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The contribution of sluggish cognitive tempo to processing speed in survivors of pediatric brain tumors

Rachel K. Peterson^{a,b}, Kelly Jones^a, Lisa A. Jacobson^{a,b}

^aKennedy Krieger Institute Neuropsychology Department;

^bJohns Hopkins School of Medicine Department of Psychiatry and Behavioral Sciences

Abstract

Sluggish Cognitive Tempo (SCT) describes a pattern of under-activity, poor initiation, and slowness. It was first reported within the Attention Deficit Hyperactivity Disorder (ADHD) literature and found to be positively associated with the inattentive symptoms of ADHD and negatively or not significantly associated with the hyperactivity/impulsivity symptoms of ADHD. SCT has since been considered applicable to the pediatric oncology population given the emergence of inattentive, sluggish symptoms secondary to cancer treatment. The present study examined the unique contribution of SCT to various processing speed skills in a clinical sample of pediatric brain tumor (BT) survivors in order to determine the degree to which SCT explained timed “cognitive” processing components. Measures included speeded naming, graphomotor speed, and speeded inhibition. Hierarchical linear regression analyses were used to predict performance-based measures of processing speed. After controlling for verbal ability and inattention, SCT, particularly Daydreamy SCT ($\beta = -0.698$, $p = 0.023$), explained 28% of variance in speeded inhibition. SCT did not add significantly to the prediction of speeded naming or graphomotor speed. Findings suggest that the “daydreamy” aspect of SCT, rather than “sluggishness” per se, may be related to more complex, cognitively-demanding tasks with greater executive functioning burdens in BT survivors. Implications for intervention for oncology survivors as well as future research directions are discussed.

Keywords

Neuro-oncology; pediatrics; SCT; executive function; ADHD

Survivors of pediatric brain tumors are at risk for cognitive deficits attributable to disease and treatment factors that impact the Central Nervous System (Ris & Noll, 1994). Models have been proposed to conceptualize the core neurocognitive skills affected in survivors and their impact on functional outcomes.; Palmer (2008) suggested that processing speed deficits, which are impacted by factors such as the tumor itself, cancer treatment, and the patient’s age, are the primary pathway for cognitive deficits in brain tumor (BT) survivors. According to this model, working memory deficits that emerge

^{Correspondence} CONTACT Rachel K. Peterson PetersonR@KennedyKrieger.org 1750 E.Fairmount Avenue, Baltimore, MD 21231, USA.

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following cancer-directed treatment are a result of weaknesses in processing speed and attention, and have a cascading effect on other cognitive skills such as IQ and academic achievement. Follow-up work by Wolfe et al. (2012) proposed that attention, working memory, processing speed, and executive function all have an equal and interrelated impact on IQ and academic achievement. King and colleagues' empirical model (King et al., 2019) suggested that oral processing speed was the core cognitive skill most widely associated with neurodevelopmental risk factors and outcomes, though working memory and attention also uniquely contribute to neurocognition. These three models underscore the importance of investigating core cognitive functions underlying neurocognition in brain tumor survivors, particularly processing speed, which plays a fundamental role in overall intellectual functioning and academic achievement in this population (Smith et al., 2014).

Processing speed as a construct has been conceptualized as a foundational ability that supports the development of higher-level cognitive skills (Schatz et al., 2000). Processing speed is thought to be dependent on the integrity of neuronal white matter (Turken et al., 2008), making it particularly vulnerable to BT treatments such as surgery (Soelva et al., 2013) and radiation therapy (Reddick et al., 2005; Rueckriegel et al., 2010). Indeed, reduced white matter has been associated with slower processing speed in BT survivors (Aukema et al., 2009). Thus, better understanding of the observed processing speed deficits in BT survivors is needed. However, processing speed can be observed across a variety of cognitive tasks including motoric (i.e., psychomotor speed), oral (i.e., speeded naming, oral fluency), and executive function tasks (i.e., tasks requiring greater demands for response selection and preparation or executive control; Jacobson et al., 2011). Thus, greater precision in examining the construct of processing speed may help to clarify our understanding of outcomes in pediatric BT survivors.

One way that researchers have conceptualized the cognitive deficits in survivors of pediatric brain tumors is the Sluggish Cognitive Tempo (SCT) framework, which describes a pattern of under-activity, poor initiation, and slowness (Penny et al., 2009). SCT was initially discussed in the context of ADHD and is associated with specific symptoms of ADHD, particularly inattention (Carlson & Mann, 2002; Harrington & Waldman, 2010; Hartman et al., 2004; McBurnett et al., 2001).

Neurocognitive deficits in brain tumor (BT) survivors have been characterized similarly to inattentive ADHD symptoms and it has been hypothesized that the symptoms captured by the SCT construct may have particular utility for conceptualizing the neurocognitive deficits in survivors of pediatric BT (Kahalley et al., 2011). The possibility of SCT in oncology survivors was first proposed by Reeves et al. (2007). In their retrospective analysis, symptoms of SCT were assessed in 80 survivors of Acute Lymphoblastic Leukemia (ALL) and 19 of their typically-developing siblings. They found significantly more SCT symptoms in survivors as compared to siblings. Their findings also indicated negative associations between SCT and estimated IQ scores as well as basic reading skills. Taken together, these findings suggested the potential utility of applying the SCT model to oncology survivors. In a follow-up study, Willard et al. (2013) found that survivors of BT demonstrated significantly greater symptoms of SCT than survivors of ALL or controls. Moreover, SCT was associated with attention problems, working memory deficits, and the presence of

a ventriculoperitoneal shunt, thereby providing additional support for the SCT construct within the oncology population.

Despite emerging evidence supporting the validity of sluggish cognitive tempo in the pediatric oncology population, little is known about the underlying mechanisms and processes responsible for these SCT symptoms. Although the moniker “SCT” presumes that these symptoms are attributable to slowed processing speed, the evidence to support this claim is mixed (for review, see Mueller et al., 2014). For example, within the ADHD literature, some researchers found an association between speeded tasks (i.e., processing speed) and SCT symptoms (Jacobson et al., 2018; Lundervold et al., 2011; Solanto et al., 2007). In contrast, other researchers did not find evidence of an association between SCT and processing speed and instead proposed that the neurocognitive correlates of SCT related to difficulties with sustained attention (Bauermeister et al., 2012; Tamm et al., 2018; Willcutt et al., 2014). Limitations were noted with these studies, however, including the limited item pool used to assess SCT in these studies, with either four (Bauermeister et al., 2012) or six (Willcutt et al., 2014) items. In many recent studies, investigators have relied on more comprehensive measures of SCT consisting of 14 items (Penny et al., 2009) or more (McBurnett et al., 2014).

Better understanding of the “cognitive” component of SCT particularly for oncology patients is important when considering cognitive implications of treatment as well as appropriate interventions secondary to treatment effects. Therefore, the purpose of this exploratory study was to identify the unique contribution of SCT to timed processing speed outcomes with varying cognitive complexity in a clinical sample of pediatric BT survivors. However, prior research has shown that cancer survivors have notable fine motor dexterity weaknesses secondary to treatment (i.e., chemotherapy associated neuropathy; Mora et al., 2016), thus weaknesses in motor processing speed would not be unexpected. To better delineate the cognitive construct of SCT, varying speeded outcomes were examined: speeded naming, graphomotor/psycho-motor speeded output, and inhibition efficiency (a combination of speed and executive function). We hypothesized that SCT would contribute more to tasks with both processing speed and executive function demands than verbal fluency or speeded motor tasks.

Methods

Participants

Retrospective data were gathered from clinical neuropsychological evaluations. The sample was comprised of 58 pediatric brain tumor survivors who had been referred for outpatient neuropsychological evaluation at an academic medical center as part of standard of care. Data were entered into the electronic medical record in the course of care and accessed following IRB approval. Patient demographics are listed in Table 1. Treatment information was confirmed from medical records to verify oncology diagnosis, age at diagnosis, sex, race/ethnicity, and type of cancer-directed treatment.

For the purpose of this study, data from age-appropriate measures of speeded output and executive functioning as well as parent-report rating scales of ADHD and SCT were

utilized. All rating scales and performance-based measures were administered at the time of the outpatient neuropsychological assessment. Composite scores were calculated using age-appropriate measures of common constructs and subsamples with data on each measure of interest are shown in Table 2.

Measures

Cognition—The Verbal Comprehension Index (VCI) from the age-appropriate Wechsler scales [Wechsler Intelligence Scale for Children – Fourth Edition, Wechsler (2003); Wechsler Intelligence Scale for Children – Fifth Edition, Wechsler (2014) (WISC-V); Wechsler Adult Intelligence Scale – Fourth Edition, Wechsler (2008) (WAIS-IV)] was used as an untimed estimate of intellectual functioning. These are gold-standard measures of verbal ability with demonstrated reliability and validity (Wechsler, 2003, 2008, 2014). Composited verbal ability scores were standard scores (mean of 100, standard deviation of 15).

Attention—The ADHD Rating Scale – Fourth and Fifth Edition (ADHD RS-IV/-5; DuPaul et al., 1998, 2016) is an 18-item scale (9 inattentive items and 9 hyperactive/impulsive items) assessing parent report of ADHD symptomatology according to DSM-IV/-5 criteria (American Psychiatric Association, 2000, 2013). For the purpose of this study, only the scale sum of the Inattentive symptoms was utilized. Items are rated on a 4-point frequency scale (0 = Never or Rarely; 1 = Sometimes; 2 = Often; 3 = Very Often). Coefficient alphas on the home version of the ADHD RS are calculated at .92 for total score, .86 for inattention, and .88 for hyperactivity-impulsivity (DuPaul et al., 1998).

SCT—The Sluggish Cognitive Tempo Scale (SCT; Penny et al., 2009) is a 14-item scale assessing parent report of associated symptoms, which have been shown to make up three distinct factors/subscales: Sleepy/Sluggish, Low Initiation/Persistence, and Daydreamy (Jacobson et al., 2018; Jacobson & Mahone, 2019). Examples of items that fall under the Sleepy-sluggish subscale include: “Seems drowsy”, “Appears tired or lethargic”, and “Is under-active or slow-moving”. Examples of items that fall under the Low Initiation subscale include: “Effort on tasks fades quickly”, “Needs extra time for assignments”, and “Is apathetic”. Examples of items that comprise the Daydreamy subscale include: “Gets lost in his/her own thoughts”, and “Seems to be in a world of his/her own”. Items are rated on a 4-point scale (0 = Never or Rarely; 1 = Sometimes; 2 = Often; 3 = Very Often). The SCT has strong psychometric properties (Penny et al., 2009), with reliability coefficients ranging from .70 (Daydreamy) to .83 (Sleepy) and .87 (Low Initiation and Total SCT).

Processing speed—Processing speed was examined across different measures. Subtests measuring similar constructs were combined into composite scores, each with a mean of 10 and standard deviation of 3.

Speeded naming was assessed using either the naming trial from the NEPSY-II Inhibition subtest or the Color Naming trial from the DKEFS Color Word Interference subtest.

Graphomotor processing speed was examined via the Coding subtest from the WISC-IV/-V or WAIS-IV.

Verbal response inhibition was assessed with the NEPSY-II Inhibition-Inhibition subtest or the Inhibition trial from the DKEFS Color-Word Interference subtest.

All of these measures have strong reliability coefficients. The reliability coefficient for the NEPSY-II Inhibition subtest ranges from 0.8 to 0.9 depending on the age range of the child. The DKEFS color-word interference subtest has a reliability coefficient of 0.7. Reliability coefficients for the WISC-IV, WISC-V, and WAIS-IV Coding subtest range from 0.7 to 0.85.

Statistical analysis—Descriptive statistics and one sample t-test analyses were performed to characterize the sample. Data were examined for normality, before hierarchical linear regression analyses were used to predict performance-based measures of processing speed. As part of this statistical technique, variables were added to the model in separate steps or blocks. Verbal ability was entered first, then ADHD-I symptom total score from the ADHD RS-IV/–5, and lastly the SCT subscales together. Given the exploratory nature of this study, correlations were performed to examine associations between the SCT subscales and medical, demographic, and neuropsychological measures.

Results

Performance on neuropsychological measures relative to normative means is provided in Table 2. Data were normally distributed. BT survivors as a group scored in the average range on measures of verbal ability, speeded naming, and inhibition; however, there was a large range of scores, spanning well below and well above average. BT survivors as a group scored significantly below the normative mean on the graphomotor speeded task (Coding; $p < 0.001$).

Utilizing hierarchical regression analysis, we examined the impact of each hypothesized predictor (verbal ability, inattention symptoms, and SCT subscales) on each of the timed outcomes (Table 3). After controlling for verbal ability and symptoms of inattention, the Daydreamy SCT subscale was the only significant SCT factor and explained 28% of the variance in Inhibition ($\beta = -.698$, $p = .023$). Given the moderate correlation between VCI and the SCT Low Initiation subscale (-0.30), a separate hierarchical regression analysis was performed without controlling for VCI. The results remained significant ($p < .05$). Interestingly, SCT did not add significantly to the prediction of speeded naming or graphomotor speed (all $p > 0.05$).

A correlation analysis (Table 4) showed a negative association between specific SCT subscales and age at diagnosis, such that younger age at diagnosis was associated with slower initiation and more daydreamy symptoms. Verbal ability was negatively associated with specific SCT scales as well. Specifically, lower verbal abilities were associated with greater SCT total symptoms and slower initiation speed. The Inattentive symptoms of ADHD were all strongly positively associated with the SCT subscales such that more ADHD-I symptoms were associated with greater SCT symptoms.

Discussion

Given initial research on the applicability of SCT in understanding neuropsychological deficits in pediatric oncology survivors as well as the well-documented weaknesses in processing speed in this population, this study aimed to investigate the relative association between SCT symptoms and speeded outcomes with increased cognitive demands in pediatric BT survivors.

In this study, the Daydreamy subcomponent of SCT accounted for a significant amount of the variance in speeded inhibition performance, above and beyond verbal ability and inattention, but did not add to prediction of verbal (speeded naming) or graphomotor speed. These findings suggest that SCT does not contribute equally to all types of speeded processing in pediatric BT survivors. Rather, the “daydreamy” aspect of SCT may be sensitive to more complex, cognitively-demanding tasks with greater executive functioning burdens. Among SCT scale items, Factor 1 (sleepy/sluggish) is characterized by energy (i.e., lethargy and fatigue), Factor 2 is related to the slowness of initiation and task completion (i.e., needing more time, slow to initiate), and the third Factor is the only scale that includes overt cognitive descriptors (i.e., in own world, lost in thoughts). Thus, the core cognitive aspects of the SCT construct assessed by the third factor would be more likely to show associations with more cognitively demanding speeded tasks as opposed to simpler ones. These findings are consistent with work by Kofler et al. (2019), who similarly found that children with SCT are not globally sluggish/slow, but rather their SCT symptoms appear to be related to a large extent to executive function weaknesses, particularly an inefficient/slowed working memory system and fast inhibition system. As a result, these youth may present as absent-minded or failing to follow through; however, this is due to these individuals requiring extended time to manage information in their working memory as well as an overactive inhibition system that likely terminates thoughts prematurely, thereby preventing intended behaviors from starting or being completed.

It is also important to note that of the processing speed tasks examined, graphomotor speed was the only speeded task where the sample, on average, performed below the mean. This finding, consistent with other published evidence, suggests that pediatric oncology survivors may be more likely to show deficits on speeded graphomotor/fine motor tasks than on speeded naming tasks, which highlights the importance of assessing processing speed in multiple ways, including via parent report of SCT. It is well documented that certain cancer treatments impact fine motor dexterity (i.e., vincristine associated peripheral neuropathy; Mora et al., 2016), which could impact performance on graphomotor speeded tasks. However, cancer treatments such as chemotherapy and radiation directly impact white matter tracts, which are associated with processing speed (Aukema et al., 2009). Perhaps this suggests that speeded naming tasks are not as sensitive to the processing speed weaknesses typically seen in pediatric oncology survivors.

Several methodological limitations must be considered. First, this is a cross-sectional study and causation cannot be determined. For example, slow initiation can be due to a number of factors outside of the cancer treatment, such as apathy, which has been highlighted in other long-term cancer studies (Fox & King, 2016). Use of a cohort study would

allow researchers to better understand the cause of the slowed initiation speed, which would thereby impact appropriate interventions for these individuals (e.g., psychotherapy for apathy versus academic accommodations for slowed processing speed). Second, the diagnostically heterogeneous BT sample limits determination of the extent to which specific disease and treatment factors, such as tumor type, greater irradiation dose/volume, and history of hydrocephalus and/or VP shunt might influence cognitive outcomes of the survivors in this sample (Duffner, 2010). In addition, this study did not include a comparison group, thereby limiting our comparisons to such associations in typically developing youth. Moreover, the processing speed measure used (i.e., Coding) involves visual-motor and fine motor demands. As such, there is the potential that impairments observed in this aspect of the processing speed domain may be attributable, at least in part, to other deficits such as fine motor weaknesses. That being said, this study sample included a large age range of patients, and sample data utilized in these analyses was not limited by level of cognitive functioning. In other words, all patients completing any of the measures of interest were included in the sample, including those with some degree of cognitive impairment, thereby utilizing a more representative sample of patients seen for neuropsychological evaluation. However, the large range in time since diagnosis may confound some of these results. Individuals within the first six months of diagnosis are in the acute phase of treatment and neurocognitive results may be impacted by fatigue secondary to treatment. As individuals move further out from time of treatment (2–5 years post-diagnosis), these acute effects resolve; however, “late effects” tend to emerge. Given the range of time since diagnosis, we are not able to discern whether the findings are acute or enduring in nature. Moreover, this is a clinical sample, which is important to consider when interpreting the results. That is, studies that utilize retrospective data acquired from a clinical population may be susceptible to bias due to the potential for differential participation of eligible study participants (Hernan et al., 2004; Ness et al., 2009). Deriving estimates from a convenient population limits the generalizability of the results and should be interpreted with caution. For example, our sample is largely Caucasian. While this is consistent with other research samples (Moody et al., 2011), it may limit the generalizability to more diverse demographic samples. Likewise, our sample is primarily female, which confers a higher risk for neurocognitive deficits (Dixon et al., 2018).

Despite these limitations, this study has important implications for clinical care. Ongoing monitoring and assessment of SCT may help to ensure that adequate supports, both at school and at home, are provided to these survivors. Within the classroom setting, students with SCT may be slower to complete their work or require additional time to complete tasks, placing them at risk for missing critical classroom instruction, interacting and participating in the learning experience, and delaying their ability to generate a reasonable approach to tasks (Jacobson et al., 2012). Likewise, SCT symptoms may confer risk for academic difficulties across domains (Jacobson & Mahone, 2019; Tamm et al., 2016). Therefore, these individuals will require accommodations and supports to compensate for or alleviate the SCT symptoms that contribute to academic weaknesses. Researchers have suggested that specific academic interventions created for adolescents with ADHD may decrease SCT symptoms. Pffiffer et al. (2007) created the Child Life and Attention Skills (CLAS) for children diagnosed with ADHD-I, adding components of self-awareness, goal setting,

behavior parent training, attention checks, and skills for independence to address difficulties with attention. Given that the symptoms of SCT include daydreaminess and adolescents with SCT can appear to be “in a fog” and unmotivated, incorporating strategies such as self-monitoring and attention checks may be beneficial. Smith and Langberg (2020) found that interventions that target homework and organization difficulties can also ameliorate some SCT symptoms.

Future oncology research should investigate the relationship between SCT and other neurocognitive skills, such as overall intellectual functioning and academic achievement, which are negatively impacted by slowed processing speed in this population (King et al., 2019; Palmer, 2008; Wolfe et al., 2012). In addition, while specific treatment modalities were not associated with SCT symptoms – likely due to small sample size, lack of power, and treatment overlap – it could be of interest to consider whether specific treatment complications such as presence of hydrocephalus, endocrine dysfunction, etc., are associated with SCT symptoms. These findings could help to predict which oncology patients would be at greater risk for specific neurocognitive outcomes and inform academic and cognitive interventions.

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Table 1.

Sample demographics.

	n (%)
Child Sex	
Female	35 (60.34)
Male	23 (39.66)
Race/Ethnicity	
Caucasian	39 (67.24)
African American	11 (18.97)
Did Not Report	6 (10.34)
Asian	1 (1.72)
Hispanic	1 (1.72)
Parent who completed rating scales	
Mother	55 (94.83)
Father	1 (1.72)
Both parents	1 (1.72)
Maternal education	
High School (HS) diploma or less	12 (20.69)
Some post-HS education	16 (27.59)
College degree	15 (25.9)
Advanced degree	13 (22.4)
Unknown/Did not disclose	2 (3.45)
Insurance	
Commercial	47 (81.03)
Medicaid	11 (18.97)
Age at evaluation in years [M(SD), range]	11.50 (4.62), 4–21
Age at diagnosis in years [M(SD), range]	8.74(4.61) 0.17–18.08
Age at diagnosis in years	
0–4	15(25.86)
5–9	20(34.48)
10–14	18(31.03)
15–21	5(8.62)
Time since diagnosis in years [M(SD), range]	2.83(3.02), 0.08–12.92
Treatment	
Only Resection	10(17.24)
Only Radiation	0(0)
Only Chemotherapy	5(8.62)
Resection + Radiation	6(10.34)
Resection + Chemotherapy	4(6.90)
Resection + Radiation + Chemotherapy	21(36.21)
Radiation +Chemotherapy	5(8.62)
Brain tumor location	

	n (%)
Supratentorial	35 (60.34)
Infratentorial	22 (37.93)
Both	1 (1.72)
Lobular tumor location ^a	
Frontal	7 (12.07)
Parietal	6 (10.34)
Temporal	10 (17.24)
Occipital	2 (3.45)
Suprasellar	17 (29.31)
Cerebellum	20 (34.48)
Brainstem	3 (5.17)
Multi-lobular	7 (12.07)
Histology	
Astrocytoma	6(10.3)
Chordoma	1(1.72)
Craniopharyngioma	4(6.9)
Dysembryoplastic Neuroepithelial Tumor (DNET)	2(3.45)
Ependymoma	7(12.07)
Ganglioglioma	4(6.9)
Germinoma	5(8.62)
Glioma NOS	9(15.52)
Medulloblastoma	9(15.52)
CNS Neuroblastoma	2(3.45)
Pilocytic Astrocytoma	6(10.34)
Pineoblastoma	2(3.45)
Primitive Neuroectodermal Tumor (PNET)	1(1.72)

^aValue is greater than 100% due to tumor location in multiple lobules.

Table 2.

Composite neuropsychological scores compared to normative values.

Composite	n	Mean	SD	Range	t	Cohen's d ⁵	P	% impaired ^c
Verbal Ability ¹	58	97.47 ^a	15.19	63–140	–1.6	–0.17	0.11	5.2
Speeded Naming ²	39	9.41 ^b	2.82	1–15	–1.31	–0.21	0.20	3.4
Inhibition ³	39	10.77 ^b	3.63	2–18	1.32	–0.21	0.20	5.2
Graphomotor Speed ⁴	58	6.93 ^b	3.08	1–14	–8.40	–0.99	<0.001	8.6

¹Composite score of Verbal Comprehension Index (VCI) from the WISC-IV/5 or WAIS-IV²Composite score of NEPSY-II Inhibition-Naming and DKEFS Color Word Interference Color Naming trials³Composite score of NEPSY-II Inhibition-Inhibition and DKEFS Color Word Interference Inhibition trials⁴Composite score of WISC-IV/5 Coding and WAIS-IV Coding subtests⁵As compared to the normative sample^aStandard Score; Mean = 100, Standard Deviation = 15^bScaled Score; Mean = 10, Standard Deviation = 3^cPercentage of individuals with scores 1.5 Standard Deviation from the mean

Table 3.

Multiple regression analyses predicting processing speed performance-based measures.

Measure	Predictor	β	p	R ² (block)	Total Model R ²
Graphomotor Speed	Verbal Ability	0.691	0.004	0.146	0.146
	Inattention Symptoms	0.071	0.073		
	SCT:	0.366	0.069		
	<i>Sleepy/Sluggish</i>	-0.174	0.346		
	<i>Daydreamy</i>	0.007	0.970		
	<i>Low Initiation</i>	-0.291	0.325		
Speeded Naming	Verbal Ability	0.009	0.251	0.422	0.422
	Inattention Symptoms	0.130	0.073		
	SCT:	0.360	0.098		
	<i>Sleepy/Sluggish</i>	-0.231	0.282		
	<i>Daydreamy</i>	0.222	0.484		
	<i>Low Initiation</i>	-0.488	0.181		
Inhibition	Verbal Ability	0.012	0.234	0.517	0.517
	Inattention Symptoms	0.772	0.003		
	SCT:	0.025	0.280		
	<i>Sleepy/Sluggish</i>	-0.160	0.411		
	<i>Daydreamy</i>	-0.698	0.023		
	<i>Low Initiation</i>	0.435	0.191		

Table 4.

Correlations between SCT subscales and medical, demographic, and neuropsychological factors.

	SCT Sleepy/Sluggish	SCT Daydreamy	SCT Low Initiation	SCT Total
Age at Diagnosis	0.05	−0.33 *	−0.30 *	−0.23
Age at Assessment	0.23	−0.17	−0.00	0.04
Time Since Diagnosis	0.18	.20	0.39 **	.34 **
Gender	0.03	0.07	0.04	0.05
Tumor Location	0.18	−0.02	0.09	0.12
Resection	0.12	0.11	0.05	0.10
Radiation	0.23	−0.07	−0.10	0.02
Chemotherapy	−0.01	−0.11	0.17	0.06
Verbal Ability	−0.05	−0.31 *	−0.30 *	−0.26 *
ADHD-I ^a	0.49 ***	0.65 ***	0.85 ***	0.83 ***

^aADHD Inattentive Type*
p < .05**
p < .01***
p < .001