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# Profiles of Executive Function Across Children with Distinct Brain Disorders: Traumatic Brain Injury, Stroke, and Brain Tumor

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**Abstract**

**Objectives:** This study examined whether children with distinct brain disorders show different profiles of strengths and weaknesses in executive functions, and differ from children without brain disorder. **Methods:** Participants were children with traumatic brain injury ($N = 82$; 8–13 years of age), arterial ischemic stroke ($N = 36$; 6–16 years of age), and brain tumor ($N = 74$; 9–18 years of age), each with a corresponding matched comparison group consisting of children with orthopedic injury ($N = 61$), asthma ($N = 15$), and classmates without medical illness ($N = 68$), respectively. Shifting, inhibition, and working memory were assessed, respectively, using three Test of Everyday Attention: Children's Version (TEA-Ch) subtests: Creature Counting, Walk-Don't-Walk, and Code Transmission. Comparison groups did not differ in TEA-Ch performance and were merged into a single control group. Profile analysis was used to examine group differences in TEA-Ch subtest scaled scores after controlling for maternal education and age. **Results:** As a whole, children with brain disorder performed more poorly than controls on measures of executive function. Relative to controls, the three brain injury groups showed significantly different profiles of executive functions. Importantly, post hoc tests revealed that performance on TEA-Ch subtests differed among the brain disorder groups. **Conclusions:** Results suggest that different childhood brain disorders result in distinct patterns of executive function deficits that differ from children without brain disorder. Implications for clinical practice and future research are discussed. (*JINS*, 2017, *23*, 529–538)

**Keywords:** Executive functions, Shifting, Inhibition, Working memory, Pediatric brain disorders, TEA-Ch

## INTRODUCTION

Executive functions refer to complex mental processes that orchestrate purposeful, goal-directed activity. These higher order processes are important for adaptive functioning because they allow for the modification of action, inhibition of inappropriate or task-irrelevant activity, and guidance of behavior in accordance with rules, internal goals, and intentions (Jurado & Rosselli, 2007; Miller & Cohen, 2001). Traditionally, executive functions have been thought to rely primarily on frontal brain regions (Stuss & Levine, 2002). However, more recent research suggests that they are subserved by a more distributed cortical network, including

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subcortical and parietal brain systems (Lewis, Dove, Robbins, Barker, & Owen, 2004).

Research has consistently suggested that multiple latent cognitive factors account for performance on executive tasks (see Jurado & Rosselli, 2007, for a review). In one influential study, Miyake and colleagues (2000) concluded that executive functions comprise three separate but interrelated factors (i.e., shifting, working memory updating, and inhibitory control), and that each factor contributes differentially to performance on executive tasks. Other proposed executive subcomponents have included selective attention (Fournier-Vicente, Larigauderie, & Gaonac'h, 2008), planning (Hobson & Leeds, 2001; Lezak, 1983), initiation/volition (Hobson & Leeds, 2001; Lezak 1983), and verbal fluency (Fisk & Sharp, 2004).

Not surprisingly, many children with developmental and acquired brain abnormalities display executive dysfunction (for reviews, see Barkely, 1997; Brocki, Fan, & Fossella, 2008; Conklin et al., 2012, 2013; de Ruiter et al., 2013; Konrad, Gauggel, Manz, & Scholl, 2000; Leblanc et al., 2005; Levin & Hanten, 2005; Mangeot, Armstrong, Colvin, Yeates, & Taylor, 2002; McDonald, Flashman, & Saykin, 2002). At the same time, a comparison of studies of individual disorders also suggests that children with brain disorders may exhibit distinct patterns of strengths and weaknesses across executive tasks (Anderson, Fenwick, Manly, & Robertson, 1998; Ewing-Cobbs, Prasad, Landry, Kramer, & DeLeon, 2004; Golberg et al., 2005; Mahone, Koth, Cutting, Singer, & Denckla, 2001). Consistent with factor analytic studies, these studies suggest that executive subcomponents are dissociable in children (see Brocki & Bohlin, 2004).

To our knowledge, however, only a handful of studies have directly compared executive function profiles across different developmental or acquired childhood brain disorders. Additionally, in the studies to date, different methods have been used to assess executive functions. For example, some research has directly compared executive abilities across groups using certain standardized tests, whereas other investigations have relied solely on parent behavior ratings for comparison. Furthermore, the domains of executive function measured often vary widely across studies.

With that said, the limited findings suggest that children with different developmental and acquired brain disorders [e.g., attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder, Tourette syndrome, traumatic brain injury (TBI)] show different patterns of executive function and differ from typically developing children. For example, research examining inhibitory control in children with ADHD and TBI has shown that both groups show inhibitory control deficits; however, children with TBI have also been found to exhibit generalized slowing in their information processing speed that is unrelated to their disinhibition (Konrad et al., 2000).

Similar investigations comparing profiles of executive function among children with ADHD and autism spectrum disorder have shown that, although both groups of children display executive function deficits, the deficits tend to be more generalized and severe in autism spectrum disorder than in ADHD (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Pennington & Ozonoff, 1996). Additionally, children with ADHD often display more selective difficulty in inhibitory control, while children with autism spectrum disorder tend to show deficits in working memory, monitoring, and cognitive flexibility (Gioia, Isquith, Kenworthy, & Barton, 2002; Happé, Booth, Charlton, & Hughes, 2006; Pennington & Ozonoff, 1996; Verte, Geurtas, Roeyers, Oosterlaan, & Sergeant, 2006).

Taken together, these findings provide evidence of distinct profiles of executive function across clinical groups. They also lend support to the notion that, while children with different developmental or acquired brain disorders may have executive dysfunction in common, they nonetheless can be distinguished from each other by the severity and/or pattern of their executive function deficits (Pennington & Ozonoff, 1996).

Several studies have examined correlates of individual differences in executive functions to determine which factors influence executive outcomes most within specific clinical groups. Examples of these correlates include disorder-specific symptomatology (e.g., associations between inhibitory control deficits and symptoms of ADHD; Happé et al., 2006; Verte et al., 2006), age at injury or treatment (Jacobs, Harvey, & Anderson, 2007), total number of lesions, and extrafrontal lesion volume (Power, Catroppa, Coleman, Ditchfield, & Anderson, 2007; Slomine et al., 2002). Westmacott, Askalan, Macgregor, Anderson, and Deveber (2010) also proposed an interactive relationship between lesion location and age at injury on executive function outcomes in children with stroke.

In the current study, we sought to examine whether children with three different childhood brain disorders [i.e., TBI, arterial ischemic stroke (AIS), brain tumor (BT)] show distinct profiles of strengths and weaknesses in executive functions as defined in Miyake's model (i.e., shifting, inhibitory control, working memory). Children with these three disorders have been shown to display deficits in executive functions, including those functions identified in Miyake's model (Brocki et al., 2008; Conklin et al., 2012, 2013; de Ruiter et al., 2013; Konrad et al., 2000; Law et al., 2011; Leblanc et al., 2005; Levin & Hanten, 2005; Mangeot et al., 2002; McDonald et al., 2002); however, to our knowledge, the groups have never been directly compared. We used data from three separate cross-sectional cohort studies, which were developed primarily to investigate relationships among executive functions and social outcomes within each group. Nonetheless, similar tests were intentionally chosen across the studies to allow for comparisons across groups, thus providing an opportunity to address the question of differences in executive function profiles. We hypothesized that the three clinical groups (i.e., TBI, AIS, BT) would show significantly different profiles of executive functions, as well as differ from children without brain disorder. As a secondary aim, we examined the relationship of pathology-related characteristics to executive functioning within each clinical group.

## METHOD

### Participants

Data for this study were drawn from three separate cross-sectional cohort studies examining cognitive and psychosocial outcomes following TBI, AIS, and BT. Participants consisted of 82 children with TBI, 36 children with AIS, and 75 children with BT, each with a corresponding comparison group comprised of children with orthopedic injury ($n = 61$), asthma ($n = 15$), or classmates without medical illness ($n = 68$), respectively. See Yeates et al. (2014), Hajek et al. (2014), and Salley et al. (2015) for more detailed descriptions of the TBI, AIS, and BT participants, respectively.

Briefly, children in the TBI group were 8–13 years of age, with injuries ranging in severity from complicated mild to severe, all resulting in hospitalization; Glasgow Coma Scale (GCS; Teasdale & Jennet, 1974) scores ranged from 3 to 15 ($M = 10.8$; $SD = 4.9$). Twenty-five children (31%) had severe injuries, 13 (16%) had moderate injuries, and 44 (54%) had complicated mild injuries.

Children in the AIS group were 6–16 years of age. Ten children (28%) had perinatal strokes (i.e., within the first 30 days of life) and 26 (72%) had childhood strokes (i.e., after 1 month of age). The strokes were unilateral in 29 cases (44% left hemisphere, 36% right hemisphere), bilateral in 4 cases (11%), and involved the brainstem or cerebellum in 3 cases (8%). The strokes were restricted to cortical regions in 2 cases (6%) and to subcortical regions in 13 cases (36%), and encompassed both cortical and subcortical regions in 18 cases (50%), with 3 cases (8%) involving the brainstem or cerebellum.

Children in the BT group were 9–18 years of age. Eighteen participants (24%) had supratentorial tumors, 17 (23%) had midline tumors, and 32 (43%) had infratentorial tumors. Tumor location was unknown for 7 participants (10%). See Table 1 for a summary of different tumor types for this group. Sixty-five children (88%) were treated with surgical resection, of which 32 (43%) were treated with resection only. Forty-one children (55%) received some form of adjuvant treatment; 10 (14%) were treated with chemotherapy only, 9 (12%) were treated with radiation only, and 22 (30%) received both chemotherapy and radiation. One child (1%) was treated with adjuvants only, and one (1%) did not receive any form of treatment.

The control group was comprised of three separate groups of children that were recruited as comparison samples for each of the three brain disorder groups. Comparison groups were comparable to the brain disorder groups on age, sex, and education. The three comparison groups did not differ in executive function performance [Wilks' $\lambda = .96$, $F(6,268) = .44$, partial eta$^2 = .02$] and were, therefore, merged into a single control group of 144 children without brain disorder. See Table 2 for a summary of basic demographic information for each of the four groups (i.e., TBI, AIS, BT, and controls).

**Table 1**. Summary of tumor type, location, and treatment for brain tumor group

| | *N* | % |
|---|---|---|
| Tumor type | | |
| Low grade glioma/astrocytoma | 35 | 47.3 |
| PNET | 17 | 23.0 |
| Craniopharyngioma | 5 | 6.8 |
| Ependymoma | 5 | 6.8 |
| Germ cell | 4 | 5.4 |
| High grade glioma/astrocytoma | 2 | 2.7 |
| Other | 6 | 8.1 |
| Tumor location | | |
| Infratentorial | 32 | 43.2 |
| Supratentorial | 18 | 24.3 |
| Midline | 17 | 23.0 |
| Missing | 7 | 9.5 |
| Adjuvant therapy | | |
| Resection only (no adjuvant therapy) | 32 | 43 |
| Chemotherapy only | 10 | 14 |
| Radiation only | 9 | 12 |
| Chemotherapy plus radiation | 22 | 30 |
| No Resection or adjuvant therapy | 1 | 1 |

### Procedure

Institutional Review Board/Human Research Ethics Committee approval was obtained for each of the three parent studies. The studies were all cross-sectional in design. Participants in the studies all completed an assessment that included standardized tests of cognitive ability, as described below.

### Measures

General intellectual ability was estimated using the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999; $M = 100$; $SD = 15$). Children with BT and their matched comparison group completed the four-subtest WASI. All other participants completed the two-subtest WASI. Correlations between two-subtest and four-subtest WASI IQ scores range from .91 to .94 in the normative sample of 6- to 18-year-old children (Wechsler, 1999).

Executive functions were assessed using three subtests from the Test of Everyday Attention: Children's Version (TEA-Ch; Manly, Robertson, Anderson, & Nimmo-Smith, 1999; $M = 10$; $SD = 3$). The TEA-Ch has been used to examine executive aspects of attention in different medical and neurodevelopmental disorders, including ADHD (Manly et al., 2001), very preterm children (Bayless & Stevenson, 2007), and TBI (Anderson et al., 1998). In this study, shifting, inhibition, and working memory were assessed using the Creature Counting, Walk-Don't-Walk, and Code Transmission subtests of the TEA-Ch, respectively. We chose these tasks based on face validity and their similarity to other experimental tasks often used to assess these aspects of executive functions (e.g., go/no-go, n-back).

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**Table 2**. Summary of demographic information across the patient and control groups

| | TBI | AIS | BT | Control | Group comparisons |
|---|---|---|---|---|---|
| *N* | 82 | 36 | 73 | 144 | |
| Age at Assessment in Years: mean (*SD*) | 10.36 (1.50) | 9.15 (3.02) | 12.29 (2.41) | 11.30 (2.27) | AIS < TBI < C < BT |
| WASI Full Scale IQ: mean (*SD*) | 99.15 (14.45) | 94.62 (13.92) | 97.49 (16.30) | 106.22 (14.03) | AIS, TBI, BT < C |
| Age at diagnosis in years: mead (*SD*) | 7.83 (1.94) | 4.17 (4.34) | 7.23 (3.54) | – – | AIS < TBI, BT |
| Years since diagnosis: mean (*SD*) | 2.54 (1.22) | 4.97 (3.28) | 5.10 (2.32) | – – | TBI < AIS, BT |
| Number of Males: *n* (%) | 54 (66) | 15 (42) | 43 (58) | 84 (58) | NS |
| Race: *n* (%) | | | | | |
| White | 64 (78) | 34 (94) | 62 (84) | 126 (88) | NS (White vs. Non-White) |
| Non-White | 13 (16) | 2 (6) | 8 (11) | 17 (12) | |
| Missing | 5 (6) | 0 (0) | 4 (5) | 1 (1) | |
| Maternal education: *n* (%) | | | | | |
| Less than high school | 9 (11) | 6 (17) | 2 (3) | 7 (5) | AIS < TBI, BT, C (proportion of mothers who completed post-secondary education) |
| High school graduate | 15 (18) | 11 (31) | 17 (23) | 25 (17) | |
| Some college | 34 (41) | 11 (31) | 27 (37) | 47 (33) | |
| College degree: | 17 (21) | 4 (11) | 13 (18) | 38 (26) | |
| Graduate degree | 7 (9) | 2 (6) | 10 (14) | 26 (18) | |
| Missing | 0 (0) | 2 (6) | 5 (7) | 1 (1) | |

*Note*. AIS = arterial ischemic stroke group; BT = brain tumor group; C = control group; TBI = traumatic brain injury group.

Briefly, the Creature Counting task assesses set-switching skills and requires participants to switch between counting stimuli in forward or reverse order, as denoted by arrows pointing up or down. The Walk-Don't-Walk subtest is a measure of response inhibition during which participants are instructed to place a mark in a box after a target tone is presented, but to withhold responding when a non-target sound is presented. The Code Transmission task assesses sustained attention and working memory, and requires participants to listen to a continuous stream of digits. They are instructed to listen for the occurrence of two consecutive target digits (e.g., two 5's in a row) and to identify the number that was presented just before the two target stimuli.

## Analyses

*T*-tests and chi square analyses were used to examine differences in demographic variables across the brain disorder and control groups. Profile analysis (i.e., a repeated-measures analysis with group and subtest as independent variables, with group treated as a between-subjects variable and subtest treated as a within-subjects variable) was used to examine patterns of TEA-Ch performance between the brain injury group as a whole relative to controls, and subsequently across the three brain injury groups relative to the control group. Both maternal education and age at testing were included as covariates.

We also ran an identical set of profile analyses using maternal education, age at diagnosis, and time between diagnosis and testing as covariates, to determine if our results changed with the inclusion of different age-related variables in the model. Additionally, because the range of ages of the participants in the four groups overlapped from 9 to 13 years only, an identical set of profile analyses were conducted among only 9- to 13-year-old participants, to determine if any significant findings were related to differences in age ranges across the groups. The 9- to 13-year-old sample consisted of 62 children with TBI, 11 children with stroke, 60 children with BT, and 109 controls.

Profile analyses were also performed within each group to examine how categorical pathology variables were associated with TEA-Ch performance, as well as to validate the use of the different executive function measures. The analysis within the TBI group compared children with complicated mild, moderate, and severe injuries. Additionally, linear regression was used to examine whether GCS scores predicted TEA-Ch performance, after controlling for maternal education and age at testing.

The analyses within the BT group examined the relationship of treatment modality (e.g., surgery only *vs.* adjuvant therapies), tumor location (coded based on the categories listed in Table 1), and tumor type (coded based on the categories listed in Table 1) to TEA-Ch performance. Multiple analyses were corrected for using the False Discovery Rate. This procedure controls for the expected proportion of type I errors while also preserving power, and is generally less stringent than procedures that control for family wise error rate (Benjamini & Hochberg, 1995). Hajek and colleagues (2014) examined the relationship of pathology-related variables to TEA-Ch performance extensively in the AIS group. Thus, we did not perform further analyses within the AIS group, but instead summarized results presented by Hajek et al. (2014).

# RESULTS

## Group Differences in Demographic Variables

Table 2 presents a summary of demographic variables by group. The groups differed significantly in intellectual

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ability, $F(3,327) = 10.01$, $p < .001$, age at testing, $F(3,332) = 19.38$, $p < .001$, and maternal education, $\chi^2(12, N = 329) = 23.28$, $p = .03$. Specifically, controls had higher Full Scale IQ scores than the TBI ($p = .001$), AIS ($p < .001$), and BT ($p < .001$) groups. The three clinical groups did not differ significantly in Full Scale IQ. With regard to age, the BT group was older than the control ($p = .002$), AIS ($p < .001$) and TBI ($p < .001$) groups; controls were older than the AIS ($p < .001$) and TBI ($p = .003$) groups; and the TBI group was older than the AIS group ($p = .007$). With regard to maternal education, a lower proportion of mothers in the AIS group completed post-secondary education as compared to the control, BT, and TBI groups ($p < .05$). The groups did not differ significantly in race (white *vs.* non-white), $\chi^2(6, N = 327) = 4.38$, $p = .63$.

The clinical groups differed significantly in age at diagnosis, $F(2,189) = 17.37$, $p < .001$, and time between diagnosis and testing, $F(2,189) = 30.98$, $p < .001$. Specifically, children in the BT ($p < .001$) and TBI ($p < .001$) groups were diagnosed at later ages than those in the AIS group. Children in the AIS ($p < .001$) and BT ($p < .001$) groups had longer time intervals between diagnosis and testing than those in the TBI group. Age at diagnosis was significantly correlated with age at testing ($r = .68$; $p < .001$) and time between diagnosis and testing ($r = -.70$; $p < .001$). Age at testing and time between diagnosis and testing were not significantly correlated ($r = .05$; $p = .43$).

## Group Differences in TEA-Ch Performance

As a whole, children with brain injury performed more poorly overall on the TEA-Ch subtests than controls, $F(1,300) = 4.76$, $p = .03$, partial eta$^2$ = .02. When examining TEA-Ch performance across the brain injury groups relative to the control group, the overall group × subtest interaction was significant, Wilks' $\lambda = .95$, $F(6,594) = 2.74$, $p = .01$, partial eta$^2$ = .03. *Post hoc* tests examining simple effects of group for each test were significant for Creature Counting ($p = .03$; partial eta$^2$ = .03) and Walk-Don't-Walk ($p = .03$; partial eta$^2$ = .03), but not for Code Transmission ($p = .34$). The simple effects of group remained significant after correcting for multiple comparisons using the False Discovery Rate.

Further examination using pairwise comparisons between groups on Creature Counting and Walk-Don't-Walk revealed several significant findings. Specifically, the TBI group performed significantly more poorly than controls ($p = .02$) on Creature Counting; the difference between controls and children with BT approached significance ($p = .09$). Children with AIS performed more poorly than controls on Walk-Don't-Walk ($p = .03$); the difference between controls and children with BT approached significance ($p = .10$). Importantly, performance on TEA-Ch subtests also differed among brain disorder groups. Children with TBI performed more poorly than those with AIS on Creature Counting ($p = .02$); the difference between children with AIS and BT approached significance ($p = .10$). On Walk-Don't-Walk, children with AIS ($p = .01$) and BT ($p = .05$) performed more poorly than children with TBI. However, none of the significant pairwise comparisons survived False Discovery Rate correction.

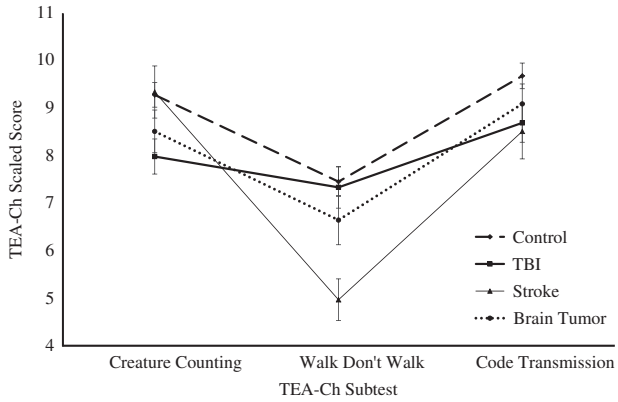


**Fig. 1.** Test of Everyday Attention: Children's Version (TEA-Ch) scores for all participants. TBI = traumatic brain injury.

Figure 1 provides a graphical representation of these results. Table 3 provides a summary of the effect sizes for the pairwise comparisons between groups on the TEA-Ch subtests. Notably, we found essentially the same results when maternal education, age at diagnosis, and time between diagnosis and testing were treated as covariates in the model ($p = .03$; partial eta$^2$ = .03).

Table 4 lists the percentages of children within each group who had impaired performance on the TEA-Ch subtests, defined as standard scores of 6 or less. Across all 4 groups of children, the proportions of children with impaired performance did not differ on Creature Counting ($p = .31$) or Code Transmission ($p = .40$). However, on Walk-Don't Walk, a higher proportion of children in the AIS group had impaired performance than the Control, BT, and TBI groups [$\chi^2(3, N = 332) = 11.77$; $p = .008$].

We observed a similar pattern of findings when comparing only 9- to 13-year-old participants ($p = .001$; partial eta$^2$ = .05). *Post hoc* tests of simple effects of group for each

**Table 3**. Summary of effect sizes for pairwise comparisons between groups on the TEA-Ch subtests

| | Creature Counting | Walk-Don't-Walk | Code Transmission |
|---|---|---|---|
| Control vs TBI | .42* | .03 | .30 |
| Control vs AIS | .02 | .81* | .37 |
| Control vs BT | .23 | .21 | .18 |
| TBI vs AIS | .44* | .76** | .05 |
| TBI vs BT | .16 | .18* | .12 |
| AIS vs BT | .25 | .50 | .18 |

*Note*. Effect sizes are reported as Cohen's *d*. AIS = arterial ischemic stroke group; BT = brain tumor group; TBI = traumatic brain injury group; TEA-Ch = Test of Everyday Attention: Children's Version.
* $p < .05$.
** $p \leq .01$.

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**Table 4**. Percentage of children within each group with impaired performance on the TEA-Ch subtests

| | Creature Counting | Walk-Don't-Walk | Code Transmission |
|---|---|---|---|
| Control | 21.5 | 42.4 | 19.4 |
| TBI | 31.7 | 39.0 | 25.6 |
| AIS | 25.0 | 72.2 | 22.2 |
| BT | 27.0 | 47.3 | 25.7 |

*Note*. Impaired performance is defined as a scaled score ≤6. AIS = arterial ischemic stroke group; BT = brain tumor group; TBI = traumatic brain injury group; TEA-Ch = Test of Everyday Attention: Children's Version.

test were significant for Creature Counting ($p = .002$; partial eta$^2 = .07$) and Walk-Don't-Walk ($p = .04$; partial eta$^2 = .04$), but not for Code Transmission ($p = .49$). The simple effect of group on Creature Counting remained significant after correcting for multiple comparisons using the False Discovery Rate; the simple effect of group for Walk-Don't-Walk approached significance after False Discovery Rate Correction ($p = .06$).

Further examination using pairwise comparisons between groups on Creature Counting and Walk-Don't-Walk showed that children with TBI performed more poorly than controls on Creature Counting ($p = .02$); the difference between controls and children with BT approached significance ($p = .06$). Conversely, children with AIS performed better than controls on Creature Counting ($p = .03$). On Walk-Don't-Walk, the difference between controls and children with AIS approached significance ($p = .06$), with children with AIS performing more poorly than controls.

Notably, performance on TEA-Ch subtests again differed among brain disorder groups when examining only 9- to 13-year-old participants. Children with TBI ($p = .001$) and BT ($p = .002$) performed more poorly on Creature Counting than those with AIS. Children with AIS ($p = .02$) and BT ($p = .03$) performed more poorly than children with TBI on Walk-Don't-Walk. On Creature Counting, the differences between controls and TBI, controls and AIS, AIS *versus* TBI, and AIS *versus* BT all remained significant after correcting for multiple comparisons using the False Discovery Rate. None of the other significant pairwise comparisons survived False Discovery Rate correction.

Supplementary Table 1 provides a summary of the effect sizes for pairwise comparisons between groups on the TEA-Ch subtests for the 9- to 13-year-old participants only. Notably, the results did not change appreciably when maternal education, age at diagnosis, and time between diagnosis were treated as covariates in the profile analysis comparing only 9- to 13-year-old participants ($p = .002$; partial eta$^2 = .06$).

Supplementary Table 2 lists the percentages of children within each group who had impaired performance on the TEA-Ch subtests for the 9- to 13-year-olds only. Across the four groups, the proportions of children who had impaired performance did not differ on Creature Counting ($p = .38$) or Code Transmission ($p = .46$). However, the difference in group proportions approached significance on Walk-Don't Walk, such that higher proportions of children in the AIS and BT groups had impaired performance relative to the Control and TBI groups [$\chi^2(3, N = 239) = 6.82$; $p = .08$].

## Within-Group Analyses of TEA-Ch Performance

### *TBI*

Within the TBI group, TEA-Ch performance did not differ between mildly, moderately, and severely injured groups ($p = .40$), or between participants with mild TBI relative to those with moderate *or* severe TBI combined ($p = .19$). In addition, GCS scores did not significantly predict performance on Creature Counting ($R^2 = .02$; Beta = .08; $t = .67$; $p = .50$) or Code Transmission ($R^2 = .09$; Beta = .12; $t = 1.03$; $p = .31$) after controlling for maternal education and age at testing. However, GCS scores significantly predicted performance on Walk-Don't-Walk ($R^2 = .17$; Beta = .24; $t = 2.19$; $p = .03$) after controlling for maternal education and age at testing. Nevertheless, the significant linear regression relating GCS to Walk-Don't-Walk performance did not survive False Discovery Rate Correction.

### *BT*

TEA-Ch performance did not differ as a function of tumor location ($p = .65$) or whether tumors were treated with surgical resection ($p = .18$). However, children who were treated with chemotherapy performed more poorly overall on the TEA-Ch subtests than those who were not, $F(1,57) = 6.83$, $p = .01$, partial eta$^2 = .01$, and those who received radiation therapy performed more poorly overall than those who did not, $F(1,57) = 6.93$, $p = .01$, partial eta$^2 = .11$. Additionally, children who were treated with resection only performed significantly better overall on the TEA-Ch subtests than those who received any form of adjuvant treatment (i.e., chemotherapy and/or radiation, $F(1,56) = 6.76$, $p = .01$, partial eta$^2 = .11$. All of the significant main effects of treatment modality on TEA-Ch performance remained significant after correcting for multiple comparisons using the False Discovery Rate.

Lastly, the overall group x TEA-Ch subtest interaction was significant when examining children with different types of tumors [Wilks' $\lambda = .64$; $F(12,100) = 2.12$; $p = .02$; partial eta$^2 = .20$]. None of the *post hoc* tests examining simple effects of children with different tumor types for each test were significant. However, the tumor type groups differed significantly in how they were treated [$\chi^2(12, N = 74) = 32.01$; $p = .001$]; a higher proportion of children with low grade gliomas/astrocytomas and other types of tumors were treated with resection only, whereas higher proportions of children with ependymoma, germ cell tumors, and high grade gliomas/astrocytomas were treated with radiation and/or chemotherapy.

### *Stroke*

Hajek et al. (2014) presented detailed information regarding the influence of pathology-related characteristics on TEA-Ch

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performance in the AIS group. To summarize, TEA-Ch performance did not differ significantly between children with perinatal and childhood strokes. TEA-Ch performance also was not associated with stroke volume, location, or severity of neurological sequelae as assessed by the Pediatric Stroke Outcome measure. Lesion laterality approached significance as a predictor of scores on the Walk/Don't Walk subtest, $F(3,43) = 2.72$, $p = .06$, such that children with bilateral AIS performed more poorly than children with unilateral AIS.

## DISCUSSION

We found that, as a whole, children with brain disorders (i.e., TBI, BT, AIS) showed similarly poor performance on measures of executive function relative to controls. Among children with brain disorder, inhibitory control performance fell within the low-average to below-average range. Shifting performance was generally within the average range across brain disorder groups, although significantly poorer than among controls. The non–brain-injured comparison groups comprising the control group did not differ on any aspects of TEA-Ch performance. Taken together, the findings suggest that childhood brain disorder is associated with poorer executive function performance overall. Of interest, a significant proportion of children in the brain disorder groups had non-frontal or multifocal pathologies based on clinical brain imaging, consistent with the notion that non-frontal lesions can contribute to executive dysfunction in children (Alvarez & Emory, 2006).

More importantly, the findings showed that different childhood brain disorders were associated with different magnitudes and patterns of executive function deficits. In fact, when considering effect sizes in pairwise comparisons, the largest differences in performance across groups did not only involve comparisons of children with brain disorder *versus* controls, but brain disorder *versus* brain disorder comparisons as well. Although the exact nature of the differences between groups on certain TEA-Ch subtests was less clear, our findings are consistent with theoretical accounts that fractionate executive functions into distinct components (e.g., Miyake et al., 2000; see also Jurado & Rosselli, 2007 for a review) and lend support to the notion that different clinical groups can be distinguished from each other by the severity and/or pattern of their executive function deficits (Pennington & Ozonoff, 1996).

Notably, because the brain disorder groups differed in age and other age-related characteristics (e.g., time since injury), we controlled for age in all analyses. However, the profiles of strengths and weaknesses in executive functions were almost identical when comparing only 9- to 13-year-old participants. These results indicate that differences in patterns of executive functions shown by children with the different childhood brain disorders are not simply attributable to differences in age at injury.

A variety of different pathology-related characteristics were examined to determine factors that were associated with executive dysfunction within each group, as well as to validate the use of the different executive function measures. Specifically, we found that children with BT who received adjuvant treatment performed more poorly overall on measures of executive function than those who did not. Furthermore, in the AIS sample, Hajek et al. (2014) reported a trend toward poorer inhibitory control in children with bilateral strokes *versus* those with unilateral strokes.

Although the commonalities among these pathology-related characteristics are not readily discerned, one possible mechanism that is common to these different factors is disruption of cerebral white matter. Indeed, adjuvant treatment in children with BTs is thought to result in executive dysfunction because of its detrimental effect on cerebral white matter (e.g., Fletcher & Copeland, 1988; Spiegler, Bouffet, Greenberg, Rutka, & Mabbott, 2004). Children with bilateral strokes may also be expected to have greater disruption to white matter brain regions than those with unilateral strokes.

If disruption of cerebral white matter is a common factor contributing to executive dysfunction in children with brain disorder, then one possible explanation for the different profiles of executive strengths and weaknesses is differential damage to the specific frontal–subcortical circuits that are thought to underlie these abilities. For example, deficits in inhibitory control may result from greater damage to ventromedial frontal brain regions, while problems in shifting may result from greater damage to dorsolateral frontal regions (Alexander, DeLong, & Strick, 1986; Middleton & Strick, 2001). This hypothesis warrants further research using advanced neuroimaging techniques.

Our findings involving children with BT are consistent with prior research supporting associations between adjuvant treatment with chemotherapy and radiation, and poorer attention and executive functions (e.g., Aarsen et al., 2009; Conklin et al., 2012; De Ruiter et al., 2013; Law et al., 2011). However, the results within the TBI group contrast with prior research documenting impairments in working memory, inhibitory control, and shifting as a function of injury severity (Levin & Hanten, 2005). Our null findings may reflect our use of the GCS as the sole measure of injury severity.

Indeed, although GCS ratings predict later functional outcomes, the predictive accuracy of GCS scores decreases as outcome measures become more narrowly defined (McNett, 2007), and other indices of TBI severity (e.g., duration of post-traumatic amnesia) may be more predictive of outcomes (Sherer, Struchen, Yablon, Wang, & Nick, 2008; van der Naalt, Zomeren, Sluiter, & Minderhoud, 1999). In addition, children with lower GCS scores do not show consistently different neuropsychological outcomes than those with higher GCS scores (Lieh-Lai et al., 1992). Our null findings could also be attributable to the fact that the TBI sample included children with complicated mild injuries, which may be associated with neurocognitive impairments that more closely resemble those seen in moderate TBI (Iverson & Lange, 2011).

Taken together, the current findings have implications for clinical practice and future research. They highlight the

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importance of examining different components of executive function to identify areas of relative strength and weakness, rather than administering a single task and concluding that executive functions are globally intact or deficient. Our results also suggest that executive functions should be thoroughly assessed even in children with non-frontal lesions. Furthermore, the current study provides proof of concept for future investigations across clinical groups, which may become more feasible with the increasing use of shared assessment approaches, such as the NIH Toolbox battery and the NINDS Common Data Elements. If additional evidence of disorder-specific profiles can be found, clinicians may be able to select tests that are most sensitive to disorder-specific patterns.

This study has several limitations. First, we used single tasks to assess discrete executive functions, and the tasks were chosen on the basis of face validity rather than being designed to assess specific aspects of executive function. Additionally, the different clinical groups were heterogeneous with regard to pathology-related characteristics, although characterization of pathology was limited to available clinical brain imaging only, which did not provide sufficient detail for comparisons across groups. This made it difficult to discern what accounted for differences in executive profiles across clinical groups, and precluded examination of specific relationships between TEA-Ch scores and brain pathology.

Finally, the clinical groups were not matched for age at injury or assessment, or time between diagnosis and testing. Disentangling the effects of all three of these age-related variables when examining outcomes associated with childhood brain injury is very challenging (Taylor & Alden, 1997). Nevertheless, we found a consistent pattern of differences across the clinical groups when including different age-related variables as covariates in our analyses.

Future research is needed to replicate these findings, ideally using more homogenous clinical groups, and perhaps experimental tasks that are designed to more purely assess different aspects of executive function. Neuroimaging data will also be important in determining how executive functions may relate to lesion volumes, locations, and white matter abnormalities.

Lastly, examination of how children with different profiles of executive function respond to different forms of remediation may be another fruitful avenue of research. For example, computerized training has been shown to improve working memory performance among children with different neurodevelopmental and medical disorders, although generalization of acquired skills to functional outcomes has been limited (e.g., Conklin et al., 2015; Grunewaldt, Lohaugen, Austeng, Brubakk, & Skranes, 2013; Klingberg et al., 2005).

Adolescents with TBI have also demonstrated improvement in executive skills (e.g., behavioral regulation, working memory) with counselor-assisted problem solving training (Kurowski et al., 2013). Other interventions that have been found to improve executive functions among healthy preschool- and school-aged children include classroom curricula that promote social pretend play, self-discipline, and child-to-child teaching, and physical activities that incorporate mindfulness and self-control (e.g., martial arts; Diamond & Lee, 2011).

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## Supplementary materials

To view supplementary material for this article, please visit journals.cambridge.org/jid_INS.

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