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An EEG Study of Children With and Without ADHD Symptoms: Between-Group Differences and Associations With Sluggish Cognitive Tempo Symptoms

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Abstract

Objective: We examined differences between those with and without ADHD symptoms on resting state electroencephalography (EEG) indices and unique relations with sluggish cognitive tempo (SCT) symptoms. **Method:** Children with ADHD symptoms (n = 21) and healthy controls (n = 20) were assessed using rating scales, a neuropsychological task measuring sustained attention and inhibitory control, and EEG activity during a resting state period. Between-group, correlational, and regression analyses were conducted. **Results:** Large differences (particularly for theta/beta ratio in frontal and frontocentral regions) were found on EEG measures between those with and without ADHD symptoms. While ADHD and SCT symptoms both related to sustained attention on a computerized task, only ADHD symptoms were related to frontal and frontocentral theta/beta ratio. **Conclusion:** Results support the conclusion that ADHD symptoms are strongly associated with theta/beta ratio in frontal and frontocentral regions. Future studies should explore unique neurophysiological correlates of SCT. *(J. of Att. Dis. XXXX; XX(X) XX-XX)*

Keywords

ADHD, EEG, neuropsychology, sluggish cognitive tempo, SCT

ADHD is one of the most commonly diagnosed childhood mental health disorders with at least 5% of children and adolescents meeting criteria for the disorder in the population (American Psychiatric Association [APA], 2013). Although ADHD is defined by its behavioral symptoms of inattention and hyperactivity/impulsivity, it has been increasingly recognized as a disorder that is associated with abnormalities in neuropsychological functioning (Willcutt et al., 2012), neurophysiological activity (Snyder & Hall, 2006), and brain structure and function as indexed by magnetic resonance imaging (MRI) and functional MRI (fMRI; Epstein et al., 2007; Shaw et al., 2013). Furthermore, the most recent version of the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; APA, 2013) moved ADHD from a section on disruptive behavior disorders (including oppositional defiant disorder and conduct disorder) to a section on neurodevelopmental disorders, lumping ADHD with disorders such as autism spectrum disorder, language disorder, and intellectual disability. Such a conceptual change reflects the increasing recognition that ADHD has strong neurodevelopmental roots.

While a large body of research has been devoted to blood flow–dependent imaging techniques (e.g., fMRI) and ADHD, a relatively smaller literature has explored neurophysiological differences using approaches such as electroencephalography (EEG). EEG offers a unique neural assessment of ADHD from blood flow–dependent measures, because EEG assesses neuronal postsynaptic electrical fields. Research using EEG patterns has found unique neural frequency differences between those with ADHD and nonclinical controls. Neurophysiological indices of ADHD frequently investigate theta band frequency (4-8 Hz) activity, beta band frequency (13-30 Hz) activity, and the ratio between these two frequencies (theta/beta ratio [TBR]). In general, children with ADHD tend to show greater frontocentral theta activity, reflecting drowsy and unfocused states, relative to controls (Breshnahan, Anderson, & Barry, 1999; Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 1998, 2001; Hobbs, Clarke, Barry, McCarthy, & Selikowitz, 2007; Loo et al., 2010; Snyder & Hall, 2006). Meta-analytic findings suggest

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that the effect size (Cohen's *d*) for this difference is 1.31 (Snyder & Hall, 2006). In addition to theta differences, some studies have shown differences on beta activity, which is thought to reflect enhanced cortical arousal and mental activity. Evidence for beta differences has been more variable, with some studies finding decreased beta (i.e., indicative of less cortical activity) in frontal and central regions (Chabot & Serfontein, 1996; Clarke et al., 2001), while other studies not finding this effect (Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996; Loo et al., 2010). Snyder and Hall (2006) reported an average effect size (Cohen's *d*) of −0.51, suggesting a smaller difference than theta activity.

In addition to these individual frequency bands, the ratio between theta and beta frequencies appears to be especially important in children with ADHD. Children with ADHD have a greater TBR relative to nonclinical controls (Breshnahan & Barry, 2002; Clarke et al., 2001; Monastra, Lubar, & Linden, 2001; Snyder & Hall, 2006). This ratio reflects the relative pattern of the theta oscillations associated with drowsy and unfocused states to beta oscillations associated with cortical excitation. Greater TBR is thought to reflect cortical underarousal and less focused neural states. Meta-analytic research supports TBR as a robust difference between individuals with ADHD and controls (Snyder & Hall, 2006). For example, Snyder and Hall (2006) found a mean effect size (Cohen's *d*) of 3.08 when comparing those with ADHD with controls. More recent attempts at meta-analysis have shown declines in group differences over years of study with more recent studies showing smaller magnitude differences between those with ADHD and controls (Arns, Conners, & Kraemer, 2013).

Finally, studies have also examined the differential relations of inattention and hyperactivity/impulsivity symptoms with TBR. In one of the largest studies to date, Loo et al. (2010) found that inattention symptoms were associated with higher TBR, but hyperactivity/impulsivity symptoms were not, and higher TBR was associated with other indicators of attentional functioning such as increased omission errors, a slower reaction time, and greater reaction time variability on a go/no-go task. Overall, Loo et al. noted that higher TBR appears to reflect several aspects of inattention.

Sluggish Cognitive Tempo (SCT)

In addition to the core ADHD symptom domains of inattention and hyperactivity/impulsivity, recent research emphasizes SCT as a unique symptom domain related to but distinct from core ADHD symptoms (Becker et al., 2016). Research on SCT dates back to the late 1980s when researchers examined differences in SCT symptoms among children with the former diagnoses of attention deficit with hyperactivity and attention deficit without hyperactivity (Lahey, Schaughency, Hynd, Carlson, & Nieves, 1987). More recently, interest in this symptom domain has been reignited (see Becker, Marshall, & McBurnett, 2014), as evidenced by the development of a number of specific rating scales that focus on the SCT symptom domain and studies examining unique SCT correlates such as neuropsychological functioning (Becker et al., 2017; Becker et al., 2016).

In relation to neuropsychological functioning, findings have been mixed regarding relationships between SCT and neuropsychological functioning. Some studies have found significant unique relationships between SCT symptoms and neuropsychological functioning domains such as sustained attention, working memory, and meta-cognitive deficits (Barkley, 2012; Becker & Langberg, 2014; Skirbekk, Hansen, Oerbeck, & Kristensen, 2011; Wåhlstedt & Bohlin, 2010; Willcutt et al., 2014) with the most consistent research support for sustained attention. In contrast, other studies have found little to no evidence or mixed evidence depending on whether rating scales or laboratory tasks are used to measure neuropsychological deficits (Bauermeister, Barkley, Bauermeister, Martinez, & McBurnett, 2012; Jarrett, Rapport, Rondon, & Becker, 2017). Despite the recent growing interest in SCT research, past work has yet to examine neurophysiological correlates of SCT using EEG.

To extend research on the relations among core ADHD symptoms, SCT, neuropsychological functioning, and neurophysiology, the current study sought to (a) explore differences on theta, beta, and TBR between those with ADHD symptoms and controls; (b) examine how EEG indices relate to measures of neuropsychological functioning and SCT symptoms; and (c) examine whether SCT uniquely relates to neuropsychological functioning after controlling for co-occurring symptomatology. Based on past studies, we predicted that those with ADHD symptoms would show greater theta activity, less beta activity, and greater TBR than controls, particularly in frontal and frontocentral regions. In addition, we predicted that the strongest group difference would be for TBR followed by theta and beta. Given the absence of data on neurophysiological indices and SCT, we had no *a priori* predictions among theta, beta, and TBR. However, because SCT seems to be related to ADHD symptoms, which are related to these frequencies, it seemed likely that SCT would relate in a similar manner. Based on past work, we predicted that SCT would be positively associated with a laboratory task measure of sustained attention and that this relationship would remain significant after controlling for co-occurring symptomatology.

Method

Participants

Participants were recruited in the southeastern United States via flyers and targeted letters to families in the area using a

Table 1. Demographic and Clinical Differences Between Groups.

	ADHD symptoms (n = 21)	Control (n = 20)	t	df	p	d
Age	9.71 (2.00)	9.38 (1.72)	−0.58	40	.57	0.18
Family income	99,187 (89,058)	89,866 (38,830)	−0.37	29	.71	0.14
CBCL Attention	69.81 (8.69)	50.71 (1.59)	−9.90	21.33	**.00**	3.06
CBCL Externalizing	61.52 (10.42)	41.10 (7.56)	−7.27	40	**.00**	2.24
CBCL Internalizing	55.62 (8.20)	46.57 (7.95)	−3.63	40	**.00**	1.12
CBCL SCT	60.05 (8.83)	51.38 (2.54)	−4.32	23.28	**.00**	1.33
Reaction Time	48.88 (9.35)	49.60 (12.15)	0.22	39	.83	−0.07
Reaction Time Standard Error	56.58 (8.10)	47.63 (9.28)	−3.28	39	**.00**	1.03
Commissions	56.10 (7.56)	47.60 (12.78)	−2.61	32.74	**.01**	0.38
	ADHD symptoms (n = 21)	Control (n = 20)	χ^2	df	p	
Gender (% male)	71.4	47.6	2.47	1	.12	
Race (% Caucasian)	76.2	81.0	0.14	1	.71	

Note. CBCL = Child Behavior Checklist; SCT = sluggish cognitive tempo. Bold = significant ($p < .05$).

local database of families with children who fell in the age range of 7 to 12 years. Interested families contacted study personnel by phone and completed a verbal consent protocol prior to completing the Vanderbilt ADHD Rating Scale, Parent Version (VARS-P) over the phone to determine study eligibility. For children in the ADHD symptoms group, inclusion criteria were as follows: (a) 7 to 12 years of age, (b) clinically elevated ADHD symptoms on the VARS-P, and (c) right-handedness. Those who reported six or more symptoms of inattention and/or six or more symptoms of hyperactivity/impulsivity on the VARS-P were selected for the ADHD symptoms group.[1] Exclusion criteria were the following: (a) past diagnosis of autism spectrum disorder or (b) past diagnosis of bipolar disorder. These exclusion criteria were chosen due to the fact that these disorders likely involve different brain-based etiologies. For children in the control group, inclusion criteria were as follows: (a) 7 to 12 years of age, (b) fewer than three symptoms of ADHD on the VARS-P, and (c) right-handedness. Children in the control group were excluded if they had previously been diagnosed with any psychiatric disorder.

Following the phone screening, participants came to our university setting to complete laboratory tasks, questionnaires, and the EEG assessment protocol. Both the parent(s) and child completed consent and assent forms at the start of the laboratory assessment. After consent and assent, child participants completed the Conners' Continuous Performance Test II (CPT-II) and questionnaires while their parent(s) completed questionnaires about their child. Following a brief break, the children were taken to another room where they completed the EEG protocol (see below for details). The entire assessment lasted approximately 2 hours, and families were compensated with US$50 for their time and effort. Child participants with ADHD symptoms who were currently taking stimulant medication (n = 8) were asked to not take medication within 24 hours of their assessment.[2]

Based on our inclusion and exclusion criteria, 21 children qualified for the ADHD symptoms group and 20 children qualified for the control group. Group differences on demographic and clinical characteristics are presented in Table 1.

Measures

Parent information form. This parent report measure includes questions about developmental, medical, family, and social history as well as questions about sociodemographic characteristics (e.g., age, gender, family income, etc.).

Child Behavior Checklist (CBCL). The CBCL (Achenbach & Rescorla, 2001) is a 113-item paper-and-pencil questionnaire completed by parents. Parents are asked to indicate how often the behavior described in each item is true of their child using a 3-point scale (*often/always true*, *sometimes true*, and *not true*). The reliability (α = .63-.97) and validity of the CBCL are well-established (Achenbach & Rescorla, 2001). The SCT item set from the CBCL has also shown adequate reliability in past research with school-age children (Becker, Luebbe, Fite, Stoppelbein, & Greening, 2013; Garner, Marceaux, Mrug, Patterson, & Hodges, 2010). These items include the following: "confused or seems to be in a fog," "daydreams or gets lost in his/her thoughts," "stares blankly," and "underactive, slow moving, or lacks energy." The reliability of the SCT construct in the current sample was high (α = .80). Age- and gender-corrected T scores were used for the current study.

Conners' CPT-II. The CPT-II (Conners, 2000) is a vigilance task in which respondents are asked to press the space bar when any letter other than "X" appears on the computer screen. Children completed the task after abstaining from stimulant medication for ADHD symptoms (if applicable) for 24 hours. The current study examined *T* scores for Hit Reaction Time, reaction time variability (i.e., Hit Reaction Time Standard Error), and Commissions (i.e., clicking on the space bar in response to the infrequent letter "X").

VARS-P. The VARS-P (Wolraich, Feurer, Hannah, Baumgaertel, & Pinnock, 1998) is a parent rating scale that measures the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; APA, 1994) symptoms of ADHD. This scale was utilized to determine ADHD symptom status for the majority of our study participants (see Note 1).

EEG acquisition. EEG was assessed using 32 tin electrodes mounted in a stretch-lycra electrode cap based on the 10-20 system, and placed on the participant's head using known anatomical landmarks. The ground electrode is mounted in the cap on the midline between the frontal pole (Fpz) and the frontal site (Fz). The reference electrode was placed on the left ear (M1), and data were acquired from an electrode placed on the right ear (M2), so that an offline digitally derived, averaged ears' reference could be computed. Eye movements were recorded to facilitate artifact scoring of the EEG using site FP1. Electro-Gel was used as the conducting medium. All electrode impedances were below 5,000 Ω. Signals were amplified with Neuroscan Synamps RT, bandpass filtered (0.05-100 Hz; 60-Hz notch filter enabled), and digitized at 500 Hz. After collection, data were generally processed in the following manner: EEG signals were visually scored, artifacts (e.g., eye movements, muscle movements) were removed, blinks were corrected using a regression-based eye blink correction (Semlitsch, Anderer, Schuster, & Presslich, 1986), and data were visually inspected a second time to ensure proper correction.

Theta, beta, and TBR were examined during a baseline resting state at the beginning of all EEG administration sessions during a 4-min resting period of eyes open (2 min) and eyes closed (2 min) counterbalanced between participants. All artifact-free epochs that were 1.024 s in duration were extracted through a Hamming window and rereferenced using an average-ears reference. Contiguous epochs were overlapped by 50%. A fast Fourier transform (FFT) was used to calculate the power spectra (e.g., theta and beta). Because theta power is related to decreased cortical activity and beta is related to cortical arousal, total power within these spectra (theta: 4-8 Hz, beta: 13-30Hz) frequency range was obtained.

Data Analysis

Data were analyzed via the SPSS 24 statistical package. Data were first analyzed for outliers by examining standardized *z* scores (i.e., between −3.29 and +3.29). A small number of outliers ($n = 15$ values across five participants) were identified and were subsequently changed to values reflecting *z* scores of 3.29 to reduce the influence of these outliers on study results (Field, 2009). Analysis of skewness and kurtosis revealed elevated *z* scores for skewness and kurtosis for study variables, so the nonparametric Spearman's correlation coefficient was utilized for correlational analyses. For comparing groups on age- and gender-corrected measures (e.g., CBCL, CPT), independent samples *t* tests were utilized. For cases where Levene's test was significant, adjusted *t* tests were utilized. Analysis of covariance (ANCOVA) was utilized for comparing those with and without ADHD using age and gender as covariates on EEG measures. Following these between-group analyses, correlations among our study measures were explored followed by regression analyses.

Results

Between-Group Analyses

Results of between-group analyses for demographic and clinical characteristics appear in Table 1, and results for EEG analyses appear in Table 2. No group differences were found for age, family income, or CPT Hit Reaction Time (ps > .05; ds = −0.07 to 0.18). In contrast, children in the ADHD symptoms group had significantly greater CBCL Attention Problems ($d = 3.06$), CBCL Externalizing Problems ($d = 2.24$), CBCL Internalizing Problems ($d = 1.12$), CBCL SCT Symptoms ($d = 1.33$), CPT Hit Reaction Time Standard Error ($d = 1.03$), and CPT Commissions ($d = .38$; ps < .05). Chi-square tests revealed no differences between groups on race or gender (ps > .05).

For EEG analyses (see Table 2), no significant differences were found between groups for theta activity in the frontal, frontocentral, or central regions (ps > .05). For beta activity, no group difference was found in the frontal region, but there were marginal differences in the frontocentral and central regions with those with ADHD symptoms showing less activity relative to controls (ps < .10; ds = −2.62 and −2.85). Finally, significant and large magnitude differences were found between those with ADHD symptoms and controls on TBR in frontal, frontocentral, and central regions (ps < .05; ds = 2.99-3.52).

Correlations and Regression Analyses

Results of correlational analyses appear in Table 3. First, and not surprisingly, group status was significantly and/or marginally correlated with EEG TBR indices (i.e., similar to between-group analyses). In contrast, CBCL SCT symptoms were not significantly or marginally correlated with TBR indices. Notably, the relationships between SCT symptoms and TBR were very small in frontal and frontocentral regions but became larger in parietal and parietal-central regions

Table 2. Differences on EEG Activity Between Groups.

	ADHD symptoms (n = 21)	Control (n = 20)	F	df	p	d
Theta						
Frontal	5.69 (0.41)	4.86 (0.43)	1.91	1	.18	1.98
Frontocentral	8.78 (0.63)	7.90 (0.65)	0.91	1	.35	1.37
Central	10.93 (0.85)	10.15 (0.87)	0.40	1	.53	0.91
Beta						
Frontal	.89 (0.14)	1.06 (0.14)	0.73	1	.40	−1.21
Frontocentral	.88 (0.13)	1.22 (0.13)	3.25	1	*.08*	−2.62
Central	0.74 (0.10)	1.04 (0.11)	3.73	1	*.09*	−2.85
TBR						
Frontal	9.16 (0.89)	5.99 (.91)	5.95	1	**.02**	3.52
Frontocentral	12.11 (1.02)	8.54 (1.04)	5.79	1	**.02**	3.47
Central	16.73 (1.38)	12.54 (1.42)	4.33	1	**.04**	2.99

Note. Values in the table reflect adjusted means after controlling for age and gender. Bold = significant group differences (p < .05). Italics = marginally significant (p < .1). Cohen (1988) recommends the following interpretation of d: small = 0.2, medium = 0.5, large = 0.8. EEG = electroencephalography; TBR = theta/beta ratio.

Table 3. Correlations Among Measures (Spearman's Rho).

		1	2	3	4	5	6	7	8	9	10
1.	TBR Frontal	1									
2.	TBR Frontal Central	**.93**	1								
3.	TBR Central	**.81**	**.93**	1							
4.	TBR Parietal	**.60**	**.76**	**.60**	1						
5.	TBR Parietal Central	**.67**	**.82**	**.96**	**.98**	1					
6.	CBCL Attention Problems	.15	.21	.18	.21	.23	1				
7.	ADHD Symptom Group	**.33**	**.36**	*.29*	*.28*	*.29*	**.88**	1			
8.	SCT	.01	.05	.12	.18	.19	**.77**	**.58**	1		
9.	Hit RT	.25	*.28*	*.29*	.17	.18	−.01	−.02	−.05	1	
10.	Hit RT Standard Error	.26	**.34**	**.37**	**.38**	**.37**	**.54**	**.52**	**.45**	**.52**	1
11.	Commissions	−.25	−.25	−.25	−.10	−.25	**.39**	**.34**	**.41**	**−.70**	.04

Note. Bold = significant (p < .05). Italics = marginally significant (p < .1). ADHD Symptom Group (0 = control; 1 = ADHD symptom group). TBR = theta/beta ratio; CBCL = Child Behavior Checklist; SCT = sluggish cognitive tempo; RT = reaction time.

(i.e., frontal and frontocentral TBR rs = .01-.05, parietal and parietal-central TBR rs = .18-.19). In contrast, the strongest correlations between ADHD symptom group status and TBR tended to be in frontal regions (i.e., TBR frontal and frontocentral rs = .33-.36; central, central-parietal, and parietal sites rs = .28-.29). An adjusted t test comparing dependent correlations (Chen & Popovich, 2002) between ADHD symptom group status and TBR, and SCT symptoms and TBR revealed that correlations were significantly different between groups for TBR at frontal (t = 2.35, p < .05) and frontocentral sites (t = 2.29, p < .05) but not for TBR at central, parietal-central, or parietal sites (ps > .05).

In relation to the CPT-II task, Hit Reaction Time Standard Error, an index of sustained attention was significantly related to TBR in all locations (rs = .34-.38) except for frontal (r = .26). In contrast, Commissions were not significantly related to any location for TBR, and only in two areas (frontocentral and central) was TBR marginally related (p < .1) to Hit Reaction Time (rs = .28-.29).

In relation to symptoms and CPT-II task performance, ADHD symptom group status was significantly and positively associated with Hit Reaction Time Standard Error (r = .52) and Commissions (r = .34) but not Hit Reaction Time (r = −.02). Similarly, SCT was significantly and positively associated with Hit Reaction Time Standard Error (r = .45) and Commissions (r = .41) but not Hit Reaction Time (r = −.05). To examine whether SCT symptoms uniquely related to neuropsychological functioning, regression analyses were utilized with the following predictors: ADHD symptom group status, CBCL SCT symptoms, and CBCL Internalizing Problems.[3] Table 4 displays results from regression analyses. For the Hit Reaction Time and Commissions T score analyses, neither model was significant (ps > .05). In contrast, the model predicting the Hit Reaction Time Standard

Table 4. Regression Analyses Predicting Neuropsychological Functioning.

	Reaction time			Reaction time standard error			Commissions		
	Constant			Constant			Constant		
Predictors	59.69	*b*	*p*	36.34	*b*	*p*	26.63	*b*	*p*
ADHD Symptom Group		1.27	***.06***		7.33	.38		4.78	.22
SCT Symptoms		0.08	***.06***		0.44	.35		0.20	.14
Internalizing Problems		−0.31	−.26		−0.24	−.23		0.23	.19
Total R^2		.05			**.33**			*.19*	

Note. Italics = marginally significant ($p < .1$). ADHD Symptom Group (0 = control; 1 = ADHD symptom group). *b* = unstandardized beta; SCT = sluggish cognitive tempo.
Bold = $p < .05$.

Error *T* score was significant ($p < .05$). More specifically, ADHD symptom group (β = .38) and SCT symptoms (β = .35) were both significant and unique predictors of this measure of sustained attention while internalizing problems was not a significant predictor ($p > .05$).

Discussion

The current study had three primary goals: (a) to explore differences on theta, beta, and TBR between those with ADHD symptoms and controls; (b) to examine how EEG indices relate to measures of neuropsychological functioning and SCT symptoms, and (c) to examine whether SCT uniquely relates to neuropsychological functioning after controlling for co-occurring symptomatology. Based on past studies, we predicted that those with ADHD symptoms would show greater theta activity, less beta activity, and greater TBR than controls, particularly in frontal and frontocentral regions. In addition, we predicted that the strongest group difference would be for TBR followed by theta and beta. Given the absence of data on neurophysiological indices and SCT, we had no *a priori* predictions among theta, beta, and TBR. However, because SCT is highly related to ADHD symptoms, which are related to these frequencies, it seemed likely that SCT might relate in a similar manner. We also predicted that SCT would be positively associated with laboratory task measures of sustained attention and that this relationship would remain positive after controlling for co-occurring symptomatology.

In general, our predictions were in line with results of the current study. Consistent with past studies, large magnitude differences were found between children with ADHD symptoms and controls on our EEG indices, particularly for TBR in frontal and frontocentral areas. For these areas, our effect sizes of 3.52 and 3.47, respectively, are on par with the value of 3.08 found in a past meta-analysis (Snyder & Hall, 2006). While significant differences were not found for theta or beta activity, it should be noted that our effect sizes were generally consistent with past studies. For example, for frontal and frontocentral areas on theta, we found values of 1.98 and 1.37, respectively, which are comparable with the value of 1.31 found in a past meta-analysis (Snyder & Hall, 2006). Similarly, for beta, we found values of −1.21 and −2.62 in frontal and frontocentral regions which are in the appropriate direction of less activity when compared with past research (Snyder & Hall, 2006; mean effect size = −0.51). At the same time, the effect sizes here were larger than we predicted (i.e., smaller than theta) and somewhat larger than past studies. Consistent with past research, TBR appears to be an index that best differentiates children with ADHD symptoms compared with controls particularly when frontal and frontocentral activity are considered.

In relation to SCT, although we did not have a specific *a priori* prediction, we assumed that SCT might relate to EEG indices given its high correlation with ADHD symptoms (e.g., $r = .58$ in our study). Interestingly, SCT symptoms were not significantly related to any EEG index and the lack of association was particularly pronounced for frontal and frontocentral areas. For example, ADHD symptom group status was correlated .33 and .36 with these areas, respectively, while SCT was correlated at only .01 and .05. These data suggest that the strong support for the association between ADHD symptoms and activity in frontal and frontocentral areas may not be the case for SCT symptoms. In contrast to this finding, symptoms of SCT were strongly associated with a measure of sustained attention, reaction time variability on the CPT-II ($r = .45$). In addition, SCT symptoms remained significantly associated with sustained attention after controlling for ADHD symptoms and internalizing symptoms. In contrast, while SCT symptoms were related to commission errors in bivariate correlations ($r = .41$), this relationship became nonsignificant after controlling for ADHD symptom group and internalizing symptoms, suggesting that SCT symptoms are more strongly associated with laboratory indices of sustained attention rather than inhibition. This latter finding is consistent with the past finding that a differentially stronger relation exists between SCT and ADHD inattentive symptoms than hyperactivity/impulsivity symptoms (Barkley, 2016; Willcutt et al., 2012).

Overall, these results are consistent with speculation that SCT might involve a distinct attention problem (Barkley, 2016), although more research is needed to determine whether SCT represents a distinct attention disorder and which specific brain regions and brain networks are associated with SCT symptoms. For example, the only other study to date that has examined SCT in relation to brain functioning (Fassbender, Krafft, & Schweitzer, 2015) found evidence for hypoactivity in the left superior parietal lobe during a cued Flanker task, which suggests that SCT may be associated with reorienting or shifting of attention rather than executive attention (Becker et al., 2016). As noted by Barkley (2016), "there is a great need for research on the etiologies and cognitive nature of SCT . . . " (p. 157).

While the current study has a number of strengths, some limitations must be noted. First, our sample was relatively small and sufficiently powered to detect only large effects. At the same time, our effect sizes are largely consistent with past studies and the number of significant results in our study speaks to the robustness of the relationships among the variables in the study. Another limitation is that our sample consisted of participants across two different studies, although it should be noted that the two ADHD symptom groups did not differ on any study variables. In addition, participants from the second sample constituted only 16.6% of our total sample. Another limitation of our study was that we did not formally diagnose ADHD, so our ADHD symptom group represents those with clinically elevated ADHD symptoms rather than those formally diagnosed with ADHD. A final limitation of our study was our inability to separately examine inattention and hyperactivity/impulsivity symptoms. As participants did not complete the VARS-P in both samples and the CBCL parent report does not separate inattention and hyperactivity/impulsivity, we were not able to separately examine relations with inattention and hyperactivity/impulsivity symptoms.

Although there are some limitations to our study, the study offers an additional examination of EEG differences between children with and without ADHD symptoms and the first examination of how resting state EEG activity relates to SCT symptoms. For both theta and TBR, our findings were closely in line with past meta-analytic findings, but we also found significantly less beta activity in those with ADHD symptoms relative to controls compared with past studies. As noted earlier, greater variability in beta findings has been observed in comparison with variables such as TBR, so future studies should continue to explore the role of beta activity in ADHD. Our results also suggest that SCT symptoms, unlike ADHD symptoms, show limited associations with frontal and frontocentral resting state activity, but our study did not implicate any specific brain region that appears to be associated with SCT uniquely. Future research should continue to explore how SCT relates to neurophysiological activity and the functioning of brain networks that were not examined in the current study. In addition, while our study focused on resting state activity, future EEG studies might use event-related potentials (ERPs) during tasks that tap into the attention networks thought to be associated with SCT (e.g., attentional orienting or shifting). Finally, our study utilized a healthy control group for comparisons. Future studies should continue to explore SCT symptoms in groups with other psychiatric symptoms (e.g., those with elevated internalizing problems) to better understand whether SCT symptoms are qualitatively different outside of the context of ADHD symptoms and whether there may be differences in relation to neuropsychological and neurophysiological functioning.

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Notes

1. Seven additional subjects from another study examining computerized cognitive training for children with ADHD and a comorbid anxiety disorder were also included in the ADHD group in this study. These children went through the exact same electroencephalography (EEG) protocol at pretreatment as participants in the current study. The evaluation process was slightly different, though, as the assessment of ADHD symptoms for this study involved a semistructured diagnostic interview rather than the Vanderbilt scale (i.e., Anxiety Disorders Interview Schedule for Children [ADIS-C]). At the same time, both assessment approaches evaluated *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) symptoms of ADHD. The same exclusion criteria applied. These seven participants did not differ from those in the original ADHD symptoms group ($n = 14$) on any study variables ($ps > .05$).
2. Of the seven participants from the cognitive training study, three were currently taking a stimulant medication for ADHD, and none of these participants were taking medication on the day of assessment, as the assessment was completed over the summer months and all three were not taking a stimulant during this time window.
3. Age and gender were not included in the analyses, as the predictors and dependent variables were age- and gender-corrected *T* scores. Internalizing symptoms were included given its consistent relations with ADHD, SCT, and neuropsychological functioning.

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