaction suggests that reactive reward cue theta power decreases with age more rapidly, while reactive nonreward cue theta power slightly decreases with age. (However, no follow-up correlations from these interactions between reward and puberty in proactive vs reactive contexts was significantly different from 0). Figure 13B and Figure 13C illustrate the pattern of relations between age and cue theta power and between puberty and cue theta power respectively, for each trial type.

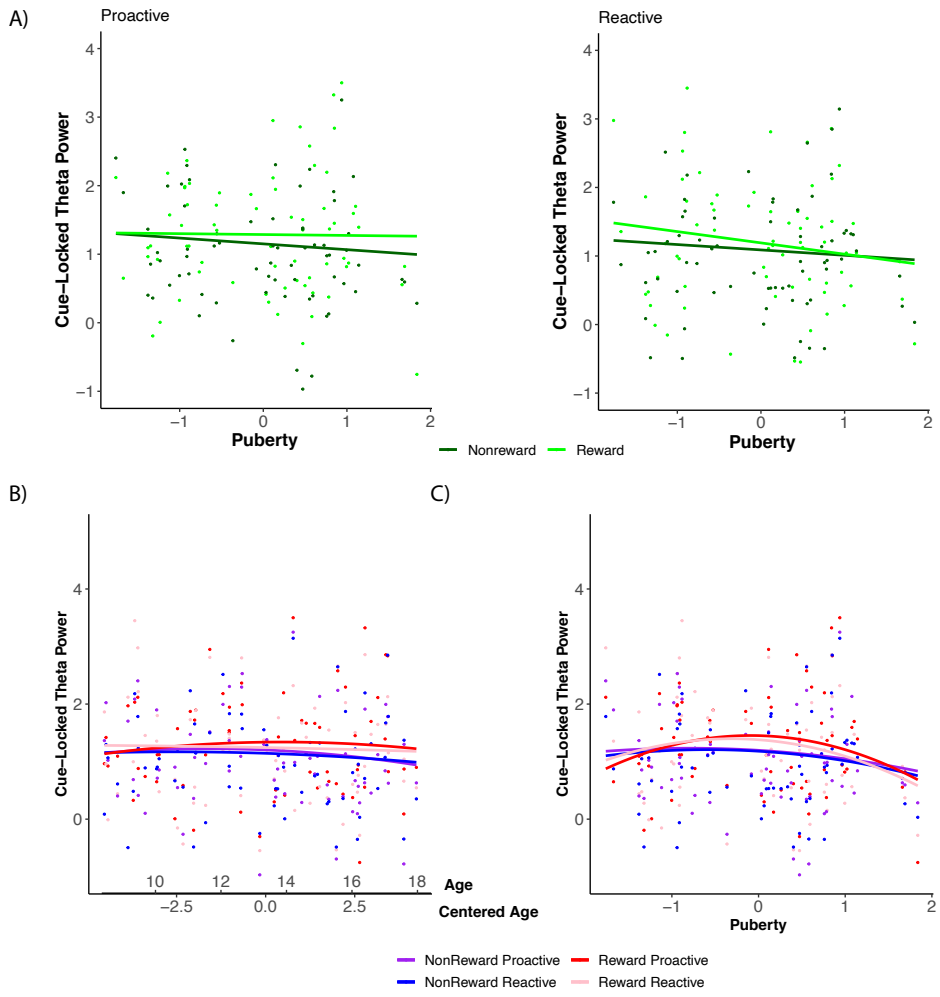


Figure 13. Effects of age and puberty on cue-locked theta power. A) Control strategy x reward x puberty interaction B) Relation between age and cue-locked theta power for and C) between puberty and cue-locked theta power for each trial type with summary lines based on MLM model parameters.

Cue-Locked Theta ICPS

Cue-locked theta ICPS after the presentation of the cue (either the question mark, circle, or triangle and either reward-cueing or nonreward-cueing) was analyzed in order to explore control recruitment associated with proactive stimulus preparation. Figure 14 displays the time frequency surfaces and topographic plots associated with each condition.

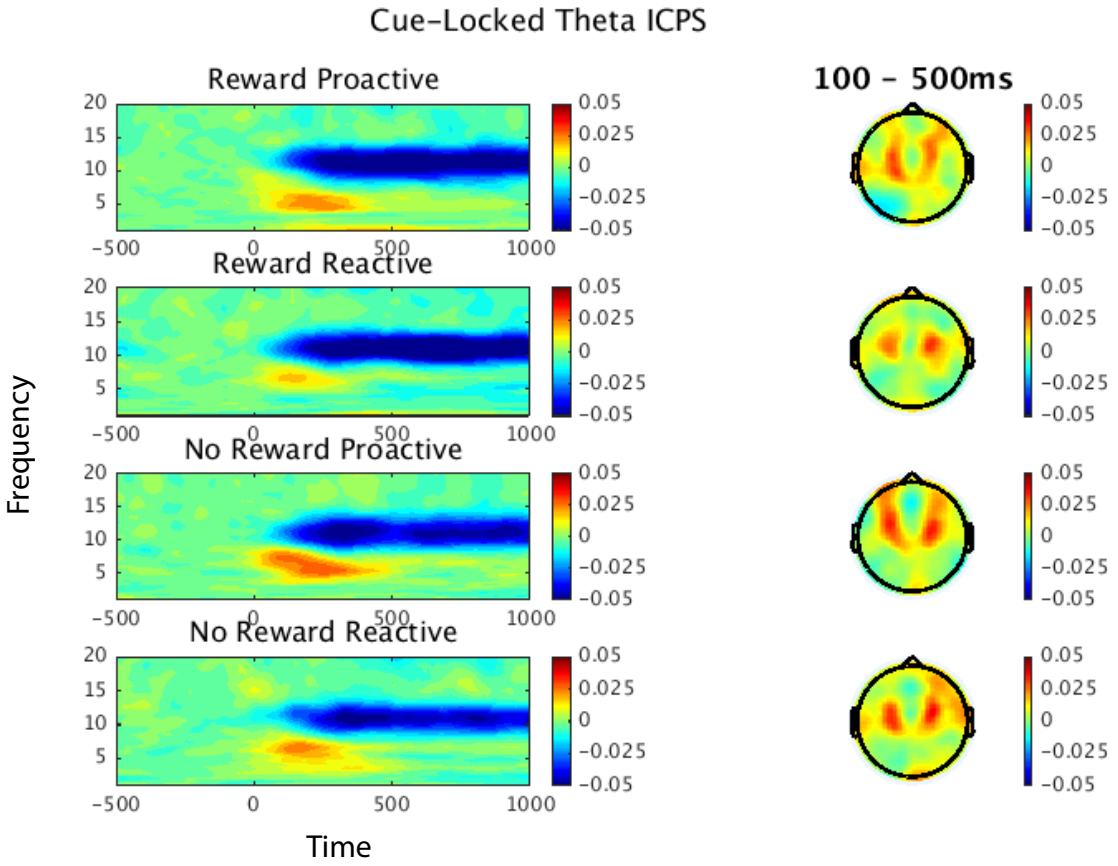


Figure 14. Time frequency surface and topographic plots for the cue-locked theta ICPS for each condition. Topographic plots were plotted for the theta frequency at 100-500 ms, consistent with cue-locked theta power. Note: these are not flanker effect plots.

Age-Related Changes in Influence of Reward on Control Strategy for Cue Theta ICPS

First, we examined the effects of reward, cue, age, and age^2 on cue-locked theta ICPS. There was a main effect of reward, $t(190.70) = -2.86$, $p = 0.005$, such that reward is associated with a decrease in cue theta ICPS. Further, there was a significant reward x age^2 interaction, $t(190.30) = 2.13$, $p = .034$ (Fig 15A). To probe this interaction, we explored the relation between age^2 and cue theta ICPS in reward and nonreward, separately. While neither follow-up test was significant, the pattern of results suggests that, for nonreward, there is a curvilinear relation between age and cue theta ICPS that reaches a peak in mid-adolescence. This developmental pattern mirrors prior research

suggesting that connectivity increases throughout adolescence (Luna, Paulsen, Padmanabhan, & Geier, 2013). However, for reward, there was little change in theta ICPS throughout pubertal development. No three-way interactions were significant.

Puberty-Related Changes in Influence of Reward on Control Strategy for Cue Theta ICPS

Next, we examined the effects of reward, cue, puberty, and puberty^2 on cue-locked theta ICPS. For this analysis, the main effect of reward became marginal, $t(190.80) = -1.812$, $p = 0.071$. The reward effect mirrored the main effects reported above, with reward associated with less theta ICPS. No other effects or interactions reached statistical significance. Figure 15B and Figure 15C illustrate the pattern of relations between age and cue theta ICPS and between puberty and cue theta ICPS respectively, for each trial type.

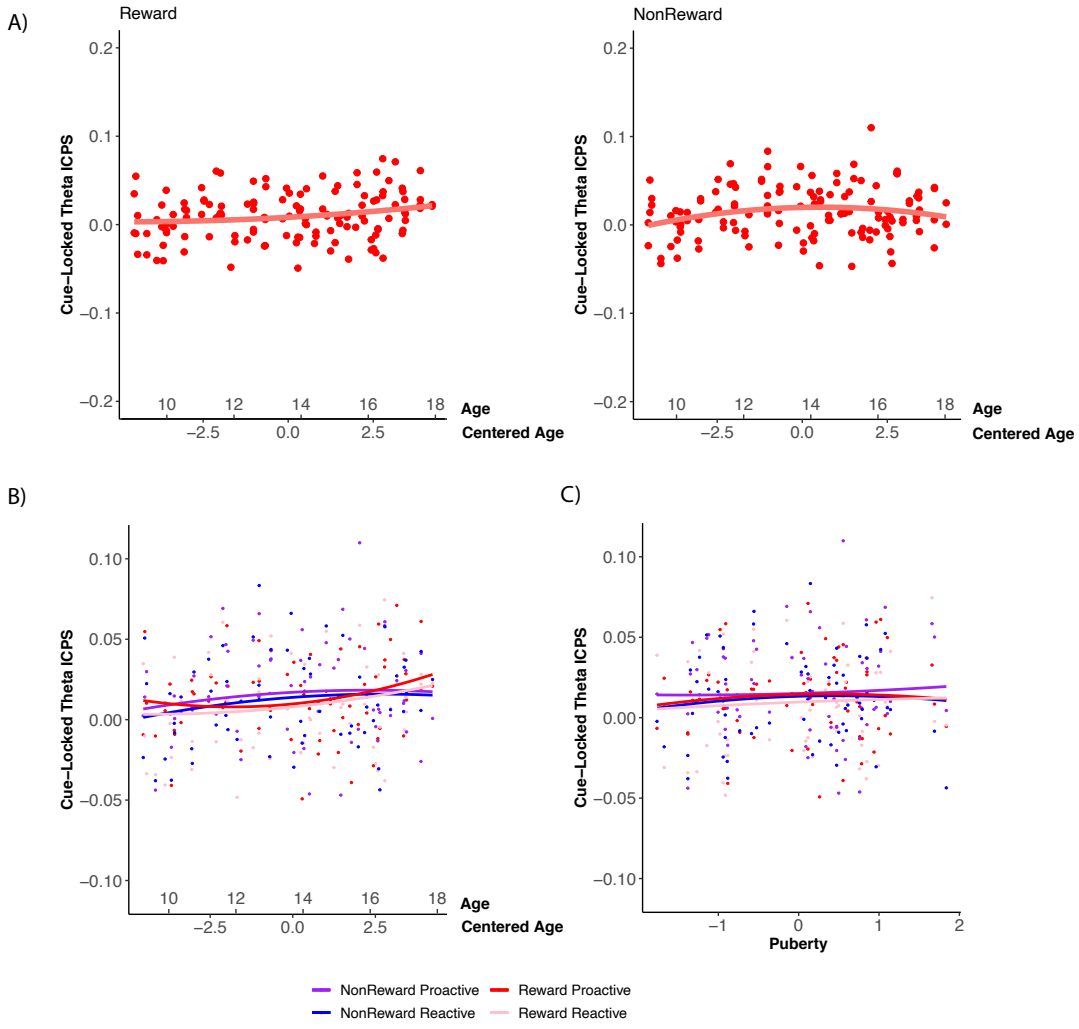


Figure 15. Effects of age and puberty on cue-locked theta ICPS. A) Reward x puberty^2 interaction B) Relation between age and cue-locked theta ICPS for each trial type. C) Relation between puberty and cue-locked theta ICPS for each trial type with summary lines based on MLM model parameters.

Stim-Locked Theta Power

Stim-locked theta power after the presentation of the stimulus (either congruent or incongruent) was analyzed in order to explore monitoring associated with reactive response preparation. Stim-locked theta power scores are calculated as flanker scores (incongruent – congruent). Figure 16 displays the time frequency surfaces and

topographic plots associated with the stim-locked theta power flanker effect in each condition.

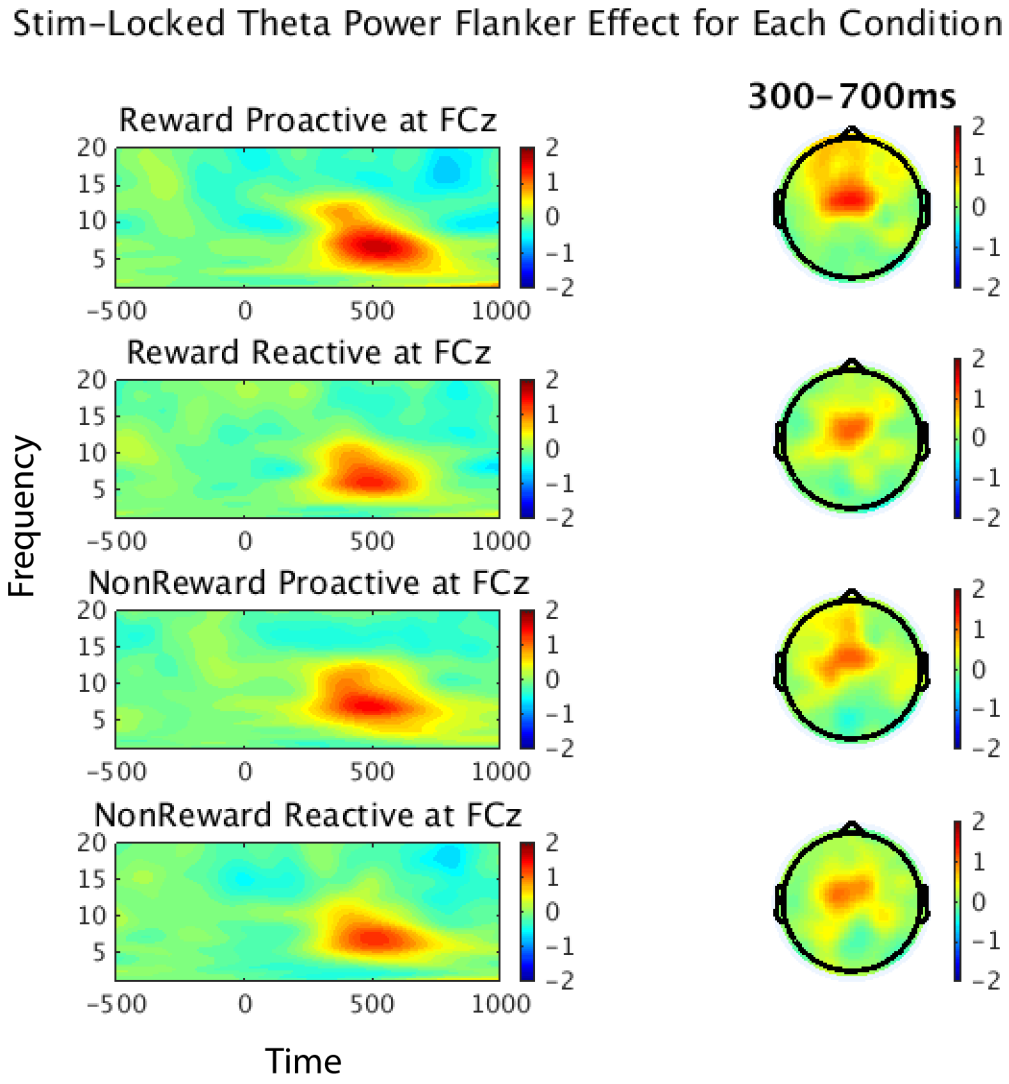


Figure 16. Time frequency surface and topographic plots for the stim-locked theta power flanker effect for each condition. Topographic plots were plotted for the theta frequency at 300-700 ms.

Age-Related Changes in Influence of Reward and Control Strategy for Stim-Locked Theta Power

First, we examined the effects of reward, cue, age, and age^2 on stim-locked theta power flanker effect. There was a main effect of control strategy, $t(193.60) = -7.43$, $p < 0.001$, such that proactive was associated with less stim theta power interference. Additionally, there was a main effect of age, $t(65.13)=2.296$, $p = 0.025$, revealing that stim theta power interference increased with age. Finally, there was a control strategy *x* age interaction, $t(193.5) = - 4.11$, $p < 0.001$. Probing this interaction by separating proactive from reactive revealed that there was only a significant relation between age and stim theta power interference for reactive trials, $t(67.19) = 3.494$, $p = 0.0008$, such that stim theta power interference increased with age specifically in a reactive context (Fig 17A).

Puberty-Related Changes in Influence of Reward and Control Strategy for Stim-Locked Theta Power

Next, we examined the effects of reward, cue, puberty, and puberty^2 on stim-locked theta power flanker effect, or interference. These effects closely mirrored the effects found in the age analysis. There was a main effect of control strategy, $t(193.27) = -7.91$, $p < 0.001$, that stim theta power interference was reduced for the proactive context. While there was no main effect of puberty, there was a control strategy *x* puberty interaction, $t(193.64) = -4.36$, $p < 0.001$, which remained significant after controlling for age. In proactive contexts, there is no relation between puberty and stim theta power interference; however, in reactive contexts, stim theta power interference increases with

age, $t(67.19) = 3.49$, $p < 0.001$ (Fig 16A). Figure 17B and Figure 17C illustrate the pattern of relations between age and stim theta power flanker effect and between puberty and stim theta power flanker effect respectively, for each trial type.

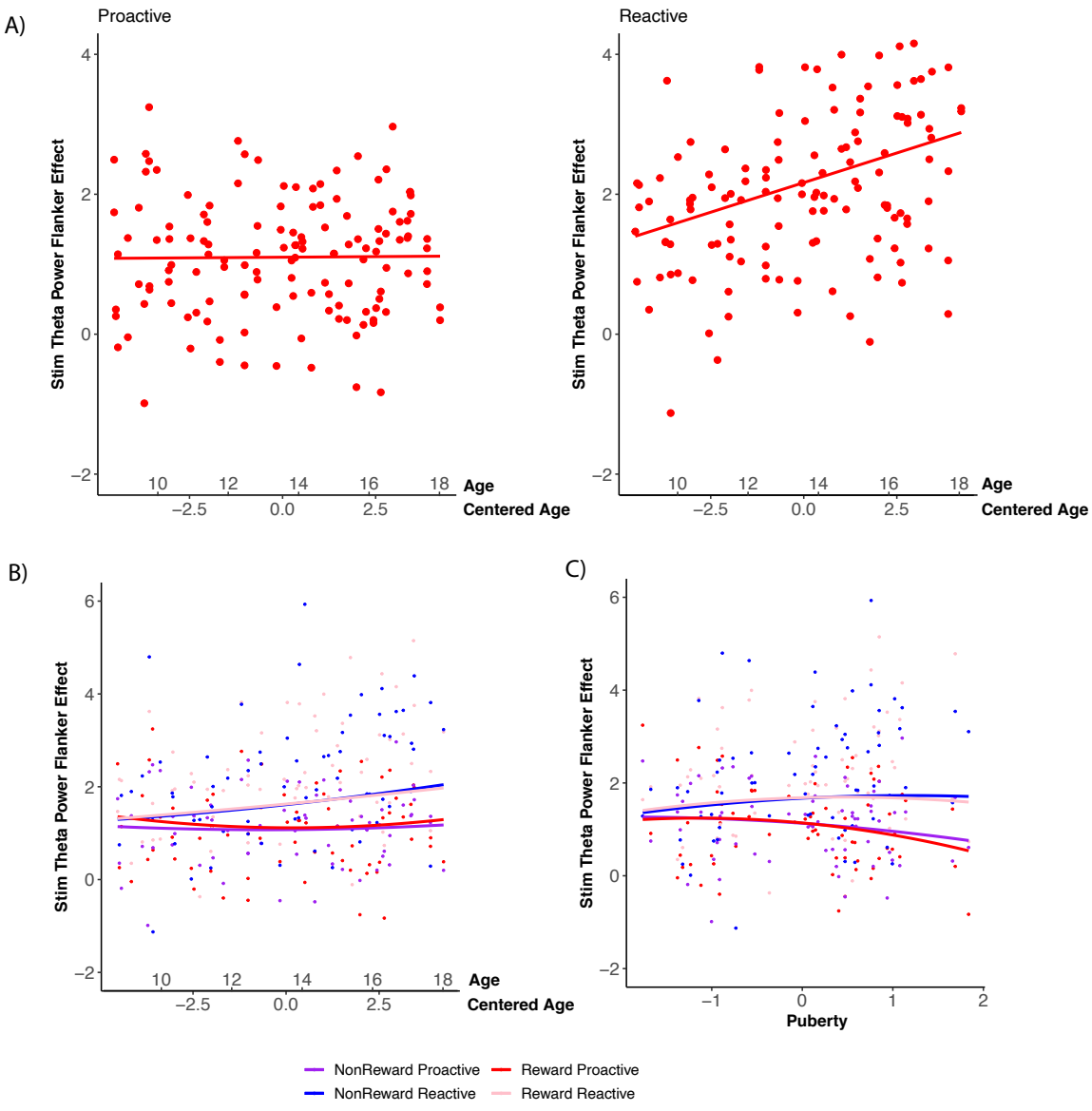


Figure 17. Effects of age and puberty on stim theta power flanker effect. A) Control strategy x Age interaction. B) Relation between age and stim theta power flanker effect by trial type. C) Relation between puberty and stim theta power by trial type.

Stim-Locked Theta ICPS

Stim-locked theta ICPS after the presentation of the stimulus (either congruent or incongruent) was analyzed in order to explore monitoring associated with reactive response preparation. Stim-locked theta ICPS scores are calculated as flanker scores (incongruent – congruent). Figure 18 displays the time frequency surfaces and topographic plots associated with the stim-locked theta ICPS flanker effect in each condition.

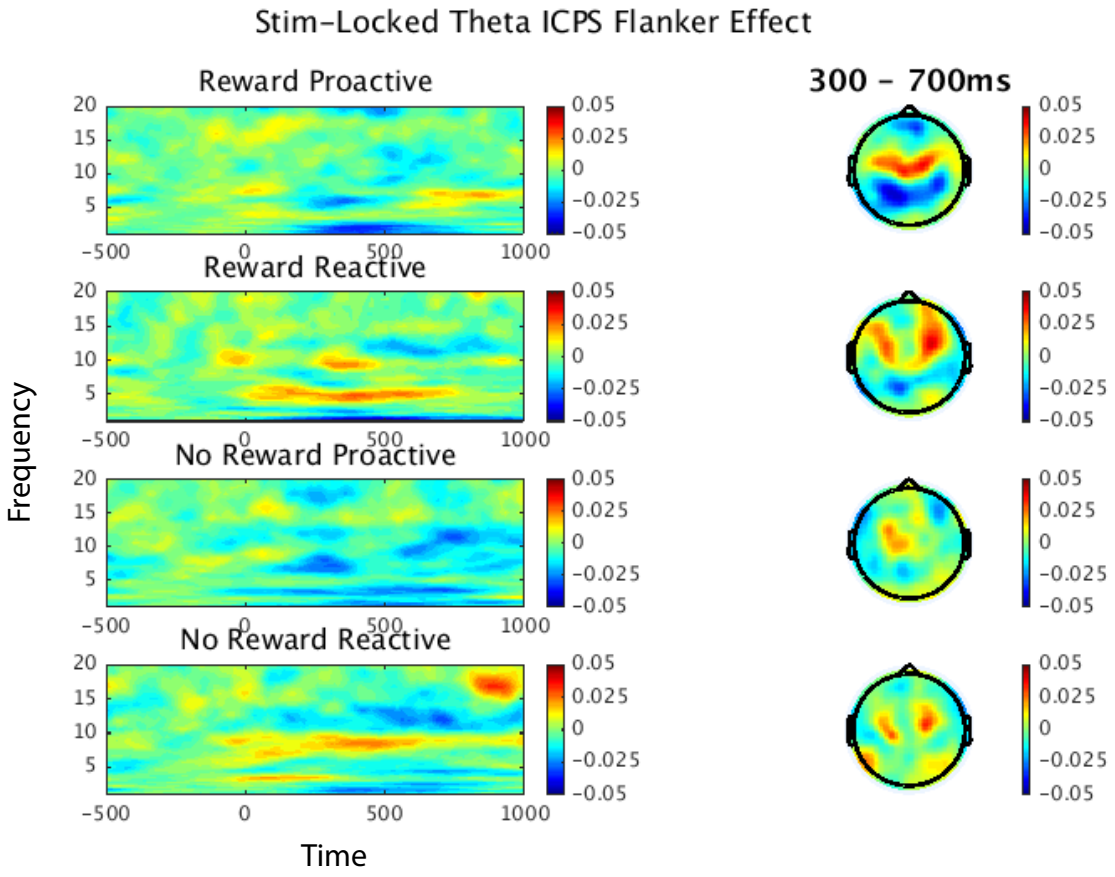


Figure 18. Time frequency surface and topographic plots for the stim-locked theta ICPS flanker effect for each condition. Topographic plots were plotted for the theta frequency at 300-700 ms, consistent with stim-locked theta power.

Age-Related Changes in Influence of Reward and Control Strategy for Stim-Locked Theta ICPS

First, we examined the effects of reward, control strategy, age, and age^2 on stim-locked theta ICPS flanker effect. There were no main effects or interactions.

Puberty-Related Changes in Influence of Reward and Control Strategy for Stim-Locked Theta ICPS

Next, we examined the effects of reward, control strategy, puberty, and puberty^2 on stim-locked theta ICPS flanker effect. There were no main effects. There was a reward *x* puberty interaction, $t(195.00) = -2.02$, $p = 0.044$, which remains significant when controlling for age, $t(189.00) = -2.54$, $p = 0.011$ (Fig 19A). To probe this interaction, we examined the relation between puberty and stim theta ICPS in reward and nonreward separately. The omnibus interaction was significant because the relation between puberty and stim theta ICPS was negative for reward, but positive for nonreward; however, neither follow-up test was statistically significant. Figure 19B and Figure 19C illustrate the pattern of relations between age and stim theta ICPS interference and between puberty and stim theta ICPS interference respectively, for each trial type.

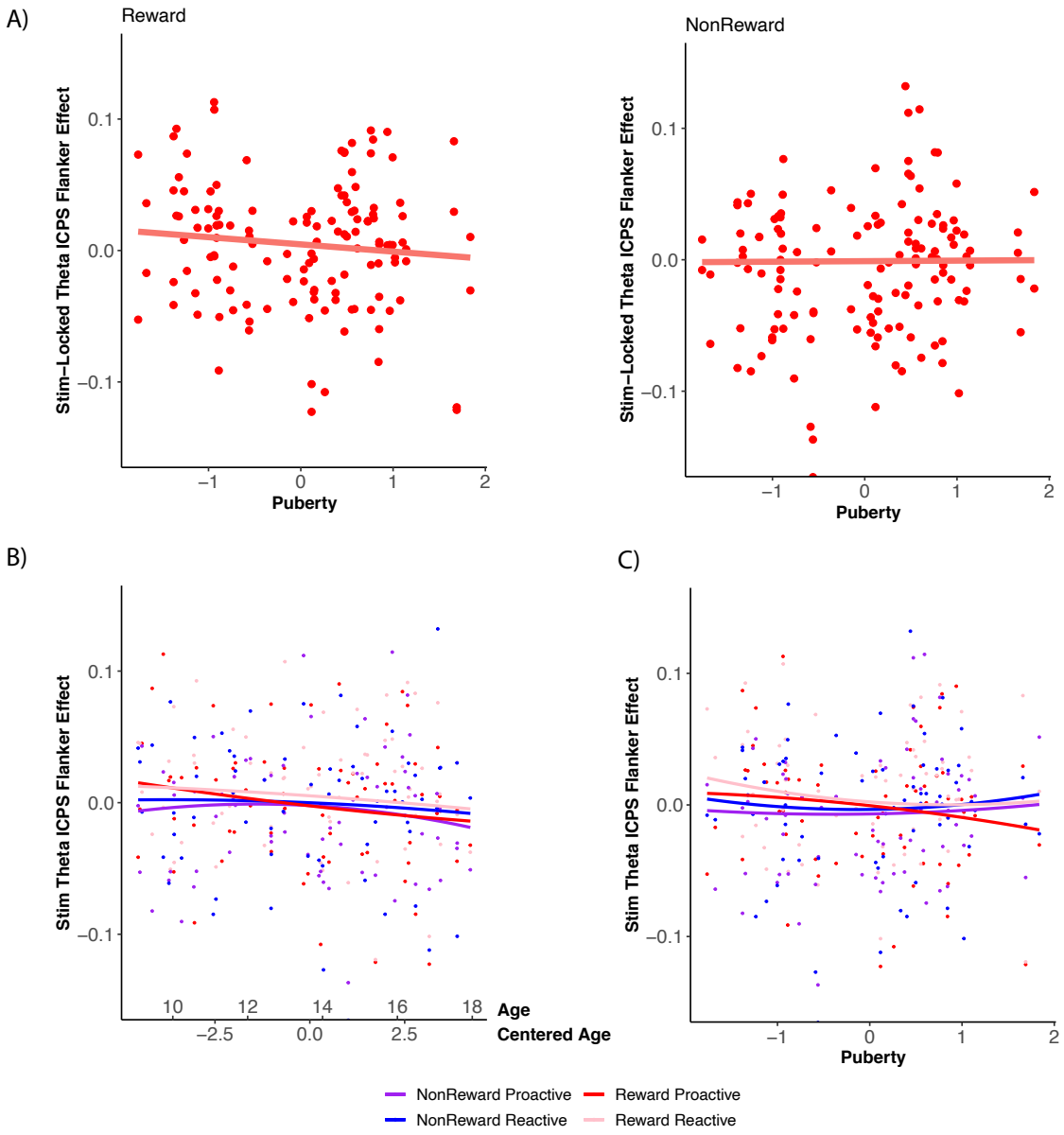


Figure 19. Age and puberty relations with Stim Theta ICPS Flanker Effect. A) Reward x puberty interaction. B) Relations between age and stim theta ICPS flanker effect by trial type. C) Relations between puberty and stim theta ICPS by trial type.

Brain-Behavior Relations

After exploring the effects of the task and age/puberty on behavioral performance and neural measures of interest, we then wanted to understand how the neural measures impact behavioral performance, above and beyond age or puberty alone, and if those

relations change as a function of age or puberty. Because we are interested in the reward manipulation, we specifically investigated how reward-related EEG activity influences reward-related behavioral performance by calculating reward – nonreward difference scores for proactive RT interference and reactive RT interference separately. We again focused on RT interference as our behavioral performance measure of interest because task effects were seen in RT interference, not accuracy interference. Appendix D details regressions that tested how each individual EEG measure predicts reward-related RT interference.

Brain-Behavior Relations when accounting for Age

In the first block of predictors in the hierarchical regression predicting reward-related proactive RT interference, age and age^2 together explained a marginal amount of variance in reward-related proactive RT interference, $R^2 = 0.083$, $F(2,57) = 2.57$, $p = 0.085$. Only age^2 reached significance as a predictor. The curvilinear relation between age and reward-related proactive RT interference was concave, $b = -1.60$, $p = 0.033$, suggesting a peak in reward-related proactive RT interference in mid-adolescence. When adding the second block of predictors that included the four reward-related proactive EEG measures, R^2 was significantly increased, $p = 0.004$. Increased reward-related proactive cue theta was associated with increased reward-related proactive RT interference, $b = 12.72$, $p = 0.03$. Additionally, increased reward-related proactive stim theta ICPS interference predicts reduced reward-related proactive RT interference, $b = -241.33$, $p = 0.0003$. The third block of predictors, including interactions with age, did not significantly increase R^2, $p = 0.46$. Table 7 details the hierarchical regression.

No block of predictors significantly explained variance in reward-related reactive RT interference (Table 8).

Table 7

Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference with Age

	Reward-Related Proactive RT Interference					
Variable	**Block 1**			**Block 2**		
	B	SE (B)	*β*	B	SE(B)	*β*
Intercept	23.10**	5.82	0.00	20.99**	5.92	0.00
Age	- 1.08	1.56	- 0.08	- 2.98†	1.49	- 0.24
Age^2	- 1.60*	0.73	- 0.29	- 1.34†	0.71	- 0.22
Rew-Rel Pro Cue Power				12.72*	5.72	0.27
Rew-Rel Pro Cue ICPS				- 15.54	104.65	- 0.02
Rew-Rel Pro Stim Power				- 2.93	4.13	- 0.08
Rew-Rel Pro Stim ICPS				- 241.33**	62.44	- 0.46
F stat			2.57†			4.03**
R^2			8.28			31.30
ΔR^2 *F stat*						4.43**

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained. Block 1 displays regression estimates for the block including age and age^2. Block 2 displays regression estimates and R^2 change for age, age^2, and the four EEG measures. Block 3 is not displayed because it was not significant (see Table E1 for Block 3).

Table 8

Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference with Age

	Reward-Related Reactive RT Interference					
Variable	**Block 1**			**Block 2**		
	B	SE(B)	*β*	B	SE(B)	*β*
Intercept	- 32.62**	6.11	0.00	- 31.92**	6.67	0.00
Age	2.43	1.64	0.19	2.34	1.67	- 0.03
Age^2	- 0.47	0.77	- 0.08	- 0.47	0.81	- 0.08
Rew-Rel Re Cue Power				- 0.99	5.91	- 0.02
Rew-Rel Re Cue ICPS				164.61	146.64	0.16
Rew-Rel Re Stim Power				1.31	4.11	0.04
Rew-Rel Re Stim ICPS				55.53	75.41	0.10
F stat			1.32			0.70
R^2			4.41			7.35
ΔR^2 *F stat*						0.42

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained. Block 1 displays regression estimates for the block including age and age^2. Block 2 displays regression estimates and R^2 change for age, age^2, and the four EEG measures. Block 3 is not displayed because it was not significant (see Table E2 for Block 3).

Brain-Behavior Relations when accounting for Puberty

In the first block of predictors in the hierarchical regression predicting reward-related proactive RT interference, puberty and puberty^2 did not explain a significant amount of variance in reward-related proactive RT interference, $R^2 = 0.04$, $F(2,57) = 1.15$, $p = 0.32$. Adding the second block of EEG measures did explain a significant amount of variance in reward-related proactive RT interference, $R^2 = 0.30$, $F(6,53) = 3.60$, $p = 0.005$, which was a significant increase in R^2, $p = 0.004$. Here, only reward-related stim theta ICPS had a significant negative association with reward-related

proactive RT interference, b = -265.63, $p < .001$. Adding the third block of interactions did not significantly increase R^2, $p = 0.74$. Tables 9 details the hierarchical regression predicting reward-related proactive RT interference accounting for puberty.

No block of predictors was significant in predicting reward-related reactive RT interference (Table 10).

Table 9

Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference with Puberty

	Reward-Related Proactive RT Interference					
Variable	**Block 1**			**Block 2**		
	B	SE (B)	*β*	B	SE(B)	*β*
Intercept	19.03**	5.43	0.00	19.01**	5.82	0.00
Puberty	- 1.24	4.40	- 0.04	- 6.46	4.10	- 0.19
Puberty^2	- 7.11	4.70	- 0.20	- 7.56	4.57	- 0.21
Rew-Rel Pro Cue Power				7.61	6.12	0.16
Rew-Rel Pro Cue ICPS				- 110.96	99.68	- 0.13
Rew-Rel Pro Stim Power				- 4.48	4.21	- 0.13
Rew-Rel Pro Stim ICPS				- 265.13**	63.81	- 0.51
F stat			1.15			3.60**
R^2			3.88			28.98
ΔR^2 *F stat*						4.68**

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained. Block 1 displays regression estimates for the block including puberty and puberty^2. Block 2 displays regression estimates and R^2 change for puberty, puberty^2, and the four EEG measures. Block 3 is not displayed because it was not significant (see Table E3 for Block 3).

Table 10

Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference with Puberty

	Reward-Related Reactive RT Interference					
Variable	**Block 1**			**Block 2**		
	B	SE(B)	*β*	B	SE(B)	*β*
Intercept	- 38.08**	5.52	0.00	- 36.90**	6.23	0.00
Puberty	8.29	4.45	0.24	8.19†	4.60	0.24
Puberty^2	3.37	4.77	0.09	2.11	5.12	0.06
Rew-Rel Re Cue Power				0.63	6.02	0.01
Rew-Rel Re Cue ICPS				133.71	149.93	0.13
Rew-Rel Re Stim Power				1.76	4.01	0.06
Rew-Rel Re Stim ICPS				60.78	75.65	0.11
F stat			1.87			0.85
R^2			6.15			8.80
ΔR^2 *F stat*						0.37

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained. Block 1 displays regression estimates for the block including puberty and puberty^2. Block 2 displays regression estimates and R^2 change for puberty, puberty^2, and the four EEG measures. Block 3 is not displayed because it was not significant (see Table E4 for Block 3).

Aim 2: Relations between EF and Reward-related Proactive and Reactive Control

Predicting Reward-Related RT Interference from Executive Function

In Aim 2, we wanted to explore the relation between EF ability, namely inhibitory control (IC), set-shifting (SS), and working memory (WM), and reward-related behavior in the context of proactive and reactive control. Centered predictors of age, age^2, all EF measures, interactions between age and EF measures, and interactions between age^2 and EF measures were entered in the path model predicting reward-related proactive RT

interference and reward-related reactive RT interference (Figure 20). Table 11 details the path estimates from this path model.

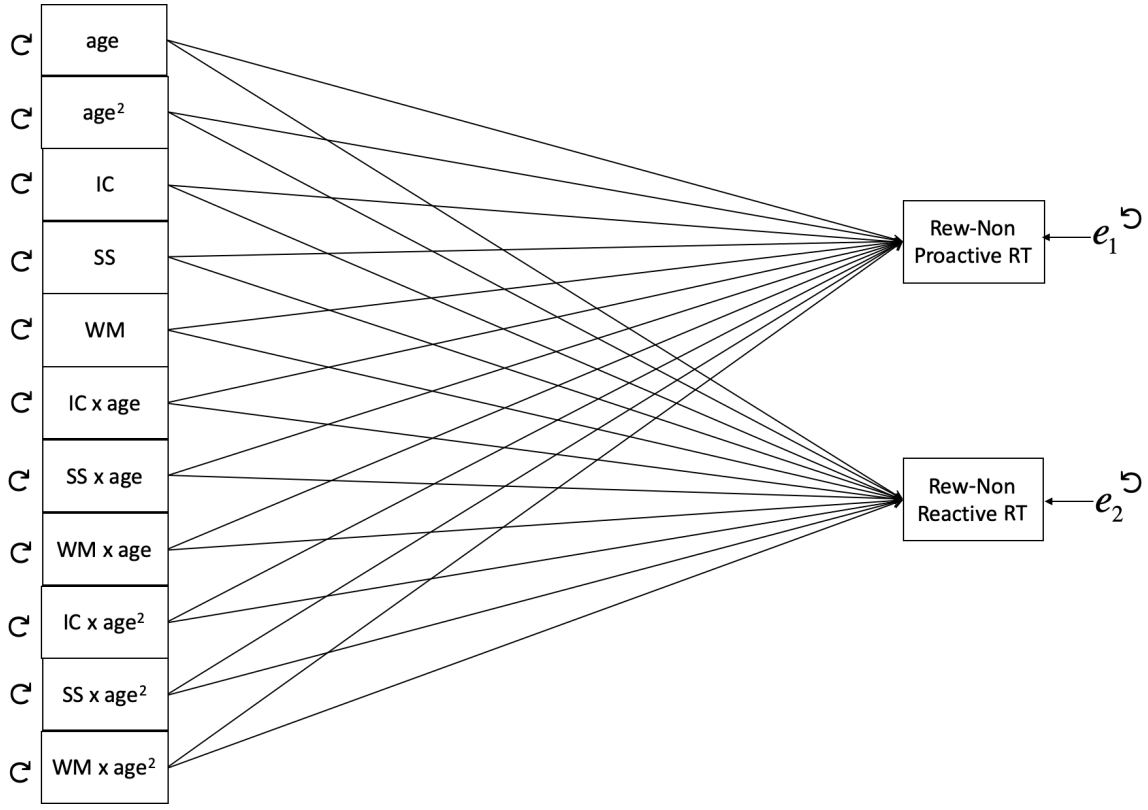


Figure 20. Path diagram depicting relations between age, age^2, EF and reward-related proactive performance, and reward-related reactive performance. Residual correlations (though not pictured) were included between all predictors and between the two outcomes.

Reward-Related Proactive RT Interference

When predicting reward-related proactive RT interference, there were no significant effects of age, age^2, or EF alone. However, there was a significant IC *x* age^2 interaction, $z = 2.68$, $p = 0.007$. Simple slope analyses (Table 12) revealed that, at low levels of inhibitory control, there is a significant effect of age^2 on reward-related proactive RT interference, *Wald* = -3.49, $p < 0.001$, such that for males with low levels of IC there is more interference in mid adolescence (Fig 21A). Simple slopes of mid and high levels of IC were not significant. Moreover, there was a significant SS *x* age^2

interaction, z = -3.00, p = 0.003. Here, simple slope analyses revealed significant relations between age^2 and reward-related proactive RT interference only at high levels of set-shifting (Table 13). At high levels of SS, younger and older adolescents had the least amount of reward-related proactive RT interference (Fig 21B).

Table 11

Regression paths from the path model of age and EF predicting task reaction time (RT).

	Reward-Related Proactive RT Interference			**Reward-Related Reactive RT Inteference**		
Predictors	Estimate	95% CI	z	Estimate	95% CI	z
Age	1.263	[-2.217,4.743]	0.711	6.414**	[2.111,10.717]	2.921
Age^2	- 1.160	[-2.660,0.341]	- 1.515	- 1.073	[-2.415,0.270]	- 1.566
WM	0.378	[-6.208,6.964]	0.112	5.529**	[1.678,9.379]	2.814
IC	- 14.859	[-33.110,3.391]	- 1.596	10.179	[-9.392,29.749]	1.019
SS	- 1.057	[-10.071,7.958]	- 0.230	- 6.149	[-15.626,3.329]	- 1.272
WM x Age	0.071	[-0.605,0.747]	0.207	- 1.580**	[-2.380,-0.780]	- 3.872
IC x Age	- 3.051	[-6.997,0.895]	- 1.516	1.737	[-4.381,7.854]	0.556
SS x Age	3.114	[-0.210,6.438]	1.836	3.430	[-0.837,7.854]	1.575
WM x Age^2	0.225	[-0.400,0.850]	0.706	- 0.720**	[-1.178,-0.262]	- 3.081
IC x Age^2	2.967**	[0.793,5.142]	2.675	- 0.302	[-2.893,2.290]	- 0.226
SS x Age^2	- 2.346**	[-3.881,-0.811]	- 2.996	- 0.760	[-3.214,1.694]	- 0.607

† p < .10, *p<.05, **p<.01. Estimates = unstandardized estimates. All predictors were centered. *p* values meet statistical significance with adjustment for multiple comparisons (q < .05; FDR).

Table 12

Simple slope analysis of relation between age^2 and reward-related reactive RT interference as a function of inhibitory control

Inhibitory Control Level	**Slope**	**SE**	**Wald**
Low IC (-1SD)	- 3.148**	0.901	- 3.492
Mid IC	- 1.160	0.766	- 1.515
High IC (+1 SD)	0.828	1.210	0.684

† p < .10, *p<.05, **p<.01.

Table 13

Simple slope analysis of relation between age^2 and reward-related reactive RT interference as a function of set-shifting

Set-Shifting Level	Slope	SE	Wald
Low SS (-1SD)	1.515	1.400	1.082
Mid SS	- 1.160	0.766	- 1.515
High SS (+1 SD)	- 3.834**	0.898	- 4.270

† p < .10, *p<.05, **p<.01.

Reward-Related Reactive RT Interference

When predicting reward-related reactive RT interference, both increasing age, z = 2.92, p = 0.003, and greater working memory ability, z = 2.814, p = 0.005, were associated with greater reward-related reactive RT interference. Further, there was an interaction between age^2 and working memory, z = -3.08, p = 0.002 (Fig 21C). Simple slope analyses revealed that, for high levels of working memory, there was a significant relation between age^2 and reward-related reactive interference with a peak in interference in mid-adolescence. Simple slopes were not significant for low or mid levels of working memory (Table 14).

When substituting puberty for age in the path analysis, there were no significant effects (Appendix F).

Table 14

Simple slope analysis of relation between age^2 and reward-related reactive RT interference as a function of working memory

Working Memory Level	Slope	SE	Wald
Low WM (-1SD)	0.770	0.908	0.848
Mid WM	- 1.073	0.684	- 1.566
High WM (+1 SD)	- 2.916**	0.911	- 3.201

† p < .10, *p<.05, **p<.01.

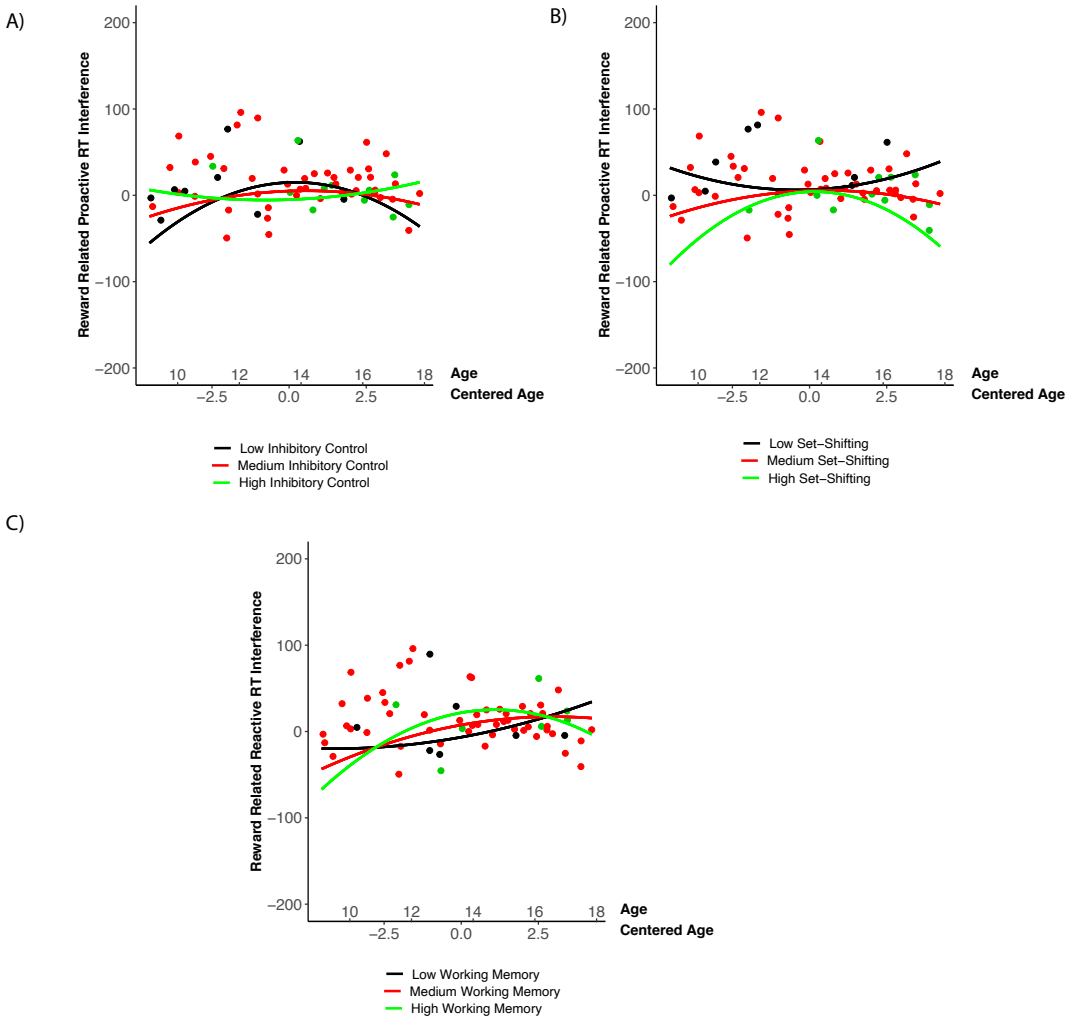


Figure 21. Relation between age^2 and reward-related RT interference in proactive and reactive contexts at varying levels of EF ability.

Mediation of Relations between EF and Reward-Related RT Interference through Monitoring or Control Recruitment

In Aim 1, we established that, in proactive contexts, both cue theta power and stim theta ICPS interference predicted reward-related RT interference. Thus far in Aim 2, we have also established that two interactions – one between IC and age^2 and another between SS and age^2 – also predict reward-related proactive RT interference. Next, we

examined if one of these neural measures mediates the relation between EF and reward-related proactive RT interference.

While reward-related cue theta power did not mediate the relation between IC x age^2, indirect effect: $b = 0.19$, $z = 0.98$, $p = 0.33$, reward-related stim theta ICPS interference marginally mediated the relation between IC x age^2 and reward-related proactive RT interference (Fig 21). Because this indirect effect did not reach statistical significance, it will not be interpreted here.

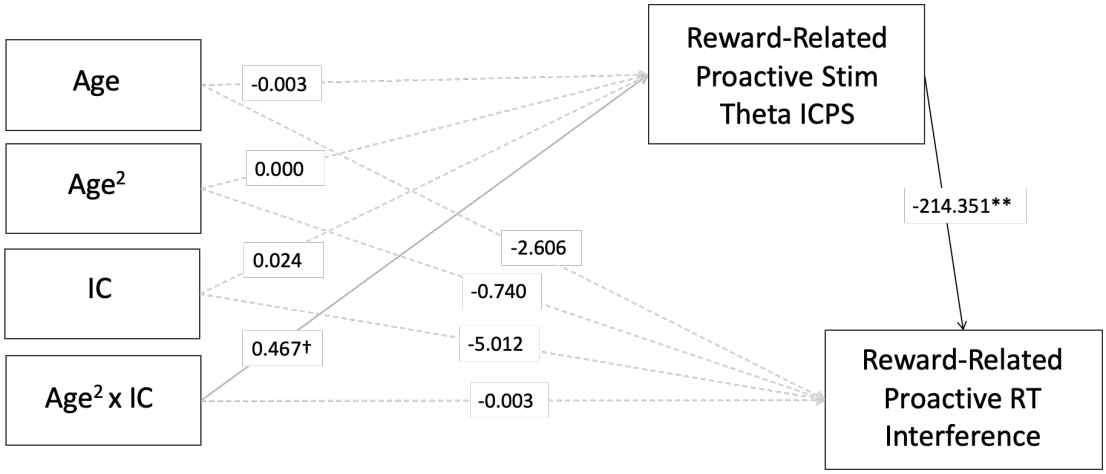


Figure 22. Mediation of inhibitory control's relation with reward-related proactive RT interference through reward-related proactive stim theta ICPS interference.

Neither reward-related proactive cue theta power nor reward-related proactive stim theta ICPS interference mediated the relation between SS x age^2 and reward-related proactive interference. See Appendix G for null results of mediation models.

Chapter 5: Discussion

The goal of the current study is to examine the effects of reward on theta oscillations during proactive and reactive control in adolescent males and how executive functions (EF) impact the interplay of reward and cognitive control. First, it was hypothesized that reward will enhance proactive control recruitment, as measured by cue-locked theta inter-channel phase synchrony (ICPS), after informative cues, especially in mid-adolescence. In the absence of informative cues, reward will upregulate the recruitment of reactive control, quantified using stimulus-locked theta ICPS, particularly in mid-adolescence. Second, it was hypothesized that cue-locked theta ICPS would predict reward-related performance in proactive contexts, but stimulus-locked theta ICPS would predict reward-related performance in reactive contexts. Finally, it was hypothesized that working memory would support reward-related performance in proactive contexts, but inhibitory control would bolster reward-related performance in reactive contexts, as mediated by cue-locked theta ICPS and stimulus-locked theta ICPS, respectively.

The current study does provide evidence that reward impacts aspects of both proactive and reactive control in adolescent males in ways different than hypothesized. Reward influences cue-related neural measures, specifically increasing cue theta power and decreasing cue theta inter-channel phase synchrony (ICPS). Control strategy, proactive or reactive contexts, is associated with changes in behavior and stimulus-related theta power. Further, both reward and control strategy differentially impact behavior and neural measures of monitoring and control recruitment at different times in adolescent development. For the first hypothesis, we found that, while reward improved

performance in reactive contexts, reward hindered performance in proactive contexts, particularly in mid-adolescence. For the second hypothesis examining how reward-related neural measures impacted reward-related performance, increased reward-related cue theta power predicted increased reward-related RT interference, while increased reward-related stim theta ICPS interference predicts reduced reward-related RT interference, specifically in proactive contexts. Finally, EF also play a role in reward-related proactive and reactive control in adolescent males. At low levels of inhibitory control (IC), reward-related proactive RT interference is increased in mid-adolescence, but high levels of set-shifting (SS) are associated with decreased reward-related proactive RT interference for younger and older adolescents. In sum, reward hinders proactive performance through increases in cue theta power, and low levels of IC exacerbate deficits in reward-related proactive performance in mid-adolescence.

Influence of Reward on Behavior in Proactive and Reactive Contexts

Reward, by itself, did not modulate performance, while informative cues did improve RT interference. Importantly, reward differentially impacted performance depending on which control strategy was to be employed. In reactive contexts, reward upregulated performance by reducing RT interference, an effect that has been seen in children, adolescents, and adults (Geier et al., 2010). However, in proactive contexts, reward hindered performance by increasing RT interference, contrary to the few studies of rewarded proactive control in adolescence. For instance, Strang & Pollak (2014) found that in children, adolescents, and adults reward blocks decreased RT compared to neutral blocks in an AXCPT task and activation in fronto-parietal regions were sustained in the reward blocks compared to neutral blocks. However, they did not directly test if reward

creates a behavioral shift from reactivity to proactivity using commonly employed AXCPT metrics, like PBSI or d' context. In another study, both adolescents and adults showed sustained behavioral improvements in a reward block compared to a baseline block, suggesting a sustained task set and proactive control (Magis-Weinberg, Custers, & Dumontheil, 2019). However, these studies define proactive control as sustained improved performance or brain activation over entire reward blocks rather than employing informative cues that allowed for preparation for a response on each trial. One novel aspect of the current study is that it specifically investigated transient effects of reward on the ability to prepare on trials with an *informative cue* that allowed for proactive control, rather than sustained effects. Reward actually hinders proactive preparation in male adolescents when preparing a response based on an informative cue. Some previous work provides evidence that adolescents are more hesitant to respond and need more evidence on a random-dot motion discrimination task when high rewards are possible, a finding more consistent with the findings here and contrary to the notion that rewards always enhance performance in adolescents (Teslovich et al., 2014).

Moreover, this deficit seen for rewarded and proactive trials is further modulated by adolescent development, specifically puberty. In proactive contexts, RT interference peaked in mid-puberty for rewarded trials. This study is the first to provide evidence for a link between puberty and rewarded proactive vs reactive control in behavioral measures. Other studies have not shown a link between puberty and behavioral changes based on reward cues, even when using multiple puberty measures (Forbes et al., 2010; Ladouceur et al., 2019). However, those studies focused on paradigms that tap reactive control, not proactive control after informative cues. Pubertal status may specifically influence

reward's impact on proactive control, an ability that is still developing throughout adolescence, unlike reactive control, which is generally online earlier in pre-adolescence.

In sum, reward-related proactive control and reward-related reactive control follow different developmental trajectories (Fig 23). While reward upregulates reactive control throughout development, reward hinders proactive control specifically during mid-adolescence or mid-puberty. Before and after puberty, reward enhances proactive control with particular upregulation of proactive control in adulthood.

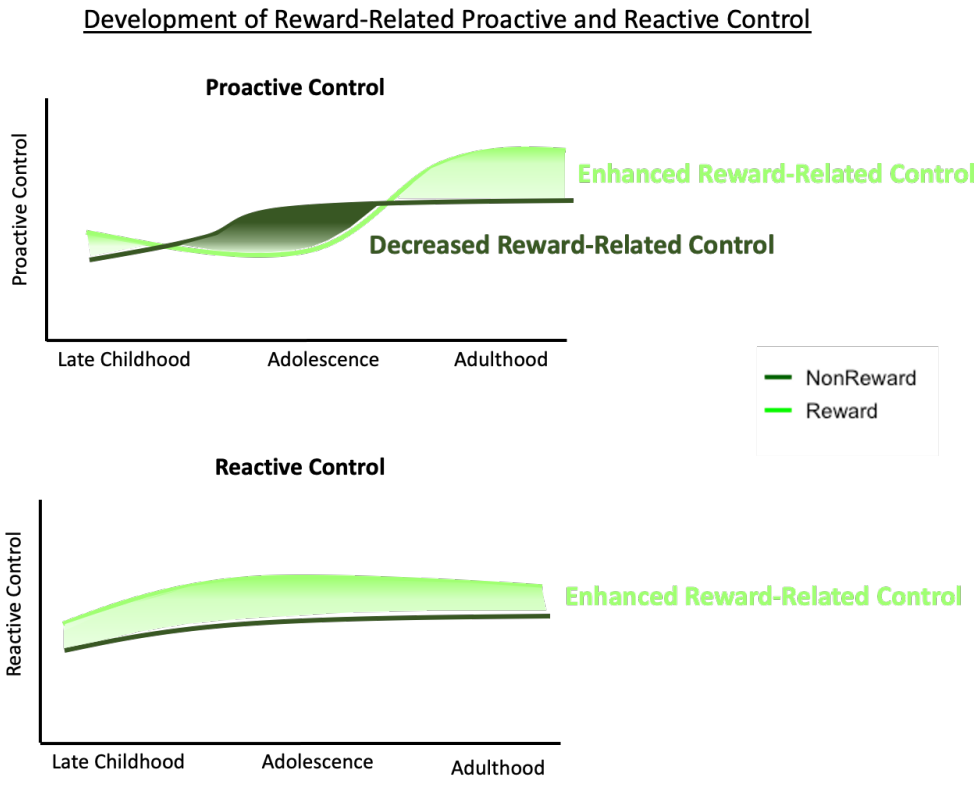


Figure 23. Conceptualization of reward-related proactive and reactive control throughout development.

Influence of Reward on Neural Measures of Proactive and Reactive Control

In addition to behavioral measures, neural indices of monitoring and control recruitment can reveal nuance about the neural processes that are impacted by reward and control strategy in adolescence. Specifically, does reward alter the salience of the cue

and, thus, the brain's monitoring of that cue? Or, does reward influence the ability to recruit frontal areas to employ top-down control in preparation for a stimulus? Does reward's impact on neural measures of proactive and reactive control change throughout adolescent development?

To answer these questions, theta dynamics were investigated. The theta frequency (4-8 Hz) is thought of as an organizing frequency for performance monitoring and cognitive control (Cavanagh & Frank, 2014; Cavanagh et al., 2012). Theta power is a well-established measure of monitoring, while theta inter-channel phase synchrony (ICPS) is a common metric that taps control recruitment (Cavanagh & Frank, 2014; Cooper et al., 2015; van Driel et al., 2012; van Driel, Swart, Egner, Ridderinkhof, & Cohen, 2015). To understand the entire picture of proactive control, preparation for a stimulus after an informative cue, and reactive control, reflexive responding when a stimulus appears in the absence of such information, both cue-locked theta power and ICPS and stim-locked theta power and ICPS were examined.

Cue Theta Power: First, to assess monitoring of preparatory cues during proactive control, cue-locked theta power was quantified. Cue theta power was increased for reward cues, consistent with previous findings that show increased theta power for preparatory reward cues in adults (Doñamayor, Schoenfeld, & Münte, 2012; Gruber, Watrous, Ekstrom, Ranganath, & Otten, 2013) and reward feedback in both adults and adolescents (Bernat, Nelson, & Baskin-Sommers, 2015b; Bowers et al., 2018). However, the effects of reward on cue theta power were also modulated by both control strategy and development. In reactive contexts, cue theta power seemed to decrease with age or puberty, regardless of reward, a trend consistent with the few studies of task-related theta

power throughout adolescence (Bowers et al., 2018; Crowley et al., 2014). However, in proactive contexts, cue theta power decreased with increasing age or puberty in nonreward but did not change with age or puberty in reward. Though, these directional changes should be interpreted with caution as follow-up tests were not significant. Here, puberty did not have a specific effect on cue theta power because puberty effects mirrored age effects and the puberty effects did not survive when controlling for age. Little work has addressed the development of cue-locked theta power; however, one study found larger cue-P3b amplitudes in younger children, but more errors on a cued continuous performance task, suggesting that younger children (e.g., 9-11 years old) show responses to informative cues, but are less able to use these cues to override prepotent responses. The current study is the first to show that theta power is at a sustained level throughout development for rewarded proactive control, possibly contributing to the performance deficits seen in rewarded proactive conditions.

Cue Theta ICPS: In order to assess control recruitment after preparatory cues during proactive control, medial-lateral cue theta ICPS was measured. Similar to cue theta power, reward impacted cue theta ICPS. However, unlike the increases seen in cue theta power after reward cues, reward cues were associated with decreases in cue theta ICPS, suggesting that reward hinders control recruitment in preparation for the stimulus. This finding is at odds with some prior work in fMRI that suggests that reward cues increase striatal-PFC connectivity in adolescence (Ladouceur et al., 2019); however, this discrepancy may just be a result of the fact that theta ICPS does not perfectly correspond to striatal-PFC connectivity. Theta ICPS more closely maps onto connectivity within prefrontal areas or within control-related networks. In fact, the pattern seen in this study

of increased rewarded cue theta power, but reductions in control-related networks after rewarding cues closely resemble patterns observed in substance use disorder (Volkow et al., 2010). Both adolescence and substance use are marked by increased reward sensitivity, but decreased cognitive control, which impacts behavior and increases the likelihood that those individuals may engage in risky behaviors or make high risk decisions.

Further, reward's impact on cue theta ICPS differed throughout adolescence. While the reward conditions showed little change throughout adolescence, in the nonreward condition, cue theta ICPS shows a slight peak in mid-adolescence. Increasing connectivity throughout adolescence without any reward is consistent with multiple studies (Hwang, Velanova, & Luna, 2010; Vink et al., 2014b). The lack of change in reward-contingent connectivity may be related to the low stakes associated with this paradigm's reward as previous studies have found selective increases with age in corticostriatal connectivity for only high stakes conditions (Insel et al., 2017).

Stim Theta Power: Stimulus-locked activity was also examined in order to understand the full picture of proactive control after cues and reactive control after the stimulus. First, stimulus monitoring was measured via stimulus-locked theta power interference, or the flanker effect. Control strategy, not reward, influenced stim theta power such that there was reduced stim theta power interference after cues that allowed for preparation. This finding is consistent with some prior work that suggests that contextual cues reduce interference (Wendt & Kiesel, 2011). However, it contradicts some work in adults (van Driel et al., 2015) that found that cued conflict increased both behavioral interference scores and stimulus-related theta interference. Further, proactive

vs reactive stim theta power differed in their developmental trajectories. With increasing pubertal scores, proactive stim theta power interference did not substantially change, suggesting that pubertal development has little impact on an informative cue's ability to reduce conflict. However, with increasing pubertal development, reactive stim theta power interference unexpectedly increases. Unfortunately, because flanker effect scores are the measure of interest, it is difficult to determine whether the increases in interference are due to increases in incongruent stim theta power or decreases in congruent stim theta power. Previous work of theta suggests that theta decreases over adolescent development (Bowers et al., 2018; Crowley et al., 2014), so it is plausible that the pronounced "interference" effects in more advanced pubertal stages are due to more rapid decreases in stim theta power in congruent trials.

Stim Theta ICPS: Next, stimulus control recruitment was quantified to explore the effects of reward, control strategy, and development. Puberty was related to stim theta ICPS in opposite directions for reward and nonreward. For reward, puberty was negatively associated with stim theta IPCS interference, suggesting that increases in pubertal status are associated with reductions in control recruitment interference after presentation of a stimulus. This finding echoes previous work that rewards increase efficiency of corticostriatal connections, particularly in adolescence (Hallquist et al., 2018). However, for nonreward, puberty was not associated with stimulus control recruitment interference. (Though, it is important to note that neither of these follow-up correlations reached statistical significance and should be interpreted with caution.)

In sum, reward is associated with changes in cue-related theta dynamics, both monitoring and control recruitment. The effects of reward on cue theta power and

behavior are also modulated by control strategy and stage of adolescent development. A majority of studies interested in reward sensitivity in adolescence focus on neural activity to the anticipation or receipt of rewards after some sort of response has been made (Kray, Schmitt, Lorenz, & Ferdinand, 2018). The current study is one of the first to investigate how rewards impact neural processes associated with *preparation* for decision-making in adolescence. Few studies have utilized tasks that allow for active preparation based on an informative cue in order to make a choice in a speeded context. One study did employ a two-choice speeded task, but one could argue that the choice associated with their stimulus was easier than a flanker stimulus (i.e., press the button of the side that the cue appeared; Galvan et al., 2006). The novelty of the current paradigm allows for conclusions that time-pressured, performance-dependent rewards negatively affect behavior and neural activity when engaging proactive control after an informative cue, but facilitates responses in the absence of informative cues. Thus, rewards seem to have negative effects for preparing a speeded response in adolescence.

Brain-Behavior Relations in Rewarded Proactive and Reactive Context

Not only did the current study seek to characterize the effects of reward, control strategy, and development on behavioral and neural measures of cognitive control, but also aimed to establish how these neural measures predicted task behavior in rewarded situations compared to nonrewarded situations. As such, we predicted reward-related task behavior from the four neural measures of reward-related cognitive control. Reward-related measures were computed via difference scores of reward – nonreward activity. The hypothesis was that reward-related cue theta ICPS, or increased control recruitment for rewarded cues, would drive improved reward-related task behavior on proactive trials,

while reward-related stim theta ICPS, or increased control recruitment after rewarded stimuli, would predict better reward-related task behavior on reactive trials. However, contrary to the hypothesis, increased reward-related stim theta ICPS interference, or an increased differentiation between incongruent and congruent trials on reward compared to nonreward trials, was the best predictor of improved reward-related behavior for *proactive* trials. Rather than upregulating top-down control after informative cues and before the presentation of the stimulus, reward seems to upregulate top-down control after the presentation of the stimulus even in the presence of informative cues. Further, this relation did not change with age or puberty, suggesting that this effect is consistent throughout adolescence.

Reward-related stim theta ICPS was not the only predictor of reward-related performance in proactive contexts. Reward-related cue theta power also predicted reward-related performance, but in the opposite direction. In fact, increased reward-related cue theta power was associated with *more* reward-related task interference, reflecting worse performance. Thus, it seems that monitoring is so increased by rewarded cues that it is interfering with subsequent performance. Similarly, this effect does not change based on age or puberty, indicating that all ages throughout adolescence are similarly negatively affected by rewarded cues. The rewarded cues may be so arousing to the participant that they become distracted and cognitive resources are pulled away from cognitive control processes, resulting in worse performance. We did see that reward negatively impacts control recruitment as well, as cue theta ICPS was reduced after reward, even though this was not a direct predictor of performance. Another possibility is that participants did not have enough time to accurately process both things that the cue

indicated on informative cues specifically: reward/nonreward and congruent/incongruent, which resulted in poor performance. Though the cue was presented for 1600 – 2000ms, a time range that should allow adequate preparatory time after informative cues (Wendt & Kiesel, 2011), adding another mapping, of reward vs nonreward, may have overwhelmed the cognitive resources of adolescents who have underdeveloped cognitive control systems.

The Role of Executive Functions in Support of Reward-Related Proactive and Reactive Control

Executive functions (EF) can be thought of as building blocks that make up cognitive control abilities; thus, the current study also sought to understand how EF abilities influence reward-related performance in proactive and reactive contexts. The three main components of EF: inhibitory control (IC), set-shifting (SS), and working memory (WM), were measured via a standardized cognition battery, the NIH Toolbox. Inhibitory control and set shifting were associated with reward-related *proactive* performance, but in different ways. At low levels of inhibitory control, or the ability to inhibit dominant responses to stimuli, reward-related proactive RT interference was increased in mid-adolescence. IC deficits could play a role in an inability to dampen the salience of the reward cue, disrupting subsequent decision-making. Individuals with low levels of IC may not be able to inhibit an arousal response to reward, and this is particularly true in mid-adolescence, when reward sensitivity is at its peak. This finding is in line with research that suggests that native deficits in inhibitory control are associated with reward-seeking behaviors and drug use (Altmann, 2004; Monsell, 2003). It is important to note that both the measure of IC and the measure of reward-related RT

interference were derived from a flanker task. It is possible that this relation is born of the fact that these are similar tasks. However, they are not identical paradigms nor are the scores calculated in the same way. First, the rewarded cued flanker task has both a reward manipulation and cue manipulation, which the NIH Toolbox Flanker lacks. In this analysis, only cued trials, which are specific to the rewarded cued paradigm, are examined. Second, the scores calculated in the NIH Toolbox Flanker incorporate both accuracy and RT into a composite score without accounting for congruency of the trial, while the behavioral measurement from the rewarded cued flanker used only includes RT and is a difference score of incongruent minus congruent.

At high levels of SS, younger and older adolescents displayed decreased reward-related proactive interference, while reward-related proactive RT interference in mid-adolescence did not benefit from high levels of set-shifting. Set-shifting, or the ability to transition back and forth between tasks, increases throughout adolescence (Dalley et al., 2007; Tarter et al., 2003). Here, in younger and older adolescents, set-shifting facilitated performance when switching between rewarded/nonrewarded for only informative cues (proactive). Not only did participants have to switch between reward and nonreward on proactive trials, but proactive trials included all cued trials, so both incongruent (triangle cues) and congruent (circle cues). Thus, participants not only had to shift from reward to nonreward, but also shift between preparing to ignore distracting flankers for incongruent-cued trials or to use the flankers to facilitate a response on congruent-cued trials. It is possible that, due to heightened reward sensitivity in mid-adolescence, mid-adolescents cannot overcome the overwhelming salience of the reward cue to utilize informative cues, even with high set-shifting abilities.

Finally, in reactive contexts, for participants with increased working memory, mid-adolescence was associated with greater reward-related reactive interference. This finding was unexpected and contradicts prior work that associated greater working memory with enhanced proactive control (Troller-Renfree, Buzzell, & Fox, in press). Indeed, increased working memory capacity is generally associated with positive outcomes like higher IQ (Luciano et al., 2001) and better learning outcomes(Alloway, 2009). Perhaps, adolescents with particularly strong working memory hold the reward aspect of the question mark cue, which interferes with responding to the stimulus. However, this is largely conjecture.

Influence of Puberty Compared to Age

Interestingly, effects of puberty were largely only observed when specifically investigating how the task manipulations, reward and control strategy, are modulated over development. Puberty impacted behavior, cue-locked theta power, and stim-locked theta power and ICPS. For both cue-locked and stim-locked theta power, the effects of age and puberty essentially mirrored each other. Analyses revealed puberty-specific effects for behavior and stim-locked theta ICPS interference, both of which survived when controlling for age. However, analyses with age alone were the most predictive when investigating brain-behavior relations and the impact of EF on reward-related proactive and reactive control. Previous work has shown that pubertal stage does impact neural activity in incentivized tasks, including cue processing (Forbes et al., 2010; Ladouceur et al., 2019), but less work has examined the impact of pubertal status on how neural activity predicts task behavior. It is possible that a puberty-independent process is

affecting how neural activity and EF support proactive and reactive control under rewarded conditions throughout adolescence (Blakemore & Choudhury, 2006).

Limitations and Future Directions

The current study is not without limitations. First, the sample size is only a moderate size. Though sufficient based on a repeated measures power analysis, the study would benefit from a larger sample size in order to detect what are most likely small effects. Second, the participants in this sample have highly educated mothers and are relatively affluent, limiting the generalizability to the population at large. This study would benefit from including participants from a more diverse socioeconomic background. Individuals with lower socioeconomic status, where money and resources are scarce, may perform differently in the presence of reward. Similarly, this sample was restricted to males, so reward could influence proactive and reactive control differently in females, especially during puberty which involves sex-specific hormone changes. Finally, the neural measures chosen to be examined a priori, cue theta power and ICPS, and stim theta power and ICPS, are not exhaustive. There are other frequencies that could play a vital role in reward processing and preparatory activity. Namely, delta power has been associated with reward processing (Bernat et al., 2015; Foti & Hajcak, 2009) and may be impacted after a reward cue compared to a nonreward cue. Secondly, alpha suppression has been associated with attention (Cooper et al., 2016; Hwang, Ghuman, Manoach, Jones, & Luna, 2016) and is modulated by cues (Mazaheri et al., 2014). Indeed, alpha power suppression and alpha phase synchrony were present after the cue in the current study. Though outside the scope of the current investigation of theta dynamics, cue-

locked alpha activity could certainly influence reward-related proactive and reactive control.

Understanding reward's impact on cognitive control is an essential step in understanding some hallmark behaviors associated with adolescence. For example, adolescence is marked by an increase in reward sensitivity and reward seeking behaviors, like substance use or risky sexual behavior. Reward may differentially affect proactive or reactive control in populations inclined to engage in these risky behaviors. In addition to being a time period associated with engaging in risky behaviors, psychopathology also begins to manifest in adolescence. The interactions between reward networks and control networks are also implicated in a variety of psychopathologies, including depression (Forbes et al., 2010). Understanding the interplay between reward and control circuitry in novel contexts (e.g., proactive vs reactive) can also elucidate novel targets for intervention in clinical populations. Future research should investigate association between reward-related proactive and reactive control and risky behaviors in adolescence and explore the influence of reward on proactive and reactive control in clinical populations.

In conclusion, this study is the first to establish that reward hinders the ability to proactively prepare after an informative cue in adolescent males, particularly around mid-puberty. Enhanced proactive cue theta power in reward compared to nonrewards plays a role in reward-related performance deficits throughout adolescence. These reward-related performance deficits after informative cues in mid-adolescence are exacerbated by low levels of inhibitory control.

Appendices

Appendix A. Calculating Reward-Related Activity via Difference Scores of Flanker Effect and Interpreting Results

All task performance and stim-locked theta measures in the above analyses utilize the flanker effect as a metric of interest. The flanker effect is, itself, a difference score. Analyzing differences in the rewarded trials vs nonrewarded trials involves creating another difference score of the flanker effect in the rewarded trials minus the flanker effect in the nonrewarded trials. The interpretation of this double difference score can become confusing. This appendix illustrates the calculation of the double difference score and how values should be interpreted. Note that this does not apply to cue-locked theta activity because congruency was not a factor for cue-locked theta analyses.

Accuracy (Percent Errors)

Below is an illustration of calculating the difference score between reward flanker effect and nonrewarded flanker effect, which was a measure of interest in the brain-behavior analyses in Aim 1 and in the path analyses in Aim 2. This calculation was done separately for reactive and proactive conditions. As a reminder, when calculating the flanker effect for accuracy [incongruent %Errors – congruent %Errors], a more positive number reflects a larger difference in percent errors in the two conditions, thus implying more interference. The flanker effect was calculated for both the rewarded trials and the nonrewarded trials, then was subsequently subtracted [Reward FE – NonReward FE]. If the resulting number was positive, that indicated that there is more interference in the

reward condition. However, is the resulting number was negative, this indicated that there is less interference in the reward condition.

Reward			**NonReward**			**Rew FE – NonRew FE**
Inc	Con	Flanker Effect	Inc	Con	Flanker Effect	
10	5	5	11	3	8	-3
11	3	8	10	5	5	3

Reaction Time

Below is an illustration of calculating the difference score between reward flanker effect and nonrewarded flanker effect for RT. This calculation was done separately for reactive and proactive conditions. As a reminder, when calculating the flanker effect for RT [incongruent RT – congruent RT], a more positive number reflects a larger difference in RT in the two conditions, thus implying more interference. The flanker effect was calculated for both the rewarded trials, then the nonrewarded trials and subsequently subtracted [Reward FE – NonReward FE]. For RT, if the resulting number was negative, that indicated that there is less interference in the reward condition. However, if the resulting number was positive, this indicated that there is more interference in the reward condition.

Reward			**NonReward**			**Rew FE – NonRew FE**
Inc	Con	Flanker Effect	Inc	Con	Flanker Effect	
500	400	100	650	450	200	-100
650	450	200	500	400	100	100

Stimulus-Locked Theta Power and ICPS

Now, here is an illustration of calculating the difference score between reward flanker effect and nonrewarded flanker effect for stim-locked theta activity. This description holds true for both theta power and ICPS. This calculation was also done separately for reactive and proactive conditions. For the flanker effect for stim-locked

theta [incongruent theta – congruent theta], a more positive number reflects a larger difference in theta in the two conditions, thus implying more interference. The flanker effect was calculated for both the rewarded trials, then the nonrewarded trials and subsequently subtracted [Reward FE – NonReward FE]. For stim-locked theta, if the resulting number was negative, that indicated that there is less interference in the reward condition. However, if the resulting number was positive, this indicated that there is more interference in the reward condition.

Reward			**NonReward**			**Rew FE – NonRew FE**
Inc	Con	Flanker Effect	Inc	Con	Flanker Effect	
2	1	1	3	1	2	-1
3	1	2	2	1	1	1

In conclusion, for all reward-related measures that are subtractions of flanker effects, accuracy (percent errors), RT, stim-locked theta power, and stim locked theta ICPS, a positive value means more reward-related interference, but a negative value means less reward-related interference.

Appendix B: Tables of results for Task Effort Survey MLMs

Comparing Blue Cues to Orange Cues within Baseline Block:

Table B1. Question 1: "How hard did you try to be correct after [insert color] shapes?"

Predictor	B	SE	p
Intercept	7.779	0.439	< 0.001**
Color	- 0.004	0.098	0.97
Age	0.107	0.118	0.37
Age^2	- 0.049	0.052	0.35
Color x Age	- 0.005	0.026	0.85
Color x Age^2	0.000	0.012	0.97

† p < .10, * *p* < .05, ** *p* < .01

Table B2. Question 1: "How hard did you try to be correct after [insert color] shapes?"

Predictor	B	SE	p
Intercept	8.179	0.395	< 0.001**
Color	- 0.003	0.092	0.97
Puberty	0.415	0.314	0.19
$Puberty^2$	- 0.879	0.352	0.015*
Color x Puberty	0.032	0.073	0.67
Color x $Puberty^2$	- 0.005	0.082	0.95

† p < .10, * *p* < .05, ** *p* < .01

Table B3. Question 2: "How hard did you try to be fast after [insert color] shapes?"

Predictor	B	SE	p
Intercept	7.892	0.414	< 0.001**
Color	- 0.037	0.010	0.71
Age	0.131	0.111	0.24
Age^2	- 0.082	0.049	0.10†
Color x Age	- 0.008	0.027	0.77
Color x Age^2	0.001	0.012	0.93

† p < .10, * *p* < .05, ** *p* < .01

Table B4. Question 2: "How hard did you try to be fast after [insert color] shapes?"

Predictor	B	SE	p
Intercept	8.076	0.379	< 0.001**
Color	0.001	0.093	0.99
Puberty	0.406	0.302	0.18
$Puberty^2$	- 0.870	0.338	0.012*
Color x Puberty	0.040	0.075	0.59
Color x $Puberty^2$	- 0.038	0.084	0.65

† p < .10, * *p* < .05, ** *p* < .01

Comparing Baseline Block to Reward Block:

Table B5. Question 1: "How motivated were you to do well in this round?"

Predictor	B	SE	p
Intercept	8.337	0.211	< 0.001**
Block	0.833	0.130	< 0.001**
Age	0.149	0.057	0.011*
Age^2	- 0.015	0.025	0.55
Block x Age	- 0.130	0.035	< 0.001**
Block x Age^2	0.002	0.015	0.90

† p < .10, * *p* < .05, ** *p* < .01

Table B6. Question 1: "How motivated were you to do well in this round?"

Predictor	B	SE	p
Intercept	8.495	0.199	< 0.001**
Block	0.829	0.123	< 0.001**
Puberty	0.347	0.159	0.03*
$Puberty^2$	- 0.307	0.177	0.09†
Block x Puberty	- 0.317	0.100	0.002**
Block x $Puberty^2$	0.012	0.112	0.92

† p < .10, * *p* < .05, ** *p* < .01

Table B7. Question 2: "How motivated were you to pay attention in this round?"

Predictor	B	SE	p
Intercept	8.402	0.283	< 0.001**
Block	0.729	0.156	< 0.001**
Age	- 0.050	0.076	0.51
Age^2	- 0.014	0.033	0.67
Block x Age	0.007	0.042	0.86
Block x Age^2	- 0.020	0.018	0.28

† p < .10, * *p* < .05, ** *p* < .01

Table B8. Question 2: "How motivated were you to pay attention in this round?"

Predictor	B	SE	p
Intercept	8.613	0.263	< 0.001**
Block	0.581	0.150	< 0.001**
Puberty	- 0.140	0.209	0.50
$Puberty^2$	- 0.382	0.234	0.11
Block x Puberty	- 0.037	0.119	0.76
Block x $Puberty^2$	0.028	0.133	0.84

† p < .10, * *p* < .05, ** *p* < .01

Comparing Rewarded Cues to NonRewarded Cues within Reward Block:

Table B9. Question 1: "How hard did you try to be correct after [insert color] shapes?"

Predictor	B	SE	p
Intercept	8.244	0.199	< 0.001**
Reward	1.357	0.173	< 0.001**
Age	- 0.006	0.053	0.91
Age^2	- 0.041	0.023	0.08†
Reward x Age	- 0.094	0.047	0.048*
Reward x Age^2	- 0.007	0.020	0.74

† p < .10, * *p* < .05, ** *p* < .01

Table B10. Question 1: "How hard did you try to be correct after [insert color] shapes?"

Predictor	B	SE	p
Intercept	8.252	0.186	< 0.001**
Reward	1.258	0.162	< 0.001**
Puberty	0.064	0.148	0.67
$Puberty^2$	- 0.334	0.166	0.048*
Reward x Puberty	- 0.297	0.129	0.024*
Reward x $Puberty^2$	0.064	0.144	0.66

† p < .10, * *p* < .05, ** *p* < .01

Table B11. Question 2: "How hard did you try to be fast after [insert color] shapes?"

Predictor	B	SE	p
Intercept	8.070	0.244	< 0.001**
Reward	1.220	0.182	< 0.001**
Age	0.064	0.066	0.34
Age^2	- 0.033	0.029	0.26
Reward x Age	0.010	0.049	0.84
Reward x Age^2	- 0.002	0.022	0.92

† p < .10, * *p* < .05, ** *p* < .01

Table B12. Question 2: "How hard did you try to be fast after [insert color] shapes?"

Predictor	B	SE	p
Intercept	8.281	0.222	< 0.001**
Reward	1.226	0.172	< 0.001**
Puberty	0.145	0.177	0.41
$Puberty^2$	- 0.518	0.198	0.011*
Reward x Puberty	- 0.006	0.137	0.97
Reward x $Puberty^2$	- 0.025	0.154	0.87

† p < .10, * *p* < .05, ** *p* < .01

Appendix C: Tables of results for behavioral and EEG MLMs

Table C1.
Accuracy Flanker Effect - Age

Predictor	B	SE	p
Intercept	0.290	0.024	< 0.001 **
Reward	0.004	0.008	0.61
Control Strategy	0.001	0.008	0.88
Age	- 0.008	0.006	0.23
Age^2	- 0.001	0.003	0.83
Reward x Age	- 0.002	0.002	0.27
Control Strategy x Age	- 0.001	0.002	0.65
Reward x Age^2	0.000	0.001	0.73
Control Strategy x Age^2	0.000	0.001	0.73
Reward x Control Strategy	0.002	0.008	0.84
Reward x Control Strategy x Age	0.002	0.002	0.26
Reward x Control Strategy x Age^2	0.000	0.001	0.82

†, p < .10, * $p < .05$, ** $p < .001$

Table C2.
Accuracy Flanker Effect – Puberty

Predictor	B	SE	p
Intercept	0.273	0.023	< 0.001**
Reward	0.000	0.007	0.97
Control Strategy	- 0.006	0.007	0.42
Puberty	- 0.002	0.018	0.91
$Puberty^2$	0.015	0.020	0.47
Reward x Puberty	- 0.010	0.006	0.09†
Control Strategy x Puberty	- 0.003	0.006	0.66
Reward x $Puberty^2$	0.003	0.007	0.67
Control Strategy x $Puberty^2$	0.006	0.007	0.34
Reward x Control Strategy	0.000	0.007	0.90
Reward x Control Strategy x Puberty	0.000	0.006	0.99
Reward x Control Strategy x $Puberty^2$	0.000	0.007	0.91

†, p < .10, * $p < .05$, ** $p < .001$

Table C3.
RT Flanker Effect – Age

Predictor	B	SE	p
Intercept	78.330	6.663	< 0.001**
Reward	- 3.056	1.869	0.10
Control Strategy	- 6.583	1.869	< 0.001**
Age	- 4.376	1.816	0.019*
Age^2	1.674	0.790	0.038*
Reward x Age	0.669	0.511	0.19
Control Strategy x Age	- 0.056	0.511	0.91
Reward x Age^2	- 0.597	0.224	0.008**
Control Strategy x Age^2	0.231	0.224	0.30
Reward x Control Strategy	14.079	1.869	< 0.001**
Reward x Control Strategy x Age	- 1.124	0.511	0.029*
Reward x Control Strategy x Age^2	- 0.203	0.224	0.37

† p < .10, * $p < .05$, ** $p < .01$

Table C4.
RT Flanker Effect – Puberty

Predictor	B	SE	p
Intercept	91.735	6.786	< 0.001**
Reward	- 5.493	1.782	0.002**
Control Strategy	- 6.397	1.782	< 0.001**
Puberty	- 9.389	5.399	0.09†
$Puberty^2$	- 3.556	6.042	0.55
Reward x Puberty	3.058	1.420	0.033*
Control Strategy x Puberty	0.790	1.420	0.57
Reward x $Puberty^2$	- 1.578	1.585	0.32
Control Strategy x $Puberty^2$	1.519	1.585	0.34
Reward x Control Strategy	15.344	1.782	< 0.001**
Reward x Control Strategy x Puberty	- 2.332	1.420	0.010†
Reward x Control Strategy x $Puberty^2$	- 3.249	1.585	0.042*

† p < .10, * $p < .05$, ** $p < .01$

Table C5.
Cue-Locked Theta Power - Age

Predictor	B	SE	p
Intercept	1.144	0.136	< 0.001**
Reward	0.100	0.043	0.02*
Control Strategy	0.058	0.043	0.18
Age	- 0.020	0.037	0.59
Age^2	- 0.004	0.016	0.81
Reward x Age	0.008	0.012	0.49
Control Strategy x Age	- 0.007	0.012	0.57
Reward x Age^2	0.003	0.005	0.54
Control Strategy x Age^2	- 0.004	0.005	0.39
Reward x Control Strategy	0.036	0.043	0.41
Reward x Control Strategy x Age	0.026	0.012	0.03*
Reward x Control Strategy x Age^2	- 0.003	0.005	0.55

† p < .10, * *p* < .05, ** *p* < .01

Table C6.
Cue-Locked Theta Power - Puberty

Predictor	B	SE	p
Intercept	1.181	0.128	< 0.001**
Reward	0.199	0.040	< 0.001**
Control Strategy	0.015	0.040	0.70
Puberty	- 0.091	0.102	0.38
$Puberty^2$	- 0.077	0.114	0.50
Reward x Puberty	- 0.020	0.032	0.54
Control Strategy x Puberty	- 0.001	0.032	0.98
Reward x $Puberty^2$	- 0.099	0.036	0.005**
Control Strategy x $Puberty^2$	0.020	0.036	0.58
Reward x Control Strategy	0.056	0.040	0.16
Reward x Control Strategy x Puberty	0.073	0.032	0.02*
Reward x Control Strategy x $Puberty^2$	- 0.050	0.036	0.17

† p < .10, * *p* < .05, ** *p* < .01

Table C7.
Cue-Locked Theta ICPS - Age

Predictor	B	SE	p
Intercept	0.014	0.003	< 0.001**
Reward	- 0.006	0.002	0.005**
Control Strategy	0.003	0.002	0.13
Age	0.001	0.001	0.12
Age^2	0.000	0.000	0.50
Reward x Age	0.000	0.000	0.28
Control Strategy x Age	0.000	0.000	0.54
Reward x Age^2	0.000	0.000	0.03*
Control Strategy x Age^2	0.000	0.000	0.92
Reward x Control Strategy	- 0.001	0.002	0.54
Reward x Control Strategy x Age	0.000	0.000	0.67
Reward x Control Strategy x Age^2	0.000	0.000	0.26

† p < .10, * *p* < .05, ** *p* < .01

Table C8.
Cue-Locked Theta ICPS - Puberty

Predictor	B	SE	p
Intercept	0.012	0.004	< 0.001**
Reward	- 0.004	0.002	0.07†
Control Strategy	0.002	0.002	0.38
Puberty	0.002	0.003	0.59
$Puberty^2$	- 0.001	0.003	0.60
Reward x Puberty	0.000	0.002	0.75
Control Strategy x Puberty	0.000	0.002	0.99
Reward x $Puberty^2$	0.000	0.002	0.48
Control Strategy x $Puberty^2$	0.002	0.002	0.25
Reward x Control Strategy	0.003	0.002	0.13
Reward x Control Strategy x Puberty	0.000	0.002	0.61
Reward x Control Strategy x $Puberty^2$	- 0.003	0.002	0.07†

†, p < .10, * *p* < .05, ** *p* < .001

Table C9.
Stim-Locked Theta Power Flanker Effect - Age

Predictor	B	SE	p
Intercept	1.614	0.138	< 0.001**
Reward	0.013	0.073	0.85
Control Strategy	- 0.539	0.073	< 0.001**
Age	0.087	0.038	0.02*
Age^2	0.003	0.016	0.83
Reward x Age	- 0.011	0.020	0.58
Control Strategy x Age	- 0.081	0.020	< 0.001**
Reward x Age^2	- 0.002	0.009	0.81
Control Strategy x Age^2	0.001	0.009	0.93
Reward x Control Strategy	- 0.080	0.073	0.27
Reward x Control Strategy x Age	0.002	0.020	0.93
Reward x Control Strategy x Age^2	0.009	0.009	0.33

† p < .10, * *p* < .05, ** *p* < .001

Table C10.
Stim-Locked Theta Power Flanker Effect – Puberty

Predictor	B	SE	p
Intercept	1.671	0.135	< 0.001**
Reward	0.011	0.068	0.87
Control Strategy	- 0.540	0.068	< 0.001**
Puberty	0.100	0.108	0.36
$Puberty^2$	- 0.043	0.120	0.72
Reward x Puberty	- 0.046	0.055	0.40
Control Strategy x Puberty	- 0.238	0.055	< 0.001**
Reward x $Puberty^2$	- 0.014	0.061	0.81
Control Strategy x $Puberty^2$	0.008	0.061	0.89
Reward x Control Strategy	- 0.004	0.068	0.96
Reward x Control Strategy x Puberty	0.003	0.055	0.96
Reward x Control Strategy x $Puberty^2$	- 0.030	0.061	0.62

† p < .10, * *p* < .05, ** *p* < .001

Table C11.
Stim-Locked Theta ICPS Flanker Effect - Age

Predictor	B	SE	p
Intercept	0.000	0.006	0.99
Reward	0.005	0.004	0.18
Control Strategy	- 0.002	0.004	0.58
Age	0.001	0.002	0.45
Age^2	0.000	0.001	0.82
Reward x Age	0.000	0.001	0.47
Control Strategy x Age	0.000	0.001	0.74
Reward x Age^2	0.000	0.000	0.88
Control Strategy x Age^2	0.000	0.000	0.40
Reward x Control Strategy	- 0.005	0.000	0.16
Reward x Control Strategy x Age	- 0.019	0.004	0.38
Reward x Control Strategy x Age^2	0.000	0.000	0.19

† p < .10, * *p* < .05, ** *p* < .001

Table C12.
Stim-Locked Theta ICPS Flanker Effect - Puberty

Predictor	B	SE	p
Intercept	- 0.003	0.006	0.53
Reward	0.006	0.004	0.17
Control Strategy	- 0.003	0.004	0.35
Puberty	0.001	0.004	0.86
$Puberty^2$	0.003	0.005	0.55
Reward x Puberty	- 0.006	0.003	0.04*
Control Strategy x Puberty	0.000	0.003	0.89
Reward x $Puberty^2$	0.000	0.003	0.99
Control Strategy x $Puberty^2$	- 0.001	0.003	0.65
Reward x Control Strategy	0.000	0.004	0.88
Reward x Control Strategy x Puberty	- 0.003	0.003	0.31
Reward x Control Strategy x $Puberty^2$	- 0.003	0.003	0.40

† p < .10, * *p* < .05, ** *p* < .001

Appendix D. Brain-Behavior relations in individual regressions.

In the main text, we investigated brain-behavior relations by adding all four EEG measures as predictors in the second block of a hierarchical regression. In this series of tables, we report regression results when individually testing the relation between each neural measure and behavior.

Table D1
Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference

Variable	B	SE (B)	X
Intercept	11.74*	4.27	0.00
Rew-Rel Pro Cue Power	6.68	6.21	0.14
F stat			1.16
R^2			1.95

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table D2
Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference

Variable	B	SE (B)	X
Intercept	12.93**	4.00	0.00
Rew-Rel Pro Cue ICPS	-44.31	110.39	- 0.05
F stat			0.16
R^2			0.28

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table D3
Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference

Variable	B	SE (B)	X
Intercept	13.09**	3.93	0.00
Rew-Rel Pro Stim Power	-2.13	4.67	- 0.06
F stat			0.21
R^2			0.36

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table D4

Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference

Variable	B	SE (B)	X
Intercept	15.38**	3.63	0.00
Rew-Rel Pro Stim ICPS	-215.30**	62.46	- 0.41
F stat			11.88**
R^2			17.00

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table D5

Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference

Variable	B	SE (B)	X
Intercept	-34.88**	4.09	0.00
Rew-Rel Re Cue Power	0.77	5.63	0.02
F stat			0.02
R^2			0.03

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table D6

Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference

Variable	B	SE (B)	X
Intercept	-33.68**	4.16	0.00
Rew-Rel Re Cue ICPS	131.90	136.39	0.13
F stat			0.94
R^2			1.58

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table D7

Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference

Variable	B	SE (B)	X
Intercept	-34.86**	4.04	0.00
Rew-Rel Re Stim Power	1.18	3.85	0.04
F stat			0.09
R^2			0.16

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table D8
Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference

Variable	B	SE (B)	X
Intercept	-35.47**	4.12	0.00
Rew-Rel Re Stim ICPS	54.05	72.59	0.10
F stat			0.55
R^2			0.95

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Appendix E. Regression table for addition of third block in hierarchical regression models

Table E1
Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference with Age

Variable	**Block 3**		
	B	SE (B)	X
Intercept	18.74**	6.38	0.00
Age	- 1.97	1.84	- 0.16
Age^2	- 1.20	0.91	- 0.21
Rew-Rel Pro Cue Power	32.59**	10.94	0.68
Rew-Rel Pro Cue ICPS	- 86.18	148.40	- 0.10
Rew-Rel Pro Stim Power	- 7.16	7.88	- 0.20
Rew-Rel Pro Stim ICPS	- 425.47**	125.66	- 0.81
Rew-Rel Pro Cue Power x Age	0.09	2.67	0.01
Rew-Rel Pro Cue ICPS x Age	46.79	58.45	0.11
Rew-Rel Pro Stim Power x Age	3.43†	1.95	0.25
Rew-Rel Pro Stim ICPS x Age	25.96	30.16	0.14
Rew-Rel Pro Cue Power x Age^2	- 3.40*	1.63	- 0.54
Rew-Rel Pro Cue ICPS x Age^2	20.10	29.35	0.14
Rew-Rel Pro Stim Power x Age^2	0.75	1.16	0.18
Rew-Rel Pro Stim ICPS x Age^2	24.72	17.05	0.44
F stat			2.29*
R^2			41.60
ΔR^2 *F stat*			

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table E2

Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference with Age

Variable	**Block 3**		
	B	SE (B)	X
Intercept	- 32.46**	7.72	0.00
Age	1.63	1.86	0.13
Age^2	- 0.51	1.00	- 0.09
Rew-Rel Re Cue Power	- 1.48	9.92	- 0.03
Rew-Rel Re Cue ICPS	243.77	268.47	0.23
Rew-Rel Re Stim Power	11.72†	6.69	0.40
Rew-Rel Re Stim ICPS	26.74	129.57	0.05
Rew-Rel Re Cue Power x Age	0.18	2.83	0.01
Rew-Rel Re Cue ICPS x Age	- 30.92	73.84	- 0.07
Rew-Rel Re Stim Power x Age	- 3.14	1.97	- 0.26
Rew-Rel Re Stim ICPS x Age	33.06	35.79	0.16
Rew-Rel Re Cue Power x Age^2	0.22	1.51	0.04
Rew-Rel Re Cue ICPS x Age^2	- 10.02	40.33	- 0.08
Rew-Rel Re Stim Power x Age^2	- 1.27	0.91	- 0.33
Rew-Rel Re Stim ICPS x Age^2	- 2.09	15.73	- 0.03
F stat			0.77
R^2			19.27
ΔR^2 *F stat*			0.83

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table E3
Hierarchical Multiple Regression Predicting Reward-Related Proactive RT Interference with Puberty

Variable	**Block 3**		
	B	SE (B)	X
Intercept	18.91**	6.30	0.00
Puberty	- 8.07	5.17	- 0.24
$Pubery^2$	- 5.32	5.93	- 0.15
Rew-Rel Pro Cue Power	12.46	8.63	0.26
Rew-Rel Pro Cue ICPS	- 154.41	147.96	- 0.18
Rew-Rel Pro Stim Power	- 7.68	7.21	- 0.22
Rew-Rel Pro Stim ICPS	- 260.28*	105.34	- 0.50
Rew-Rel Pro Cue Power x Puberty	- 5.30	6.80	- 0.11
Rew-Rel Pro Cue ICPS x Puberty	- 215.68	165.18	- 0.20
Rew-Rel Pro Stim Power x Puberty	4.77	5.24	0.15
Rew-Rel Pro Stim ICPS x Puberty	26.62	93.52	0.05
Rew-Rel Pro Cue Power x $Pubery^2$	- 4.75	7.26	- 0.13
Rew-Rel Pro Cue ICPS x $Pubery^2$	232.62	202.35	0.24
Rew-Rel Pro Stim Power x $Pubery^2$	2.69	5.65	0.11
Rew-Rel Pro Stim ICPS x $Pubery^2$	- 56.01	119.22	- 0.14
F stat			1.83†
R^2			36.29
ΔR^2 *F stat*			

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

Table E4
Hierarchical Multiple Regression Predicting Reward-Related Reactive RT Interference with Puberty

Variable	**Block 3**		
	B	SE (B)	X
Intercept	- 40.27**	7.37	0.00
Puberty	3.71	5.56	0.11
Pubery^2	3.77	6.11	0.10
Rew-Rel Re Cue Power	11.47	11.05	0.27
Rew-Rel Re Cue ICPS	- 139.61	278.61	- 0.13
Rew-Rel Re Stim Power	- 0.22	8.44	- 0.01
Rew-Rel Re Stim ICPS	17.96	111.04	0.03
Rew-Rel Re Cue Power x Puberty	- 3.44	9.96	- 0.07
Rew-Rel Re Cue ICPS x Puberty	- 18.02	238.10	- 0.02
Rew-Rel Re Stim Power x Puberty	- 2.43	6.54	- 0.09
Rew-Rel Re Stim ICPS x Puberty	128.16	126.87	0.23
Rew-Rel Re Cue Power x Pubery^2	- 17.84	14.59	- 0.45
Rew-Rel Re Cue ICPS x Pubery^2	430.95	302.71	0.49
Rew-Rel Re Stim Power x Pubery^2	6. 41	7.80	0.31
Rew-Rel Re Stim ICPS x Pubery^2	26.10	109.82	0.06
F stat			0.71
R^2			18.07
ΔR^2 *F stat*			0.64

† p < .10, *p<.05, **p<.01. R^2 is the percent of total variance explained.

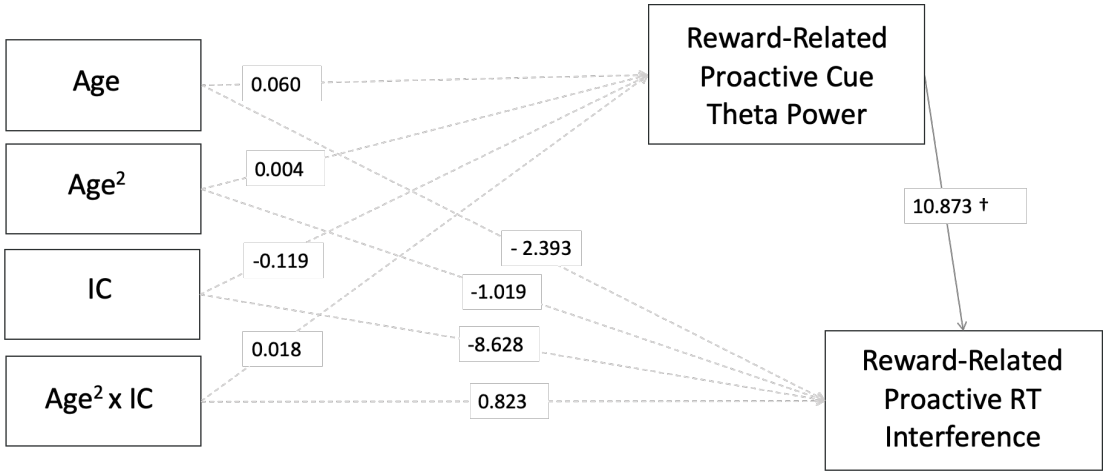
Appendix F. Null results predicting reward-related RT interference with puberty

Table F1
Regression paths from the path model of puberty and EF predicting task reaction time (RT).

	Reward-Related Proactive RT Interference			**Reward-Related Reactive RT Inteference**		
Predictors	Estimate	95% CI	z	Estimate	95% CI	z
Puberty	2.841	[-2.217,4.743]	0.697	8.670	[2.111,10.717]	1.939
Puberty^2	- 5.594	[-2.660,0.341]	- 1.268	0.993	[-2.415,0.270]	0.184
WM	0.462	[-6.208,6.964]	0.210	0.383	[1.678,9.379]	0.151
IC	- 6.259	[-33.110,3.391]	- 0.693	11.266	[-9.392,29.749]	1.184
SS	- 4.772	[-10.071,7.958]	- 1.154	- 8.187*	[-15.626,3.329]	- 1.272
WM x Puberty	0.121	[-0.605,0.747]	0.069	- 3.382	[-2.380,-0.780]	- 3.872
IC x Puberty	- 2.764	[-6.997,0.895]	- 0.302	4.308	[-4.381,7.854]	0.556
SS x Puberty	6.224	[-0.210,6.438]	1.056	7.456	[-0.837,7.854]	1.575
WM x Puberty ^{2}	1.542	[-0.400,0.850]	0.438	1.235	[-1.178,-0.262]	- 3.081
IC x Puberty ^{2}	12.218	[0.793,5.142]	1.004	- 2.504	[-2.893,2.290]	- 0.226
SS x Puberty ^{2}	- 8.084	[-3.881,-0.811]	- 1.082	3.491	[-3.214,1.694]	- 0.607

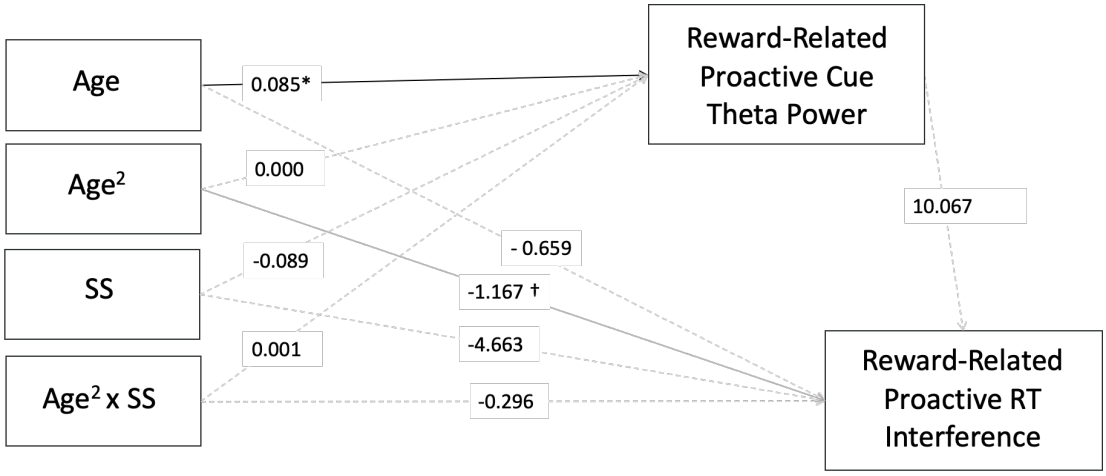
† p < .10, *p<.05, **p<.01. Estimates = unstandardized estimates. All predictors were centered. Note that the one significant result does not survive for multiple comparison correction.

Appendix G. Null results of mediation models.



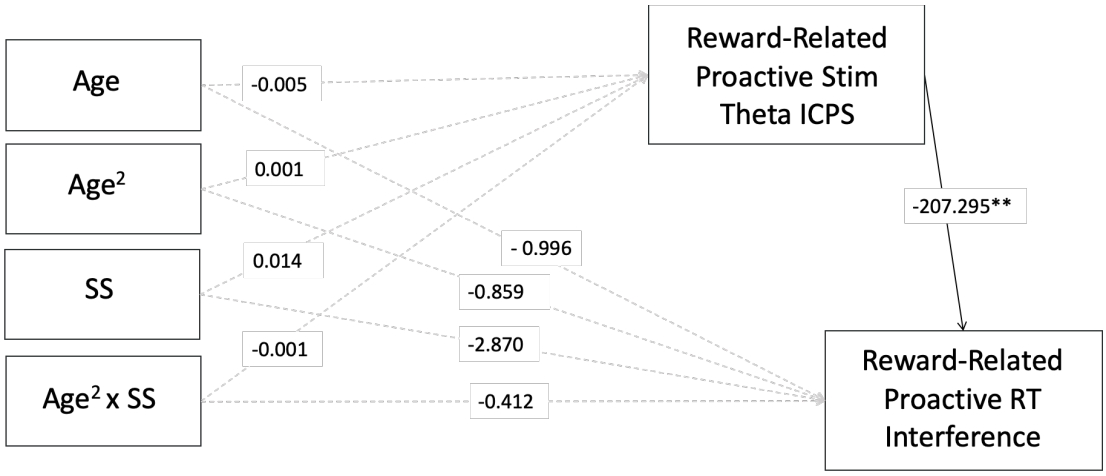
Indirect Effect of IC x age² through Cue Theta Power : b = 0.192, *z* = 0.196, *p* = 0.327.

Figure G1. Relation between IC x age^2 and reward-related proactive RT interference as mediated by reward-related proactive cue theta power.



Indirect Effect of SS x age² through Cue Theta Power : b = 0.007, *z* = 0.043, *p* = 0.965.

Figure G2. Relation between SS x age^2 and reward-related proactive RT interference as mediated by reward-related proactive cue theta power.



Indirect Effect of SS x age² through Stim Theta ICPS : b = 0.130, z = 0.607, p = 0.544.

Figure G3. Relation between SS x age^2 and reward-related proactive RT interference as mediated by reward-related proactive stim theta ICPS.

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