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Measurement of executive functioning with the National Institute of Health Toolbox and the association to anxiety/depressive symptomatology in childhood/adolescence

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Abstract

Introduction: Despite preliminary research, there remain inconsistent findings with regard to the role of executive functioning (EF) deficits in childhood anxiety and depression. This report examined the association of The National Institute of Health (NIH) Toolbox to clinical neuropsychological measures and to childhood, anxiety/depressive symptomatology. Methods: One-hundred eight children and adolescents completed the three EF measures from the NIH Toolbox (List Sorting Working Memory Test [LSWMT], Dimensional Change Card Sorting Test [DCCST], and Flanker Test of Attention and Inhibition [Flanker]) in an outpatient neuropsychology program. These tests were compared to established measures of EF in terms of linear correlations and detection of impairment. Heaton's Global Deficit Score (GDS) was utilized to calculate impairment. The Toolbox-EF measures were paired with parent-reported EF symptoms (Behavior Rating Inventory of Executive Function [BRIEF2]) to identify the role of EF in childhood anxiety/depressive symptomatology.

Results: Toolbox-EF measures displayed medium sized correlations with their clinically comparable counterparts, and generally did not differ in their detection of impairment. Toolbox-GDS was associated with depression diagnosis and clinically significant child-reported anxiety

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and depressive symptoms. Together, Toolbox/BRIEF2 accounted for 26.8–30.9% of elevated depressive symptom variance, but only 13.2–14% of elevated anxiety symptom variance. Further, EF impairment was associated with depression across self report, parent report, and clinical diagnosis.

Discussion: The NIH Toolbox-EF measures display comparable psychometric properties to clinically available EF measures in a pediatric (primarily psychiatric) neuropsychology setting. The Toolbox appears to display an appropriate ability to detect EF deficits secondary to self-reported depression in childhood.

Keywords

Executive functioning; NIH Toolbox; childhood; anxiety; depression

Psychiatric disorders are the leading cause of disability in the United States (Murray et al., 2013) and 22% of U.S. adolescents will experience at least one severely impairing psychiatric disorder (Merikangas et al., 2010). Psychiatric disorders are also the most costly to treat of any childhood health conditions (e.g. asthma, physical trauma, etc.), with $13.8 billion spent on treatment for psychiatric disorders in 2011 (Soni, 2014). Cognitive dysfunction is a core, transdiagnostic component of numerous psychiatric disorders (Masand & Pae, 2015; Millan et al., 2012) and occurs in as many as half of individuals with affective disorders (Godard, Grondin, Baruch, & Lafleur, 2011; Gu et al., 2016; Gualtieri & Morgan, 2008; Wagner et al., 2018). One of the most commonly affected cognitive domains in psychopathology is executive functioning (EF) (Etkin, Gyurak, & O'Hara, 2013; Snyder, Miyake, & Hankin, 2015).

EF is a collection of self-regulatory control processes that are divided into core subdomains of working memory (i.e. maintaining/manipulating data not perceptually present), inhibition (i.e. suppressing or controlling attention, thoughts, behaviors) and flexibility (i.e. shifting flexibly between tasks/sets) (Diamond, 2013; Miyake et al., 2000). Significant EF differences between cases and controls have been consistently identified across the majority of adult (Millan et al., 2012; Snyder et al., 2015) and childhood (Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008) affective disorders. EF predicts a host of clinical outcomes in studies of adult affective psychopathology, including long-term functional recovery (Gruber, Rosso, & Yurgelun-Todd, 2008; Jaeger, Berns, Loftus, Gonzalez, & Czobor, 2007), overall functioning (Martino et al., 2009), quality of life (Cotrena, Branco, Shansis, & Fonseca, 2016), and social/occupational functioning (O'Donnell et al., 2017; Withall, Harris, & Cumming, 2009). EF deficits are state-independent and persist during affective episode remission (Lee, Hermens, Porter, & Redoblado-Hodge, 2012).

Starting in early childhood, EF is a highly stable, heritable, and transdiagnostic trait-like feature of psychopathology (Bannon, Gonsalvez, Croft, & Boyce, 2006; Friedman et al., 2016; Hatoum, Rhee, Corley, Hewitt, & Friedman, 2018; Polderman et al., 2007). EF deficits in child/adolescent psychopathology has been associated with poorer academic outcomes (Biederman et al., 2004; Miller & Hinshaw, 2010), global functioning (Clark, Prior, & Kinsella, 2002; Gardiner & Iarocci, 2018; Miller & Hinshaw, 2010; Ware et al., 2012), social functioning (Miller & Hinshaw, 2010; Rinsky & Hinshaw, 2011), and

long-term functional outcomes (Lee et al., 2013; Miller & Hinshaw, 2010). In children and adolescents, lower cognitive flexibility has been associated with higher depressive symptoms and predictive of higher anxiety symptoms at follow-up (Han et al., 2016). Lower inhibition has been associated with polygenetic risk for depression (Schork et al., 2018) and higher general internalizing symptoms (Vuontela et al., 2013), including specific depressive symptoms of negative mood, low self-esteem, and interpersonal problems (Kavanaugh et al., 2016; Kavanaugh & Holler, 2014a, 2014b). Lower working memory has also been associated with higher anhedonia (Kavanaugh & Holler, 2014b) and higher internalizing symptoms at follow-up (Rinsky & Hinshaw, 2011).

The National Institute of Mental Health's Research Domain Criteria (RDoC) calls for the examination of transdiagnostic constructs across multiple levels of measurement (e.g., genetic, neural, cognitive) to improve the understanding on the underlying neurobiological underpinnings (Garvey, Avenevoli, & Anderson, 2016). The RDoC criteria therefore focuses on endophenotypes that may cut across psychiatric disorders. EF has received considerable interest as a potential endophenotype or critical underlying construct in affective psychopathology. Despite the potential for neuropsychology to integrate into neuroscience research, RDoC has specifically recommended that researchers avoid implementing clinical neuropsychological tests to measure neurocognitive constructs (National Advisory Mental Health Council Workgroup on Tasks and Measures for Research Domain Criteria (RDoC), 2016). As has been well-described by Robert Bilder, this concern from our colleagues may in part be because most commonly utilized neuropsychological tests were developed over 50 years ago (Bilder, 2011; Bilder & Reise, 2019). Neuropsychology must transition into tests that more appropriately integrate modern technology/neuroscience to maintain its relevance in a quickly changing healthcare and research climate.

The National Institute of Health Toolbox for the Assessment of Neurological and Behavioral Function (NIH Toolbox) is a new battery of neurobehavioral measures that utilizes an iPad to efficiently assess a range of functions (including cognition) in clinical and research settings across the lifespan (Gershon et al., 2013). The purpose of this study was to examine the association between EF and childhood anxiety/depression in an outpatient pediatric neuropsychology setting, with a particular focus on child/adolescent psychiatry. First, it was hypothesized that the EF measures from the NIH Toolbox (i.e. Toolbox-EF) would be closely associated with more established, clinical neuropsychological tests. Second, it was hypothesized that the Toolbox-EF and the Behavior Rating Inventory of Executive Function (BRIEF2) would independently predict unique aspects of anxiety/depressive symptomatology, specifically clinically significant parent and child-reported anxiety/depressive symptoms and clinically-assigned anxiety/depression diagnoses.

Methods

Participants

All children and adolescents (N = 108; 5 to 18 years; 5 year olds, n = 2; 65.7% Male) that completed the EF measures on the NIH Toolbox within a single pediatric neuropsychology program were included in this study (2017–2019). No exclusion criteria were implemented in order to examine the potential utility of the Toolbox-EF measures across the full range

of neurocognitive/psychiatric functioning. Participants completed the measures within the outpatient neuropsychology program as either (1) clinical patients, (2) research participants, or (3) both clinical patients and research participants. Specifically, 78 participants completed the measures as part of an outpatient pediatric neuropsychological evaluation. Children referred for evaluation within this service generally experience potential neurocognitive deficits secondary to a primary psychiatric, neurodevelopmental, or medical/neurological disorder. A separate group of 25 participants completed the measures as part of participation in a cognitive training research study within the same neuropsychology program. The research study examined the feasibility of an internet-based, cognitive training program for children and adolescents with EF deficits. The study was advertised in the hospital and in the community. Finally, 5 participants were initially evaluated as outpatients but then later enrolled in the cognitive training study. IRB approval was obtained for the cognitive training study and the chart review of the clinical evaluations, both of which were conducted within the outpatient neuropsychology program.

Demographic data for the entire study sample are provided in Table 1. No research procedures were conducted to establish psychiatric diagnoses and the psychiatric diagnoses reported here were clinically assigned prior to the study. Depressive disorder criteria included major depressive disorder, depressive disorder unspecified, and disruptive mood dysregulation disorder. Anxiety disorder criteria included generalized anxiety disorder, panic disorder, specific phobias, anxiety disorder unspecified, obsessive-compulsive disorder, and post-traumatic stress disorder. Regarding medication, psychostimulants and alpha-2 agonists (i.e. ADHD-related medications) were loaded into analyses as a covariate due to the known effect of these medications on EF (Coghill et al., 2014; van Stralen, 2018).

Measures

Self-report—The Children's Depression Inventory-Second Edition, self-report (CDI-2) is a self-rated scale of depressive symptoms in children aged 7–17 years (Kovacs, 2011). It includes 28 multiple-choice items that are categorized into two scales of Emotional Problems and Functional Problems, and four subscales of Negative Mood/ Physical Symptoms, Low Self-Esteem, Ineffectiveness, and Interpersonal Problems. Internal consistency of the self-report CDI-2 has been high, with alpha coefficients above .80 across various studies (Kovacs, 2011). The Screen for Child Anxiety Related Disorders, self-report (SCARED) is a self-rated scale of anxiety symptoms in children ages 8–18 years (Birmaher et al., 1999). It includes 41 multiple-choice items that are categorized into subscales of Panic/Somatic, Generalized Anxiety, Social Anxiety, Separation Anxiety, and School Avoidance, with excellent internal consistency (Cronbach's alpha = .90). The total scores (T-scores: CDI; raw: SCARED) were utilized from each of these scales. Only participants within the recommended age range were administered these questionnaires.

Parent-report—The Behavior Rating Inventory of Executive Function-Second Edition (BRIEF2), parent form (Gioia, Isquith, Guy, & Kenworthy, 2015) is a parent-rated scale of executive functioning in children aged 5 to 18 years. It includes 63 multiple-choice items that are categorized into subscales of Inhibit, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Organization of Materials, and Task Monitor. Internal

consistency is high across both clinical and standardization samples, with alpha coefficients of .85 and .89, respectively (Gioia et al., 2015). As this study is specifically focused on empirically-based core EF subdomains (Diamond, 2013), only Inhibit, Working Memory, and Shift subscales were utilized (T-scores). The Behavior Assessment System for Children – Third Edition (BASC-3), parent form (Reynolds & Kamphaus, 2015) is a parent-rated scale of emotional-behavioral functioning in children aged 6–18. It includes 173 (6–11 years) to 175 (12–18 years) multiple-choice items that are categorized into index scores of Externalizing Problems, Internalizing Problems, Behavioral Symptoms, and Adaptive Skills. This study only utilized the subscales of Anxiety and Depression (T-scores) as a parent-report assessment of symptomatology. Internal-consistency reliability coefficients are good to excellent (with the majority of values above .90) and are consistent across gender, age, and clinical groups (Reynolds & Kamphaus, 2015). Only participants within the recommended age range were administered these questionnaires.

NIH toolbox—The National Institutes of Health Toolbox for the Assessment of Neurological and Behavioral Function (i.e. NIH Toolbox) is a brief neuropsychological screening battery of neurocognition across brain-based disorders (Gershon et al., 2013). The tasks are administered on an iPad, with all instructions presented orally and visually on the screen (O'Donnell et al., 2017). Working memory was assessed with the List Sorting Working Memory Test (LSWMT). Inhibition was assessed with the Flanker Inhibitory Control and Attention Test (Flanker). Flexibility was assessed with the Dimensional Change Card Sort Test (DCCST). Administration of these three EF tasks (i.e. Toolbox-EF measures) takes approximately 15 minutes (Weintraub et al., 2013). While each subdomain is only assessed with a single task, the results of all task can be averaged to create a "common EF" metric to represent the RDoC construct for each patient (Miyake et al., 2000). These tests display excellent psychometric properties in children and adolescents (Tulsky et al., 2013; Zelazo et al., 2013).

Neuropsychological measures—The Wechsler Abbreviated Scale of Intelligence-Second Edition (WASI-II), Wechsler Intelligence Scale for Children-Fourth Edition (WISC-V), or Wechsler Adult Intelligence Scale of Intelligence-Fourth Edition (WAIS-IV) were used to assess intelligence (Wechsler, 2008, 2011, 2014). When available, the Working Memory Scale on WISC-V/WAIS-IV was utilized to assess working memory. The NEPSY-Second Edition (NEPSY-II) subtest, Inhibition, was utilized to measure inhibition (i.e. Inhibition-Inhibition score) and flexibility (i.e. Inhibition-Switching)(Korkman, Kirk, & Kemp, 2007). Standardized scores were utilized for all measures. The standard protocol for the research study and clinical practice generally involves the Toolbox-EF measures, BASC-3, CDI-2/SCARED, NEPSY-II: Inhibition (Switching subtest not administered in research protocol), and WASI-II, WISC-V, or WAIS-IV. The BRIEF2 was a part of the research protocol and originally part of the clinical protocol. During the period that the BRIEF2 was used clinically, it was administered universally to all participants. As is common for standard pediatric neuropsychology practice, the BRIEF2 is administered to obtain data on rater-based EF weaknesses, i.e. EF weaknesses observed in the child's day-to-day environment. This is complimentary data to that obtained from performance-based EF measures. The BRIEF2 was eventually removed from the standard battery. However,

particularly in the clinical sample, the standard protocol was often adjusted based on the referral question and patient-specific characteristics (resulting in variable sample size in this report). Other clinical EF tests were not included here due to small sample size (e.g., Trail Making Test, n = 29).

Global deficit score—Heaton's Global Deficit Score (GDS) is a measure of neurocognitive deficit severity that utilizes a 5-point scale to categorize test performance from intact (0) to severe impairment (5) (Carey et al., 2004; Heaton et al., 2004). Individual test scores are then averaged together to create a GDS composite score (for each domain or across the whole battery). Specifically, the GDS is rated as 0–5 (utilizing z-scores); **0** = −1.0 and above (i.e. normal); $-1.5 \leq$ **1** < -1.0 (mild impairment); $-2.0 \leq$ **2** < -1.5 (mild to moderate impairment); $-2.5 \leq$ **3** < -2.0 (moderate impairment); $-3.0 \leq$ **4** < -2.5 (moderate to severe impairment); **5** < -3.0 (severe impairment). A subsequent cutoff of ≥0.5 displays strong properties in detecting neurocognitive impairment in adult HIV samples (Carey et al., 2004; Heaton et al., 2004). This indicates that on average, the participant is at least mildly impaired on 50% of tests (Carey et al., 2004; Heaton et al., 2004). Recently, the GDS was able to predict the length of hospitalization in a children's inpatient psychiatric program sample. A cut off of .43–.63 displayed optimal sensitivity/specificity in detecting prolonged length of hospitalization (Kavanaugh, Studeny, Cancilliere, & Holler, 2019).

Statistical analyses

The range of clinically obtained standardized scores (e.g. scaled scores, T-scores) were universally converted to z-scores and the GDS was then calculated for each test. The overall GDS was calculated for the NIH Toolbox (i.e. average GDS for the 3 tests; "Toolbox-GDS") and the BRIEF2 (i.e. average GDS for the three scales of inhibition, working memory, and shifting; scores reversed to be in correct direction; "BRIEF-GDS"). First, Toolbox/BRIEF2 differences between samples were examined. Toolbox-EF measures were then compared to established, clinically available tests that purportedly measure the same underlying EF constructs: Wechsler: WMI and NIH Toolbox: LSWMT, NEPSY-II: Inhibition-Inhibition and the NIH Toolbox: Flanker, and NEPSY-II: Inhibition-Shifting and the NIH Toolbox: DCCST. Pearson correlation analyses examined the associations between GDS scores. Paired samples t-test analyses examined the differences in impairment detection between GDS scores. Analyses of covariance (ANCOVA; after controlling for age, sex, ADHD diagnosis (yes/no), and ADHD-related medications) examined EF differences between participants with/without depressive disorder diagnoses and between participants with/without anxiety disorder diagnoses.

A series of logistic regression analyses examined the role of EF in elevated anxiety/depressive symptoms. In each analysis, age, sex, ADHD diagnosis (yes/no), and ADHD-related medications were loaded and followed by the BRIEF-GDS and Toolbox-GDS. Dependent variables consisted of the BASC-3: Anxiety and Depression subscales, the CDI-2: Total, and the SCARED: Total. Clinically significant or elevated symptoms were defined as a T-score ≥ 60 (i.e. one standard deviation above mean) for BASC-3 and CDI-2. The SCARED does not produce T-scores, and instead has a cutoff of ≥ 25 (or 30 for more severe symptom cutoff). Of note, we could have defined cutoff of T ≥ 70 for BASC-3/CDI-2

and raw ≥ 30 for SCARED to reflect more severe symptom ranges, but we selected the milder cutoff due to concerns about small sample size. Significance levels were set at $p < .05$.

Results

There was no difference between research and clinic groups in age (F[1,106] = .189; $p = .664$), sex (F[1,106] = .973; $p = .326$), race (X^2[4] = 6.927, p = .140), maternal education (x^2[8] = 11.395, p = .180), Toolbox-GDS (F[1,95] = 2.664; $p = .106$), and BRIEF-GDS (F[1,58] = .174; $p = .678$). All subsequent analyses were conducted with the combined sample.

Correlations among measures

As noted in Table 2, there was a statistically significant association between NEPSY-II: Inhibition and NIH Toolbox: Flanker, between WISC-V: WMI and NIH Toolbox: LSWMT, and between NIH Toolbox: DCCST and NEPSY-II: Inhibition-Switching. No Toolbox-EF measures were correlated with BRIEF2 measures (Table 2).

Impairment differences across measures

Paired sample t-tests indicated there was no statistically significant difference in GDS between WISC-V: WMI and NIH Toolbox: LSWMT (t [39] = .597; $p = .554$; GDS = .78 [1.27] & .65 [1.15], respectively) or between NEPSY-II: Inhibition and NIH Toolbox: Flanker (t [88] = .740; $p = .461$; GDS = .89 [1.14] & .80 [1.09], respectively). There was a statistically significant difference between NEPSY-II: Inhibition-Switching and NIH Toolbox: DCCST (t [54] = −2.083; $p = .042$; GDS = .53 [1.04] & .25 [.65], respectively), in that the NEPSY-II was more sensitive to impairment than Toolbox.

Differences between depression/no depression and anxiety/no anxiety

As noted in Table 3, there was a statistically significant difference between depression/no depression groups in Toolbox-GDS (F [1, 91] = 4.892, $p = .029$), but not BRIEF-GDS (F [1, 54] = .005, $p = .942$). In follow-up analyses of specific EF subdomains, only Flanker (F = 9.800; p = .002), but not LSWMT (F = 1.173; p = .282) or DCCST (F = .055; p = .815) was associated with diagnosis after controlling for previously stated variables. There was no statistically significant difference between anxiety/no anxiety groups in Toolbox-GDS (F [1, 91] = 3.512, $p = .064$) or BRIEF-GDS (F [1, 54] = .008, $p = .930$; Table 3).

EF predictors of elevated anxiety/depressive symptoms

All four overall models were statistically significant, including self-reported depressive symptoms (X^2 [6] = 21.342, p = .002; 57.8% of total variance [BRIEF/Toolbox: 30.9% of variance]; correctly classified 82.1% of cases), self-reported anxiety symptoms (X^2 [6] = 16.697, p = .010; 45.9% of total variance [BRIEF/Toolbox: 13.6% of variance]; correctly classified 75.0% of cases), parent-reported depressive symptoms (X^2 [6] = 21.157, p = .002; 44.6% of total variance [BRIEF/Toolbox: 26.8% of variance]; correctly classified 76.9% of cases), and parent-reported anxiety symptoms (X^2 [5] = 18.725, p = .005; 40.5% of total variance [BRIEF/Toolbox: 13.0% of variance]; correctly

classified 78.8% of cases). As noted in Table 4, after controlling for age, sex, ADHD diagnosis, and ADHD-related medications, BRIEF-GDS independently predicted parent-reported depressive symptoms, while Toolbox-GDS independently predicted self-reported anxiety and depressive symptoms. In follow-up analyses of specific EF subdomains (when significant findings were obtained), only Toolbox: DCCST (B = 1.028; p = .041) predicted self-reported depressive symptoms. No subdomains predicted self-reported anxiety symptoms or parent-reported depressive symptoms.

EF impairment criteria

Based on these results, we examined the implementation of formal EF impairment diagnostic criteria. Mimicking the GDS approach (Carey et al., 2004; Cysique et al., 2014; Marquine et al., 2018), we set ≥ .5 as neurocognitive impairment for Toolbox-GDS. In the GDS approach (within a dementing, adult population), the neurocognitive GDS is supplemented with a reported decline in 2 to 3 domains of activities of daily living (ADLs). As a decline in ADLs is not as relevant to child psychiatry, we instead implemented the BRIEF2 into the criteria for EF impairment as reflecting the clinical impairment or daily living symptoms of EF. Criteria was set as BRIEF-GDS ≥ 1 (i.e. 1 SD above mean) on any of the inhibition, working memory, or flexibility domains. Both Toolbox and BRIEF2 criteria were required for impairment status. There were no group differences between age, sex, ADHD diagnosis, and ADHD-related medications (Table 5). Significant differences were detected between EF impaired and intact groups in self-reported depressive symptoms, parent-reported depressive symptoms, parent-reported anxiety symptoms, and depression diagnosis, but not self-reported anxiety symptoms and anxiety diagnosis.

Discussion

This study investigated the potential utility of the NIH Toolbox-executive functioning (EF) measures in detecting clinically meaningful neurocognitive impairment. First, the tests were compared to clinically available EF tests. The List Sorting Working Memory Test (LSWMT) is designed to measure working memory, the Flanker Inhibitory Control and Attention Test (Flanker) is designed to measure inhibition, and the Dimensional Change Card Sort Test (DCCST) is designed to measure flexibility. All three tests displayed statistically significant and medium-sized associations with their established clinical counterpart tests (r = .33: inhibition to r = .41; flexibility). The LSWMT and Flanker did not differ from their clinical test counterparts in the detection of impairment, while the DCCST was somewhat less sensitive in detecting flexibility impairment. The rates of impairment were also similar between tests for flexibility (26.7–28.1%), working memory (28–35.4%), and inhibition (45.1–48.6%). Taken together, results indicate that the Toolbox-EF measures are broadly comparable to clinically available EF measures when utilized within the child/adolescent psychiatric setting.

Within this context, this study then examined the role of EF in childhood anxiety and depression. Measured via Toolbox, EF deficits were associated with a clinical diagnosis of depression and self-reported anxiety/depressive symptoms, but not parent-reported symptoms or anxiety diagnosis. Alternatively, EF deficits measured via BRIEF2

were associated with parent-reported depressive symptoms, but not self-reported anxiety/depressive symptoms, parent-reported anxiety symptoms, or clinical diagnoses of anxiety/depression. When examining specific EF subdomains, response inhibition was associated with depressive disorders, while cognitive flexibility was associated with self-reported depressive symptoms. Working memory was not independently associated with any anxiety/depression measures.

These findings are consistent with a prior meta-analysis that identified EF deficits in major depression during childhood/adolescence (Wagner, Müller, Helmreich, Huss, & Tadić, 2015), and extend specific findings regarding the association between inhibition/flexibility and depressive symptoms (Han et al., 2016; Kavanaugh et al., 2016; Kavanaugh & Holler, 2014a, 2014b). One potential novelty of current findings is the utilization of a combination of performance (i.e. Toolbox) and rater (i.e. BRIEF2)-based EF measurement. These measures proved to be complimentary in identifying anxiety/depression, as BRIEF2 measures were more closely related to parent-rated symptoms while Toolbox-EF measures were more closely related to diagnoses and self-reported symptoms. In fact, after controlling for relevant variables, together the Toolbox/BRIEF2 accounted for 26.8–30.9% of elevated depressive symptom variance (along with 13.0–13.6% of elevated anxiety symptom variance). Broadly, results appear to confirm the established the role of EF deficits in depression, although the role of EF deficits in anxiety was not consistently observed. Of note, current findings were obtained within a clinical neuropsychology program (including a high rate of ADHD [81%]) and may not reflect a standard child anxiety/depression presentation.

At the neurobiological level, the link between EF and depression can be explained by their shared neuroanatomical correlates, particularly the frontoparietal network or central executive network (CEN)(Kaiser, Andrews-Hanna, Wager, & Pizzagalli, 2015; Niendam et al., 2012; Pizzagalli, 2011). In this perspective, EF measures may be conceptualized as probes of CEN integrity, with identified EF deficits reflecting dampened or suboptimal CEN activity that could contribute to depression vulnerability. At the neurocognitive level, deficits in cognitive flexibility may lead to perseverative tendencies, such struggles to ignore maladaptive thoughts or feelings. Deficits in response inhibition may similarly cause impulsivity in decision making or information processing, which could potentially lead to a negative interpretation of neutral or positive stimuli (e.g., emotional expressions of others). At the behavioral or measurement level, EF deficits and depression share many symptoms (e.g., irritability, inattention, and rigidity) that can make it hard to differentiate these two symptom clusters.

With the objective of moving toward a universal definition of neurocognitive impairment in child psychopathology, we tested one potential approach to defining EF impairment. Heaton and colleagues have long utilized the Global Deficit Score (GDS) to detect neurocognitive impairment in adult HIV, with a GDS ≥ .5 set as the optimal cutoff for detecting impairment (Carey et al., 2004; Heaton et al., 2004). In that approach, the GDS is utilized to categorize intact (GDS < .5), mildly impaired (.5 ≤ GDS < 1.5), or moderately impaired (GDS ≥ 1.5) neurocognition. This is paired with measurement of activities of daily living (ADLs) to identify asymptomatic neurocognitive impairment (no ADL decline), mild neurocognitive

disorder (mild to moderate ADL decline), and dementia (severe ADL decline)(Kamminga et al., 2017; Marquine et al., 2018). In our initial attempt to integrate the GDS into the pediatric setting, we found that a GDS = .43–.63 was able to detect prolonged hospitalization within a children's inpatient psychiatric program (Kavanaugh et al., 2019).

In an attempt to model the approach of Heaton and build on preliminary findings, we set impairment as GDS > .5 on Toolbox-EF, and to reflect clinical or functional impairment, BRIEF-GDS ≥ 1 (i.e. 1 SD above mean) on any of the inhibition, working memory, or flexibility domains. Compared to those with Intact EF, those with Impaired EF displayed higher parent-reported anxiety/depressive symptoms, higher self-reported depressive symptoms, and higher rates of depression. This provides the first attempt of our knowledge to implement formal diagnostic criteria for EF impairment in childhood psychopathology. The findings are promising and suggest that adapting the GDS model in childhood psychopathology may prove useful (at least for children evaluated within a neuropsychology program). However, this is a small first step that will need to be continuously tested, refined, and potentially replaced by a better approach in the ultimate goal of a universally accepted and empirically supported approach to identifying neurocognitive impairment in childhood psychopathology.

Current results indicate that the Toolbox-EF measures are at least comparable to established neuropsychological measures and sensitive/specific enough to detect EF deficits underlying childhood depression. There may be a few potential advantages of the Toolbox-EF measures. The measures can be administered/scored in ~15 minutes by bachelors-level professionals (e.g., testing technicians and research assistants). As the measures are administered or guided by verbal/visual cues from the iPad, there may be improved standardization as compared to standard neuropsychological measures. The measures can be administered in both clinical and research settings, and in collaboration with a neuropsychologist, can be interpreted by non-neuropsychological providers. The field of neuropsychology needs to move toward a new era of neurocognitive measurement, and the NIH Toolbox reflects one, of many, promising tools to help achieve this goal.

This study is not without limitations. First, this sample included only those children and adolescents with potential neurocognitive impairments, including those children referred for neuropsychological evaluation and those children recruited to participate in a cognitive training study. Future studies will need to obtain a spectrum of psychopathology and EF (enriched for clinically elevated symptoms) to most appropriately examine the association between these variables. Second, the missing data was not handled with complex analyses such as maximum likelihood or multiple imputation. This may have resulted in differences between children that were included and excluded from each analysis. Future studies will need to address this limitation. Third, clinical measures of overall depression and anxiety were utilized that capture a range of depression/anxiety-related symptoms. Future studies will need to utilize more specific RDoC-consistent measures of positive and negative valence to systematically investigate the EF-affect association. Fourth, no measures of performance validity (i.e. tests to establish the validity of neurocognitive performance; e.g., Test of Memory Malingering [TOMM]) were consistently implemented into the battery.

Future studies must utilize stand-alone measures of performance validity to appropriately measure and interpret EF.

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Table 1.

Demographic and clinical data (n = 108).

Male	**65.7% (n = 71)**
Age	10.42 (3.05)
Full Scale Intelligence (FSIQ)	94.70 (14.65)
Outpatient Mental Health Services	81.6% (n = 21)
Grade	4.95 (3.02)
Academic Services: IEP/504 Plan	59% (n = 63)
Mother/Primary Caregiver Education	n = 97
HS Degree/GED	31.9% (n = 31)
College Degree	35% (n = 34)
Master's or Doctoral Degree	27.8% (n = 27)
Father/Secondary Caregiver Education	n = 85
HS Degree/GED	42.3% (n = 36)
College Degree	31.7% (n = 27)
Master's or Doctoral Degree	17.6 (n = 15)
Parent & Child Primary Language: English	100%
Child's Race	
Asian	2.0% (n = 2)
Black or African American	6.1% (n = 6)
White	74.7% (n = 74)
Other	17.2% (n = 17)
Child's Ethnicity	
Hispanic	14.1% (n = 14)
Non-Hispanic	85.9% (n = 85)
Psychiatric Diagnoses	
Adjustment Disorder	9% (n = 10)
Anxiety Disorder	54% (n = 58)
Attention Deficit Hyperactivity Disorder	81% (n = 88)
Inattentive Type	14% (n = 15)
Hyperactive/Impulsive Type	3% (n = 3)
Combined Type	55% (n = 59)
Unspecified Type	10% (n = 11)
Autism Spectrum Disorder	9% (n = 10)
Bipolar Disorder	1% (n = 1)
Depressive Disorder	34% (n = 37)
Developmental Coordination Disorder	5% (n = 5)
Intellectual Disability	5% (n = 5)
Language Disorder	5% (n = 5)
Learning Disorders	25% (n = 27)
Obsessive-Compulsive Disorder	2% (n = 2)
Oppositional Defiant Disorder	9% (n = 10)

Male	**65.7% (n = 71)**
Post-Traumatic Stress Disorder	6% (n = 6)
Psychosis	1% (n = 1)
Social Communication Disorder	5% (n = 5)
Tic Disorder	3% (n = 3)
Psychiatric Medications	
Alpha-2 Agonist	17% (n = 18)
Antidepressant	31% (n = 33)
Anxiolytic	1% (n = 1)
Atypical Antipsychotic	6% (n = 7)
Mood/AED	6% (n = 7)
Stimulant	38% (n = 41)
Medical/Neurological Disorders	9% (n = 10)

Note. IEP: Individualized Education Program. Medical/neurological disorders: Hippocampal dysplasia, hypothyroidism, spondyloepiphyseal dysplasia, concussion, acute lymphoblastic leukemia, brain tumor, rhabdomysarcoma, HIV, epilepsy

Table 2.

Descriptive and correlational data for toolbox, BRIEF2, and clinical measures.

	Descriptive Data				Correlation to Toolbox		
	n	**Mean (SD)**	**GDS (SD)**	**Impairment/Elevated**	**Flanker**	**LSWMT**	**DCCST**
Toolbox: Flanker	105	87.41 (13.75)	.89 (1.17)	48.6%	-	-	-
Toolbox: LSWMT	100	94.19 (15.35)	.52 (1.01)	28%	.177	-	-
Toolbox: DCCST	105	91.91 (13.12)	.52 (1.06)	26.7%	.422**	.231*	-
NEPSY-II: Inhibition	91	7.42 (3.27)	.90 (1.14)	45.1%	.332**	−.048	.206
NEPSY-II: Switching	57	7.86 (3.28)	.58 (1.12)	28.1%	.288*	.305*	.406**
Wechsler: WMI	48	88.92 (16.09)	.81 (1.30)	35.4%	.308*	.403**	.381**
BRIEF2-Inhibit	60	67.32 (12.83)	2.2 (1.78)	73.3%	−.070	.131	−.160
BRIEF2-WM	60	69.70 (8.90)	2.4 (1.63)	85%	−.149	.000	.227
BRIEF2-Shift	60	65.85 (13.39)	2.1 (1.86)	66.7%	.146	−.013	−.020
CDI-2: Total	56	57.30 (13.77)	-	39.3%	.180	−.012	.005
SCARED: Total (raw)	54	25.37 (15.06)	-	48.4%	.079	−.033	.026
BASC-3: Anxiety (parent)	89	59.24 (12.05)	-	47.2%	.257*	−.071	−.001
BASC-3: Depression (parent)	89	62.36 (13.64)	-	39.3%	.151	−.148	−.008

Impairment: < 1 standard deviation below mean for performance-based EF, > 1 standard deviation above mean for rater-based EF; clinically elevated symptoms: ≥ 60 for CDI-2/BASC-3 and ≥ 25 for SCARED.

Table 3.

Toolbox-GDS differences based on clinical diagnoses of anxiety and depressive disorders.

	Depressive disorder (n = 32)	**No depressive disorder (n = 65)**	**F**
Toolbox GDS	.890 (.14)	.516 (.10)	4.892 *
BRIEF2 GDS	2.26 (.28)	2.23 (.23)	.006
	Anxiety Disorder (n = 44)	**No Anxiety Disorder (n = 53)**	**F**
Toolbox GDS	.767 (.11)	.449 (.13)	3.512
BRIEF2 GDS	2.23 (.23)	2.26 (.28)	.008

Estimated marginal means and standard errors are reported. F value is reported after controlling for age, sex, ADHD diagnosis, and ADHD-related medications.

* $p < .05$.

Table 4.

Logistic regression analyses of EF predictors of elevated anxiety/depressive symptoms.

	CDI-2: Total (n = 39)			SCARED: Total (n = 35)		
	B	**Wald**	**p**	**B**	**Wald**	**p**
Age	.720	4.095	.043	.269	1.587	.208
Sex	2.683	4.749	.029	2.300	5.830	.016
ADHD Diagnosis	2.747	1.917	.166	−1.106	.509	.476
ADHD Medication	1.062	1.495	.222	.15	.044	.833
BRIEF-GDS	.553	1.660	.198	.320	.720	.396
Toolbox-GDS	2.455	6.328	.012	1.301	3.866	.049
	BASC-3: Depression (n = 52)			**BASC-3: Anxiety (n = 52)**		
	B	**Wald**	**p**	**B**	**Wald**	**p**
Age	.474	7.758	.005	.472	7.109	.008
Sex	−.833	.999	.318	1.413	2.797	.094
ADHD Diagnosis	−.909	1.005	.316	−.282	.090	.764
ADHD Medication	.563	.893	.345	.729	1.326	.250
BRIEF-GDS	.982	8.893	.003	.573	3.834	.050
Toolbox-GDS	.595	1.485	.223	.726	2.039	.153

Table 5.

Clinical differences between impaired and intact EF.

	Intact EF (n = 33)	Impaired EF (n = 23)	F/X^2
Age	10.8 (2.8)	10.1 (2.7)	.891
Male	67%	61%	.198
ADHD-Related Meds	.8 (.7)	.6 (.7)	1.142
ADHD Diagnosis	88%	74%	1.802
BASC-3: Depression	60.2 (14.0)	69.1 (15.0)	4.650*
BASC-3: Anxiety	58.0 (11.6)	66.4 (14.9)	5.220*
CDI-2:Total	52.4 (8.7)	60.9 (14.7)	5.084*
SCARED: Total	22.7 (14.1)	25.7 (13.3)	.449
Depression Diagnosis	27%	61%	6.321*
Anxiety Diagnosis	48%	65%	1.536

Note.

*$p < .05$.