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Personality Change due to Traumatic Brain Injury in Children and Adolescents: Neurocognitive Correlates

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Abstract

Personality Change due to traumatic brain injury (PC) in children is an important psychiatric complication of injury and is a form of severe affective dysregulation. The aim of the study was to examine neurocognitive correlates of PC. The sample included children (n=177) aged 5-14 years with traumatic brain injury from consecutive admissions to 5 trauma centers were followed prospectively at baseline and 6 months with semi-structured psychiatric interviews. Injury severity, socioeconomic status, and neurocognitive function (measures of attention, processing speed, verbal memory, IQ, verbal working memory, executive function, naming/reading, expressive language, motor speed, and motor inhibition) were assessed with standardized instruments. Unremitted PC was present in 26/141 (18%) participants assessed at 6 months post-injury. Attention, processing speed, verbal memory, IQ, and executive function, were significantly associated ($p < .05$) with PC even after socioeconomic status, injury severity, and pre-injury

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attention-deficit/hyperactivity disorder were controlled. These findings are a first step in characterizing concomitant cognitive impairments associated with PC. The results have implications beyond brain injury to potentially elucidate the neurocognitive symptom complex associated with mood instability regardless of etiology.

Keywords

Traumatic brain injury; children and adolescents; cognition; personality change

Introduction

Personality change due to traumatic brain injury (PC) is the most common and important acquired psychiatric disorder following brain trauma in children and adolescents [1-5]. We have previously described the phenomenology of PC in detail, however the neurocognitive correlates of PC have not been studied [5]. The rationale for investigating the neurocognitive profile of PC is that this may shed light on the neurobiological systems or networks that are perturbed, and provide a link between neurobiology and observed behaviors [6,7].

PC has five specific subtypes including affectively labile, aggressive, disinhibited, apathetic, and paranoid. The first three subtypes are common and often co-occur while the latter two subtypes are uncommon [5]. Studies show that most, if not all, cases of PC are accounted for by having at least the affectively labile subtype with impairing irritability [3]. The irritability has numerous potential antecedents including a frustrated need for constant attention, sensitivity to criticism, concrete thinking, delay in gratification, unpredictability or change in routine, intellectual or concentration deficits increasing the effort in task completion, communication difficulties resulting in misunderstanding humor and instructions, and sensitivity to pain or accidental mild injury [5]. The course of PC is continuous rather than episodic. The temper tantrums or rages that are associated with PC may be interspersed with euthymia or with persistent irritability varying case by case. PC manifests as a disorder of mood regulation with associated behavioral disruption. Consequently, PC rarely occurs alone as a new-onset psychiatric disorder after TBI. The comorbid new-onset disorders include anxiety disorders [8], depressive disorders [9], mania/hypomania [10], ADHD [2], and oppositional defiant disorder [2]. There are a number of misperceptions regarding the diagnosis of PC. Most importantly, PC is not a personality disorder. Rather PC is a collection of clinically significant symptoms described by its phenomenologically evocative named subtypes. Children, adolescents, and adults who develop PC are so impaired and radically altered as to be considered to have had a change in their personality.

PC occurs in up to 40% of cases of consecutively hospitalized children with severe traumatic brain injury (TBI) [2]. The disorder is significantly associated with measures of severity of injury rather than psychosocial variables [2-4]. PC is associated with lesions within the superior frontal gyrus within the first year post-injury [3,4] consistent with proposed models of affective dysregulation [11-13]. In the second year after injury, PC is associated with frontal white matter lesions and pre-injury adaptive function suggesting that function is

limited by network (rather than gray matter) damage as well as pre-injury brain-behavioral reserve within each individual [4].

In the absence of studies of the neurocognitive correlates of PC, our approach was to examine the relationship of PC with neurocognitive domains that are each known to be sensitive to the effects of TBI. These domains include attention, processing speed, verbal memory, intellectual function, working memory, executive function, naming, expressive language, and motor speed [14]. Pediatric TBI studies generally have found that motor inhibition measured by the Stop Signal Task is not significantly related to brain injury and only even inconsistently related to the diagnosis of post-injury new-onset (secondary) ADHD [15]. We therefore did not expect PC to be related to motor inhibition even though emotional and/or behavioral inhibition is present in PC.

Methods

The methodology of this study has been reported in detail previously [3].

Participants

Children and adolescents (n=177) aged 5-14 years were enrolled from consecutively hospitalized patients after a single TBI at five academic medical centers including three in Texas, one in San Diego, and one in Toronto. Enrollment ranged from severe to mild TBI at every center except San Diego, where recruitment was limited to severe to complicated mild TBI. Youth with pre-injury schizophrenia, autistic disorder, mental deficiency, and injury from child abuse or penetrating bullet injury were excluded. Children and adolescents with pre-injury ADHD were excluded in San Diego. Parents or guardians of all children and adolescents signed informed consent, and all children and adolescents signed an assent to participate in accordance with each site's Institutional Review Board. Demographic and injury indices are shown for participants in Table 1.

Psychiatric measures—The Neuropsychiatric Rating Schedule (NPRS) [1] is a semi-structured interview to identify symptoms and subtypes of PC. Parents and children served as informants in the interview that took place at baseline (within a month post-injury), and at 6 months post-injury. At baseline, the lifetime pre-injury psychiatric history was the focus of inquiry, and at the 6-month assessment the focus was the period from injury to the 6 months post-injury. The NPRS interview generated ratings defining the five major subtypes of PC. We waived the one-year duration of symptomatology criterion to allow us to monitor the course of the disorder for the first six months after injury and to assess the neurocognitive correlates of PC. The NPRS has been shown to provide reliable and valid diagnoses of the common subtypes of PC [1]. Good convergent validity and good discriminant validity has been demonstrated for PC subtypes by using subscales from validated parent and teacher-completed questionnaires that measure lability, aggression, impaired social judgment/ disinhibition, apathy, and psychotic symptoms. Inter-rater agreement for NPRS items is fair to excellent, test-retest reliability is fair to good, and sensitivity to change has been demonstrated [1].

Other DSM-IV psychiatric diagnoses [16] were derived by using the Schedule for Affective Disorders and Schizophrenia for School-Aged Children, Present and Lifetime version (K-SADS-PL) [17]. The K-SADS-PL is a parent-child interview that generates diagnoses based on a clinician synthesizing data collected from parent and child separately, reviewing present and lifetime symptoms (at baseline) and symptoms present or past from injury to 6 months (at 6 month assessment).

Best-estimate psychiatric diagnoses [18] were generated by the interviewer after integrating the accounts of the parent and the subject from the NPRS and the K-SADS interviews and, when available, from the Survey Diagnostic Instrument [19] completed by the teacher.

TBI Classification—The classification of severity of TBI was based on the lowest post-resuscitation score on the Glasgow Coma Scale (GCS) [20] which was recorded from clinical notes. The GCS is the standard measure of severity of acute brain injury associated with TBI. The scale measures eye opening, motor, and verbal responsiveness. Scores range from 3 (unresponsive) to 15 (normal).

Socioeconomic Status (SES)—SES assessment was derived from the Four Factor Index [21]. Classification depends on scores generated from a formula involving both the maternal and paternal occupational and educational levels. Scores range from 8-66 with lower scores indicating lower educational and occupational levels and lower SES.

Neurocognitive Measures

Attention: Attentional processes in single and dual task performance were measured with the Divided Attention Task (DAT) [22]. The DAT assesses the ability to allocate attentional resources when simultaneously engaged in performing two independent tasks. Timed comparisons were evaluated for performing the single task of finger tapping versus simultaneously performing the dual tasks of right finger tapping and reciting a nursery rhyme. The outcome variable is number of words correct.

Processing Speed: Symbol Search and Coding subtests of the Wechsler Intelligence Scale for Children –III (WISC-III) [23] measure visual scanning, tracking ability, and psychomotor processing speed. These tasks require interpreting whether a target symbol appears in a row of symbols, and digit-symbol codes. Correct responses less the number of errors completed within the allotted 120 seconds for each subtest was noted. Together, these measures provide a Processing Speed Index.

Verbal Memory: The California Verbal Learning Test-Children's Version [24] (CVLT-C) was administered and assesses verbal learning and memory abilities. Children were instructed to learn 15 words in 3 categories across 5 learning trials and 1 distraction trial. Verbal memory for long delay free recall was assessed and expressed as a z-score.

Intellectual Function: The Wechsler Abbreviated Scale of Intelligence (WASI) [25] was administered to all children at 6 months post-injury. The WASI is a test of intelligence that consists of the following four subtests: Vocabulary, Similarities, Block Design, and Matrix reasoning. These four components evaluate a person's verbal and non-verbal knowledge and

reasoning, and general cognitive functioning, and together, produce a full-scale intelligence quotient (FSIQ) which was the variable reported here as a standard score.

Verbal Working Memory: Working memory was evaluated with a computerized N-back experimental task [26]. The Letter Identity task has 3 levels of memory load:1-back, 2-back, and 3-back. As well, there is a 0-back condition that imposes minimal memory load while controlling for attention to the task. Children were instructed to match the same alphabetic letters printed in different cases. For each level, there were 40 trials in which a string of 40 letters appeared 1 at a time for 2 seconds on the screen. The child responded by pressing a button with the preferred hand when a match occurred or, in the 0-load condition, when a designated target appeared. Training and practice occurred before experimental trials. The percentage of hits (i.e., detection of targets) was recorded.

Executive Function: A modified version of the Tower of London (TOL) [27] was used to assess planning skills that involve the ability to look ahead, follow rules, conceive of alternative solutions to the problem, and to weigh and make choices. The test required that children arrive at the most direct, fewest move solution by determining the order of moves necessary within set rules (e.g., “pick up one bead at a time”) to rearrange three colored beads on pegs of three disks of differing heights. We report data of total solution time, i.e., the time, measured in seconds, taken to come to the final solution for that trial.

Naming: The Rapid Automatized Naming task [28] was administered by asking the participant to rapidly name line drawings of five common objects which are reproduced ten times each and interspersed on a board. This task is related to processing speed and reading. The time required to complete the task was measured and expressed as a z-score.

Expressive Language: Expressive language was assessed with the Clinical Evaluation of Language Fundamentals – Third Edition (CELF-3) [29] Formulated Sentence Subtest consisting of 22 items. Children were shown an image with a target word/phrase and they were instructed to construct a sentence in response. Standard scores were recorded and analyzed.

Motor Speed: The Grooved Pegboard Test (GPT) assesses fine motor coordination and manual dexterity [30]. Participants are instructed to insert 25 grooved pegs into a pegboard using one hand at a time as quickly as possible. Time to completion expressed as a z-score for the dominant hand was recorded and used in the analysis.

Motor Inhibition: The Stop Signal Task (SST) assesses ability to inhibit ongoing response in a choice reaction time task [31]. The stop signal reaction time will be used in the analyses. A prolonged stop signal reaction time has been identified as one of the signature executive deficits associated with ADHD.

Data Analysis

Effect sizes were used to compare neurocognitive scores between the groups with PC versus without PC. However, because these scores could be influenced by severity of injury [14],

socioeconomic status [32], and pre-injury ADHD [6], linear regressions were conducted for each neurocognitive measure of interest controlling for these variables. Alpha was kept at .05 because each of the regressions tested an independent hypothesized relationship between the neurocognitive measure and PC guided by previous findings that each neurocognitive domain is sensitive to the effects of TBI [14].

Results

Of the original 177 children and adolescents, 141 (80%) returned for the 6-month psychiatric assessment. The returning participants were not significantly different from those who did not return with regard to age, race, gender, GCS score, or SES. PC occurred in 31/141 (22%) at some point from injury to the 6-month assessment. However, in 5 cases the symptoms of PC remitted before the 6-month assessment, yielding 26/141 (18%) of unremitted cases of PC. The affectively labile subtype occurred in 23 of the 26 cases. Of the 3 cases of PC that did not meet criteria for the affectively labile subtype, 2 were diagnosed with the aggressive subtype of PC, and 1 was diagnosed with the disinhibited subtype. A detailed list including each child diagnosed with PC along with their PC subtypes and specific brain lesions was previously published [3]. As in previous reports, comordity is common [2]. Unremitted PC was significantly associated with the following unremitted new onset disorders: 1) new-onset ADHD was present in 6/19 children with PC versus 11/96 children with no PC; Fisher's Exact = .035; and 2) new-onset oppositional defiant disorder/ conduct disorder/disruptive behavior disorder, not otherwise specified (ODD/CD/DBD) was present in 7/24 children with PC versus 4/110 children with no PC; Fisher's Exact = .001. PC was not significantly associated with new-onset unremitted depressive disorder (major depression/dysthymic disorder/depressive disorder, not otherwise specified) which was present in only 6 children including 3/25 children with PC versus 3/113 with no PC; Fisher's Exact = .073. The denominators differ for the above disorders depending on pre-injury diagnoses, e.g., children with pre-injury ADHD would not be eligible to develop new-onset ADHD.

Relationship of Neurocognitive Measures to PC

Tables 2 and 3 provide details of the relation of each neurocognitive domain and PC. Table 2 demonstrates large effect sizes for processing speed (*d*=.89) and for full-scale IQ (*d*=1.14), moderate effect sizes for verbal memory (*d*=.66), verbal working memory (*d*=.73), expressive language (*d*=.73), executive function (*d*=.68), attention (*d*=.54), small effect size for naming (*d*=.36), and trivial effect sizes for motor speed (*d*=.13) and motor inhibition (*d*=17).

Table 3 illustrates in detail the linear regression analyses of the 10 neurocognitive domains and their respective relationships with PC, controlling for injury severity as measured by the GCS score, SES, and pre-injury ADHD (present versus absent). PC was independently related to full-scale IQ (p=.04), divided attention (p=.02), processing speed (p=.013), verbal memory (p=.013), verbal working memory (p=.017), and executive function (p=.012) and not significantly related to naming, expressive language, motor speed, or motor inhibition. Consistent with extensive literature, naming/reading was significantly related to SES [33].

Expressive language was significantly related to injury severity and SES. Motor speed was significantly related to injury severity. Motor inhibition was related to ADHD at a trend level.

All statistical analyses reported in Tables 2 and 3 were repeated comparing only the participants with the affective lability subtype of PC (n=23) versus all other participants. Results were essentially unchanged.

Discussion

The most important finding from this investigation is that PC was significantly associated with deficits in important neuropsychological domains including intellectual function, processing speed, divided attention, verbal memory, working memory, expressive language, and executive function. Furthermore, these associations (except for expressive language) remained significant even when severity of brain injury, SES, and pre-injury ADHD were taken into account.

The relationship between PC and neurocognitive function is clearly not uniform as evidenced by effect sizes ranging from large to small. This is not surprising given previous brain lesion-behavior correlates implicating the dorsal frontal area especially the superior frontal gyrus in PC. The weak relationship between PC and reading and expressive language may be because these neurocognitive domains are relatively crystallized and more closely related to SES than to a specific pattern of brain damage [33]. Neurocognitive processes including executive function, verbal memory, working memory, and attention, which are mediated by frontal networks [14] might be expected to be deficient in children with disrupted affective regulation caused by damage to similar or overlapping neuronal networks. Our negative Stop Signal Task findings suggest, as anticipated, that motor inhibition is distinct from the disinhibition or overreactivity of emotional response characteristic of PC.

Individuals with TBI are known to have difficulty understanding negative emotions such as anger, sadness, and fearfulness compared to positive emotions such as happiness [34,35], and this may be associated with marked increased reactivity to negative emotional stimuli manifested verbally or behaviorally in children with PC. Further, children with TBI exhibit impairments in understanding a form of affect regulation involving social suppression of emotional expressions [34,36]. Ecologically, better understanding of affect regulation predicts less rejection-victimization in the classroom [37].

The relationship between PC and the neuropsychological domains studied is strikingly similar to the corresponding relationship previously reported for another condition characterized by significant affective dysregulation, namely bipolar disorder [6]. For example, the respective effect sizes for bipolar disorder and PC and the specific neuropsychological domain are verbal memory (d=0.77 versus d=.66), attention (d=0.62 versus d=.54), executive function (d=0.60 versus d=.68), working memory (d=.60 versus d=.73), naming/reading (d=0.40 versus d=.36), motor speed (d=0.33 versus d=.13). The corresponding relationship bipolar disorder and PC with full-scale IQ (d=0.32 versus d=1.14) was notably different, a finding driven most likely due to the association of IQ and PC with greater

severity of injury [2]. Another less striking difference from a recent study suggested a significant relationship with a small effect size between bipolar disorder and motor inhibition on the Stop Signal Task in contrast to the trivial relationship in the current study [38].

It is difficult to outline a clear mechanism whereby affective dysregulation, which is the core feature of PC (and an important feature of bipolar disorder), is related to the neurocognitive findings. One possibility is that the pattern of brain network damage leads independently to both PC and neurocognitive dysfunction. Clinically, this seems likely because affective dysregulation and neurocognitive problems are evident within the first few days of brain injury in children. Another possibility is that regulation of affect is modified by multiple neurocognitive processes. For example, an individual with slow processing, problematic divided attention, and poor memory may become overwhelmed and frustrated by environmental and interpersonal stimuli. Deficits in their working memory, planning and problem solving ability may lead to selection of more angry or aggressive responses because of difficulties working adaptively with new challenges. A third possibility, although much less likely, is that the affective dysregulation leads to the array of neurocognitive problems. This is unlikely because the child's explosive irritability, while frequent, is not constant and generally not present during the formal neurocognitive testing.

Our findings must be appreciated within the context of limitations of this study. First, the neurocognitive measures administered in the study included a broad array of domains known to be sensitive to brain injury. However, more specific neurocognitive measures, and psychophysiological and functional brain imaging modalities targeting recognition and understanding of negative emotion, suppression of emotional expression, and executive function with emotional distractors were not used. This limited the potential for a more in-depth understanding of mechanisms underlying the expression of PC. Second, a continuous measure of affective lability might shed more light on the relationship with neurocognitive domains. Third, attrition was approximately 20%. However, participants were not significantly different to non-participants with respect to age, race, gender, injury severity, and SES. Fourth, this study examined only short term (6 month) outcome of PC and its relationship with neurocognitive measures. We intend to examine whether the relationships reported here are sustained with regard to PC persisting to 12 and 24 months post-injury in the same pediatric TBI cohort.

Strengths of the study should also be appreciated. The cohort studied is a large sample of non-referred consecutively hospitalized children and adolescents with semi-structured psychiatric interviews and standardized neuropsychological tests encompassing multiple domains of function. Data reported in this study extend previously published findings that focused on psychosocial and lesion correlates of PC in the same cohort. The investigation of the neuropsychology of PC is a unique aspect of this study.

Conclusions and Implications

Ultimately the purpose of understanding the neural and psychological mechanisms inherent in children and adolescents with PC is to develop treatment strategies. In theory, stimulation

of brain networks whose function is to regulate the expression of affect (e.g., dorsal frontal areas) or partial inhibition of networks responsible for generating affect (e.g., ventral frontal area, amygdala) could be helpful. Clinical trials of mood stabilizers are very difficult to accomplish, especially for children with PC, but should be done. Cognitive rehabilitation targeting processing speed, attention, problem solving, and memory may enhance cognitive control (a form of executive function) over emotional expression and should be tested [39]. It is conceivable that more specific emotional probes such as utilized in social cognition and affect recognition studies will further clarify important mechanisms in children with PC and possibly other disorders of affective dysregulation [34,36,40-42].

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Table 1

Demographic and Psychosocial data of traumatic brain injury cohort (n=177)

Demographic Variables		**N**
Age at injury mean (SD)	10.13 (2.77)	177
Gender: males (%)	125 (71%)	177
Socioeconomic Status mean (SD)	37.01 (12.90)	173
Race		
Caucasian	100 (56.5%)	
Hispanic	32 (18.1%)	
African American	31 (17.5%)	
Asian	5 (2.8%)	
Other	9 (5.1%)	
Psychosocial Variables		
Preinjury lifetime psychiatric disorder number (%)	56 (31.6%)	177
Injury Variables		
Lowest post-resuscitation GCS Score mean (SD)	10.85 (4.20)	177
Severe Traumatic Brain Injury (GCS 3-8)	64 (36%)	
Moderate Traumatic Brain Injury (GCS 9-12)	26 (15%)	
Mild Traumatic Brain Injury (GCS 13-15)	87 (49%)	
Depressed skull fracture (N; %)	17 (9.6)	177
Mechanism of Injury	*N (%)*	*177*
Hit by motor vehicle	49 (27.7)	
Fall	41 (23.2)	
Auto, truck, bus passenger	40 (22.6)	
Sports or play	15 (8.5)	
Recreational vehicle/Off-road vehicle	10 (5.6)	
Bicycle	9 (5.1)	
Motorcycle-moped	5 (2.8)	
Hit by a falling object	5 (2.8)	
Other	3 (1.7)	

Table 2

Attention, Processing, Verbal Memory, Intellectual Function, Working Memory, Executive Function, Naming, Expressive Language, and Motor Speed correlates of PC 6 months post-iniury

	PC (n=26)	No PC (n=115)	t	df	sig	Cohen's d
Attention						
Divided Attention rhyme/right tapping	72.0 (26.0) n=25	85.4 (23.3) n=105	2.53	128	.013	.54
Processing Speed						
WISC-3 Processing Speed Index scale score	93.1 (19.6) n=24	109.9 (18.0) n=110	4.06	132	.000	.89
Verbal Memory						
CVLT-C long delay free recall (z-score)	−.31 (1.43)	.52 (1.07) n=114	2.77	31.7	.009	.78
Intellectual Function						
WASI Full Scale IQ standard score	87.4 (8.8) n=21	101.8 (15.6) n=105	5.88	49.1	.000	1.14
Working Memory N-Back task						
Letter Identity target detection	.55 (.22) n=24	.70 (.19) n=106	3.22	128	.002	.73
Executive Function						
Tower of London Total solution time (sec)	260.7 (71.8)	341.7 (152.7) n=114	4.04	83.6	.000	.68
Naming						
Rapid Automatized Naming (z-score)	−.28 (2.07)	.34 (1.27)	1.47	29.4	ns	.36
Expressive Language						
CELF-3 Formulated Sentences standard score	7.9 (2.3) n=24	9.9 (3.1) n=111	3.60	43.9	.001	.73
Motor Speed						
Grooved Pegboard dominant hand time (z-score)	.18 (1.18)	.30 (.64) n=112	.70	136	ns	.13
Motor Inhibition						
Stop Signal Reaction time (milliseconds)	460.8 (244.8) n=16	420.2 (226.0) n=83	−.47	97	ns	.17

Legend: CVLT-C = California Verbal Learning Test – Children's Version; CELF = Clinical Evaluation of Language Fundamentals; PC = Personality change due to traumatic brain injury; WASI = Wechsler Abbreviated Scale of Intelligence; WISC = Wechsler Intelligence Scale for Children

Table 3

Linear Regression Analyses for PC and Neurocognitive Outcomes

Dependent Variables	Independent Variables	R^2	df	F	Sig of F (p)	B	Beta	t statistic	p
Attention		.09	4, 124	3.18	.016				
	PC					−13.25	−.26	−2.36	**.020**
	GCS					−.17	−.03	−.30	.762
	SES					.33	.17	1.93	.056
	ADHD					9.46	.15	1.67	.097
Processing Speed		.22	4, 128	8.87	.000				
	PC					−10.62	−.21	−2.51	**.013**
	GCS					1.22	.26	3.05	.003
	SES					.18	.12	1.45	.150
	ADHD					−9.47	−.19	−2.35	.020
Verbal Memory		.16	4, 133	7.63	.000				
	PC					−.64	−.21	−2.53	**.013**
	GCS					.04	.16	1.82	.072
	SES					.02	.22	2.66	.009
	ADHD					.11	.04	.43	.669
Intellectual Function		.42	4, 119	21.52	.000				
	PC					−6.56	−.16	−2.08	**.040**
	GCS					1.24	.34	4.33	.000
	SES					.50	.41	5.70	.000
	ADHD					−2.67	−.06	−.89	.378
Working Memory		.16	4, 124	5.91	.000				
	PC					.51	−.22	−2.43	**.017**
	GCS					0.00	.01	.15	.881
	SES					0.00	.29	3.40	.001
	ADHD					−.02	−.04	−.42	.673
Executive Function		.10	4, 134	3.61	.008				
	PC					−81.59	−.22	−2.55	**.012**
	GCS					−1.16	−.03	−.38	.705
	SES					−1.02	−.09	−1.05	.295
	ADHD					−74.56	−.20	−2.37	.019
Naming		.09	4, 134	3.11	.017				
	PC					−.40	−.11	−1.23	.220
	GCS					.03	.08	.94	.350
	SES					.02	.21	2.43	.017
	ADHD					−.25	−.07	−.79	.434
Expressive Language		.23	4, 128	9.50	.000				

Dependent Variables	Independent Variables	R^2	df	F	Sig of F (p)	B	Beta	t statistic	p
	PC					−1.07	−.13	−1.60	.113
	GCS					.16	.21	2.47	.015
	SES					.08	.32	4.02	.000
	ADHD					−.56	−.07	−.88	.382
Motor Speed		.08	4, 131	2.91	.024				
	PC					.06	.03	.37	.710
	GCS					.05	.29	3.21	.002
	SES					.00	.03	.33	.742
	ADHD					−.11	−.06	−.66	.510
Motor Inhibition		.00	4, 93	.97	.430				
	PC					15.97	.03	.24	.812
	GCS					−4.96	−.09	−.77	.445
	SES					1.35	.07	.66	.508
	ADHD					97.73	.18	1.73	.088

Legend: The specific tests for each domain of neurocognitive function correspond to those listed in Table 2. Bold face type indicates where PC is independently significantly related to the neurocognitive domain.