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Research Article

Cracking under pressure: Cognitive load influences performance in youth with NF1

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Abstract

Objectives: To extend the current understanding of executive function (EF) deficits in youth with neurofibromatosis type 1 by investigating the impact of cognitive load on performance compared to typically developing children. **Methods:** In this prospective multicenter study, 42 children with neurofibromatosis type 1 (NF1) (ages 7–18) completed neuropsychological assessments of intellect and executive functioning. Age- and sex-matched controls ($n = 42$) were drawn from the normative database for the tasks of executive control (TEC). Multivariate and supplementary univariate analyses examined group differences and task effects (inhibitory control and working memory demand). Associations between TEC performance and parent-reported executive dysfunction (BRIEF) were also explored. **Results:** Both groups showed reduced accuracy and speed with increased inhibitory demand and made fewer errors with increased working memory demand. However, children with NF1 were significantly less accurate and consistent across tasks, particularly under higher cognitive load, while controls improved or maintained performance. Significant group × cognitive load interactions were observed, and laboratory-based deficits in NF1 were associated with parent-reported executive dysfunction. **Conclusions:** Children with NF1 experience unique and multidimensional decrements in EF performance in response to increased cognitive load, unlike typically developing peers. These deficits appear to be clinically relevant. Targeting working memory and inhibitory control may reduce susceptibility to cognitive overload and improve outcomes for children with NF1.

Keywords: Cognition; neurofibromatosis; executive; function; child; adolescent

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Statement of Research Significance

Research Question(s) or Topic(s): We aimed to examine executive function deficits in children with Neurofibromatosis Type 1, focusing on how increased cognitive load contributes to the overwhelm frequently reported by these patients. **Main Findings**: Children with Neurofibromatosis Type 1 demonstrate greater susceptibility to tasks with higher cognitive load, exhibiting significantly poorer accuracy and consistency in executive function performance compared to typically developing peers. **Study Contributions**: By identifying the unique negative impact of increased cognitive load on executive functioning in children with Neurofibromatosis Type 1, our findings highlight the need for targeted interventions. Such interventions may help reduce cognitive overload and with the goal of improving academic achievement and social–emotional well-being in this population.

Introduction

Neurofibromatosis type 1 (NF1) is an autosomal dominant genetic condition affecting approximately 1 in 2500–3000 live births (Boyd et al., 2009). Variants within the *NF1* gene on chromosome 17q11.2 downregulates production of the tumor suppressor protein neurofibromin, which leads to uncontrolled cell proliferation and contributes to heterogenous impacts across multiple body systems, including the central nervous system (Boyd et al., 2009). While clinical diagnostic criteria for NF1 are based on the presence of common physical features (e.g., café-au-lait macules, freckling, iris Lisch nodules, neurofibromas, optic pathway glioma) (Legius et al., 2021), the condition is frequently associated with significant cognitive and behavioral morbidity, with studies indicating that most children with NF1 (up to 80%) show cognitive deficits in one or more areas (Crow et al., 2022; Hyman et al., 2005). Developmentally,

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children with NF1 are more likely to demonstrate motor, language, and cognitive delays (Lorenzo et al., 2011). Intellectual functioning most often falls in the low average to average range, though multiple studies show small but significant reductions in overall intellectual functioning compared to normative and sibling controls (Hyman et al., 2005). There is also an increased prevalence of neurodevelopmental disorders, including attention-deficit/hyperactivity disorder (Boulanger & Larbrisseau, 2005; Torres Nupan et al., 2017), Autism Spectrum Disorder (Chisholm et al., 2018; Garg et al., 2013), and Specific Learning Disorders (Boulanger & Larbrisseau, 2005; Hyman et al., 2006).

A growing body of research aiming to understand the cognitive phenotype of NF1 identifies a wide range of affected areas, with particularly high likelihood of deficits in visual-perceptual skills, visual-motor skills, attention, and executive function (EF) (Hyman et al., 2005; Payne et al., 2021). EF are higher-level cognitive processes involved in carrying out and regulating cognitive and emotional functions and goal-directed behavior (Diamond, 2013) and are important in academic skills development including reading, writing, and mathematics in the general population (Samuels et al., 2016; Willoughby et al., 2019) as well as NF1 (Geoffray et al., 2021; Hou et al., 2023; Hou et al., 2020; Pride et al., 2012). Research shows that EF deficits are a core cognitive feature of NF1, even in the absence of ADHD (Payne et al., 2012); with previous research showing that compared to typically developing children, youth with NF1 show deficits across all aspects of EF, including working memory, planning/problem solving, inhibitory control, and cognitive flexibility (Beaussart et al., 2018; Smith et al., 2020). While EF challenges are clearly pervasive in NF1, there is nonetheless substantial variability in cognitive profiles, and longitudinal studies also show within-person variability in cognitive outcomes over time (Payne et al., 2019; Van Der Vaart et al., 2016). Therefore, additional research is needed to better understand specific cognitive profiles of NF1 and factors that contribute to inter- and intra-individual heterogeneity.

One central factor, which may support understanding of the variable cognitive presentation of youth with NF1 is *cognitive load*. Cognitive load theory (Sweller, 1988; Van Merriënboer & Sweller, 2005) postulates that what an individual learns and/or how they perform on a given task is highly dependent on the cognitive "load" – or cognitive resources/effort necessary for a task. Cognitive load can vary based on intrinsic (e.g., inherent difficulty or complexity of the material or activity) and extrinsic factors (e.g., how the material is presented). Cognitive overload occurs when the demands or load of a given task (intrinsic and extrinsic) exceed an individual's available cognitive resources, which can lead to poor performance and/or limited learning. When cognitive resources are spread too thin, an individual may struggle to keep track of all the necessary information and/or allocate sufficient mental effort to the critical elements of the task, which leads to mistakes, slower processing, and difficulty organizing, analyzing, or synthesizing information.

According to cognitive load theory, an individual's working memory capacity can be a "bottleneck" for learning/cognitive performance, such that weaknesses in working memory may increase susceptibility to cognitive overload and have widespread effects on cognitive task performances, particularly in the context of high task demands (Sweller, 1988; Schnotz & Kürschner, 2007; Van Merriënboer & Sweller, 2005). Given the increased risk of working memory problems in children with NF1 (Beaussart et al., 2018; Smith et al., 2020), they may be particularly vulnerable to cognitive overload and the associated widespread impact on cognition and learning. Indeed, a prior study showed that adolescents with NF1 demonstrate a greater decrement in performance across a variety of EF tasks as the cognitive demands increase (i.e., greater inhibitory control, cognitive flexibility, and working memory) compared to healthy age and gender-matched controls (Rowbotham et al., 2009).

To our knowledge, cognitive load has not yet been directly studied in youth with NF1. Studies of children with ADHD, which is highly prevalent in NF1, have shown that increased cognitive load impacts other aspects of EF. Specifically, under higher cognitive load, children with ADHD show greater difficulties with response inhibition (Martinelli et al., 2017; Seymour et al., 2016) and delay discounting (Martinelli et al., 2017) compared to typically developing peers.

The current study aims to extend the current literature on EF in youth with NF1 by further investigating the impact of cognitive load on performance for children with NF1 compared to typically developing children. We hypothesize that an increase in cognitive load will have a greater negative impact on performance accuracy and speed in children with NF1 compared to the comparison group.

Method

Participants and procedures

Study participants were recruited from two primary children's hospitals' Neurofibromatosis Clinics, one in the U.S. and one in Australia. All research procedures were completed in accordance with the Helsinki Declaration, and the protocol was approved by the institutional review board for human protections at Children's National Hospital and Murdoch Children's Research Institute.

Parents and children aged 7 to 18 diagnosed with NF1 were invited to participate at each center. Exclusion criteria included the presence of any significant neurological comorbidity including central nervous system tumor requiring current treatment, seizures, and any sensory impairment that would interfere with the patient's ability to complete the computerized task or neuropsychological assessment. Parents engaged in informed consent and children engaged in assent. Participants completed a single study visit involving a brief neurocognitive evaluation of the child including an intellectual screener (Wechsler Abbreviated Scale of Intelligence–Second Edition (WASI-II) Wechsler, 2011) and the Test of Executive Control (TEC) (Isquith et al, 2006). The participant's parent or primary guardian completed a parent rating of EF difficulties (Behavior Rating Inventory of Executive Function; BRIEF (Gioia et al., 2000). A total of 48 children with NF1 were enrolled in the study (US $n = 14$; AUS $n = 34$). Six children with NF1 were excluded due to validity concerns (i.e., performance on the TEC which flagged embedded validity indices), none were excluded based on criteria noted above, resulting in a final sample of 42 children with NF1. Three participants had diagnosed brain tumors with previous treatment but were included as they were not on current treatment and tumors and neurological status was stable. No participants had a history of seizures, and no patients were excluded for sensory impairment precluding participation. The control sample was drawn from the normative dataset for the TEC ($n = 42$) and as such, no additional measures were available for comparison. The control sample was matched based on age and sex with the NF1 group. Sample demographics are presented in Table 1.

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Table 1. Sample characteristics

		NF1		Controls	
Sex		*n*	%	*n*	%
	Female	24	57.1%	24	57.1%
	Male	18	42.9%	18	42.9%
Race		n	%	n	%
	Asian	3	7.1%	0	0.0%
	Black	0	0.0%	1	2.4%
	Biracial	1	2.4%	1	2.4%
	Latino	0	0.0%	4	9.5%
	Native American	0	0.0%	1	2.4%
	White	38	90.5%	35	83.3%
		M	*SD*	*M*	*SD*
Age		11.19	3.11	11.19	3.11
Cognitive functioning		*M*	*SD*	*n/a*	
WASI full scale IQ (*n* = 38)		91.37	12.15	*n/a*	
WASI verbal reasoning (*n* = 38)		94.05	12.28	*n/a*	
WASI perceptual reasoning (*n* = 38)		91.50	12.80	*n/a*	
BRIEF general executive composite		64.48	12.49	*n/a*	
BRIEF Behavioral Regulation Index		60.48	14.14	*n/a*	
BRIEF metacognition index		65.38	11.68	*n/a*	
BRIEF working memory scale		64.05	12.00	*n/a*	
BRIEF inhibit scale		60.31	14.62	*n/a*	

Notes: NF1 = Neurofibromatosis Type 1, WASI = Wechsler Abbreviated Scale of Intelligence, BRIEF = Behavior Rating Inventory of Executive Function.
WASI Standard Scores have a mean of 100 and SD of 15; BRIEF T-Scores have a mean of 50 and SD of 10. Higher scores on the BRIEF indicate greater problems. NF1 and control groups were matched by age and sex. IQ and BRIEF data were available for the NF1 group only.

Measurement of cognitive load

Performance-based measurement of cognitive load is based on secondary task performance methodology, in which the cognitive load of a primary task is reflected by the performance on a concurrent secondary task (Paas et al., 2003). In other words, when an individual completes two concurrent cognitive tasks, they experience higher cognitive load as primary task demands increase, resulting in worsening performance on the secondary task. In the current study, cognitive load was assessed using the TEC, a standardized, computer administered test, which combines *n*-back (primary, working memory task) and go/no-go (secondary, inhibitory control task) paradigms (Isquith et al, 2006). The participants were seated at a computer where they are presented with a sequence of visual stimuli (e.g., pictures of common objects with low verbal demand). A keyboard with a red and a blue response button was provided. Participants were trained to respond as quickly as possible without making an error. Participants were instructed to press the blue key for all stimuli (standard stimuli) except the identified target stimuli, for which they were to press the red key. Blue key responses (frequent) are called Standard responses while red key responses (infrequent) are called Target responses. During the primary *n*-back task, participants are presented with a sequence of visual stimuli (e.g., pictures of common objects with low verbal demand) and asked to indicate whether the currently presented stimulus matches the one presented *n* steps earlier in the sequence. The TEC *n*-back includes levels of increasing working memory load, including 0-back (e.g., identify a target that matches an established category) and 1-back (e.g., identify when a target matches the one that came immediately before it). For older children (>8 years), a third level is included in the task (2-back; identify when a target matches the one that came

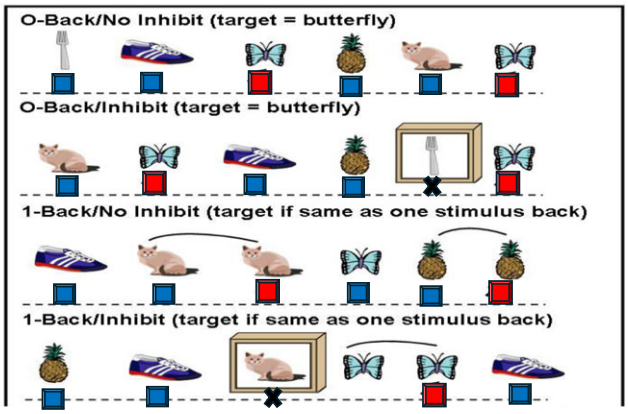


Figure 1. *Illustration of TEC tasks.*

two pictures before it). The current study limited analysis to the two levels of working memory load administered to all age ranges (0-back and 1-back). The secondary go/no-go task consists of presence or absence of an inhibitory signal added to the *n*-back paradigm, such that participants respond as usual to the *n*-back when the signal is absent, and do not provide any response (e.g., inhibit) when the signal is present. Inhibition signals only occur in the Standard (blue button) trials. The primary n-back and secondary go/no-go tasks are completed simultaneously. Tasks are fully crossed to manipulate working memory (e.g., 0, 1-back) and inhibitory control (e.g., absence/presence of inhibitory signal). The test yields 4 tasks presented in a fixed sequence (see Figure 1): 0B (0-Back), 0 BI (0-Back with inhibit demand), 1B (1-Back), and 1 BI (1-Back with inhibit demand). The n-back tasks require the participant to respond to frequent (i.e., Standard) stimuli and infrequent (i.e., Target) stimuli. This constant response demand establishes a prepotent response set, making inhibition on the no-go inhibitory cue more challenging (Isquith et al, 2006).

The primary outcomes include response accuracy (e.g., correct responses, commission errors), speed (e.g., average response time [mean]), and consistency (e.g., overall variability in response time [standard deviation], variability in response time across tasks [inter-condition variability]). T-scores, determined relative to the normative sample, are presented. Higher T scores indicate worse performance. A T-Score of 60 or higher is considered clinically elevated on the TEC (Isquith et al, 2006).

Statistical analysis

Statistical analyses were conducted using SPSS v30.0. First, group differences in overall TEC performance were examined via a general linear model (GLM), which included the between-subjects effects (NF1 vs controls) for each of the TEC summary scores representing overall response accuracy (e.g., correct responses, commission errors), speed (e.g., average response time [mean]), and consistency (e.g., overall variability in response time [standard deviation], variability in response time across tasks [inter-condition variability]). A comparison of the rate of clinically elevated TEC summary T-scores across youth with NF1 and healthy controls was also conducted using a Chi-Square test.

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Table 2. Interaction of working memory, inhibition, and group effects on response accuracy

	Target correct			Standard correct			Commissions		
	F (1,82)	*p-value*	ηp^2	*F (1,82)*	*p-value*	ηp^2	*F (1,82)*	*p-value*	ηp^2
Inhibition	1.74	0.191	0.021	**4.475**	**0.037**	**0.052**	n/a	n/a	n/a
Working memory	0.839	0.362	0.010	0.113	0.738	0.001	**5.121**	**0.026**	**0.059**
Inhibition*Working memory	2.916	0.091	0.034	0.063	0.803	0.001	n/a	n/a	n/a
Group	**5.103**	**0.008**	**0.076**	**7.576**	**0.007**	**0.085**	**8.552**	**0.004**	**0.094**
Inhibition*Group	0.094	0.760	0.001	2.215	0.141	0.026	n/a	n/a	n/a
Working Memory*Group	1.461	0.230	0.018	**6.288**	**0.014**	**0.071**	0.239	0.626	0.003
Inhibition*Working Memory*Group	0.004	0.952	0.000	0.033	0.856	0.000	n/a	n/a	n/a

Note: Partial-Eta Squared (ηp^2) effect sizes are interpreted as follows: 0.01 = small, 0.06 = medium, >0.14 = large. Commission errors were scored only for trials with no inhibitory control demand – thus the within-subject effects for working memory load (but not inhibitory control) are presented.

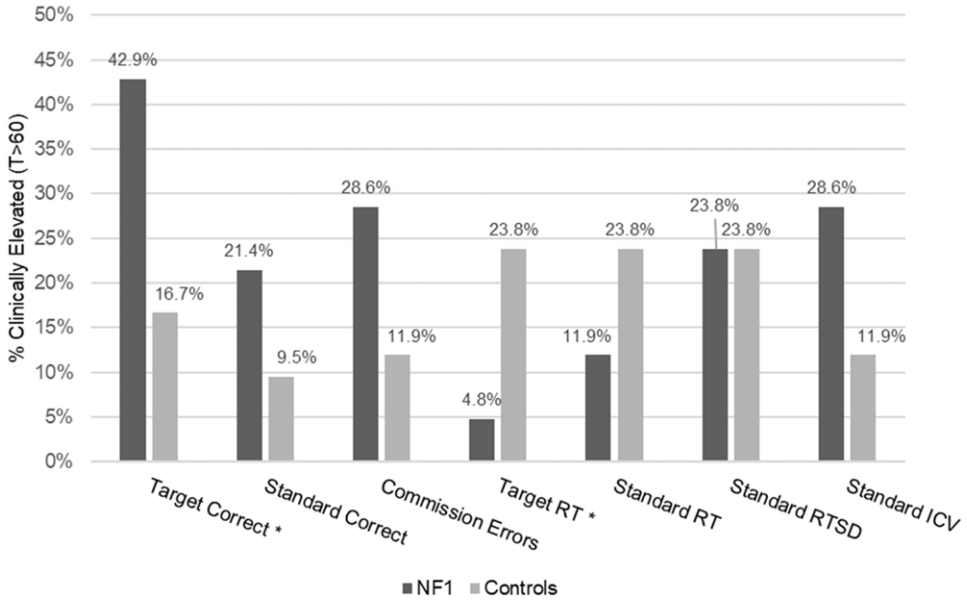


Figure 2. Clinically elevated scores on the TEC in youth with NF1 vs. controls. *Note:**Significant difference between groups (p<0.05).

Next, a single mixed design (repeated measures and between-subjects factors) General Linear Model was used to examine the within-subject effects of working memory (comparison between 0-back and 1-back tasks) and inhibition (comparison between n-back only and n-back with inhibition). The model also included analysis of the interaction between these tasks. Specifically, the interactions of between-subject factors (group) and within-subject factors (working memory, inhibition) were included to examine whether the effect of within-subject factors (working memory, inhibitory control) on task performance (target response accuracy and speed, standard response accuracy and speed, and standard response consistency) differed across youth with NF1 and healthy controls.

In addition to the primary multivariate repeated-measures analysis, we conducted supplementary univariate repeated-measures ANOVAs to examine between-group differences in working memory load and inhibition load. Estimated marginal means were compared using Bonferroni-adjusted pairwise comparisons.

Pearson product-moment correlation was conducted using the BRIEF Global Executive Composite (GEC) to explore associations between TEC performance and reported EF on the BRIEF Parent report in the NF1 patients only.

Results

Overall task performance (between-subject effects; Table 2)

Youth with NF1 showed reduced accuracy overall compared to controls. Specifically, they had fewer correct responses collapsed across standard and target trials and made more impulsive errors (commission errors). On average, response speed or variability did not significantly differ across groups (response time mean, response time standard deviation). However, youth with NF1 showed more variability in their response time across tasks (inter-condition variability). A significantly higher percentage of youth with NF1 exhibited deficits in target correct responses than controls while at the same time a significantly lower percentage of youth with NF1 showed deficits in target response time (See Figure 2).

Impact of cognitive load: multivariate analysis

Multivariate effects of inhibition and working memory

The primary multivariate repeated-measures analysis revealed significant main effects of inhibition (p = .018) and working memory (p = .008), as well as a significant inhibition × group

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Table 3. Interaction of working memory, inhibition, and group effects on response time (RT)

	Target RT			Standard RT		
	F (1,82)	*p-value*	*ηp²*	*F (1,82)*	*p-value*	*ηp²*
Inhibition	0.011	0.918	0.000	**5.276**	**0.024**	**0.060**
Working Memory	1.509	0.223	0.018	0.512	0.476	0.006
Inhibition*Working Memory	2.023	0.159	0.024	0.001	0.605	0.000
Group	0.479	0.491	0.006	0.004	0.95	0.000
Inhibition*Group	**4.781**	**0.032**	**0.055**	1.688	0.197	0.020
Working Memory*Group	0.436	0.511	0.005	0.012	0.913	0.000
Inhibition*Working Memory*Group	0.152	0.698	0.002	0.171	0.68	0.002

Note: Partial-Eta Squared (ηp^2) effect sizes are interpreted as follows: 0.01 = small, 0.06 = medium, >0.14 = large.

Table 4. Interaction of working memory, inhibition, and group effects on standard response time variability

	Standard RTSD			Standard ICV		
	F (1,82)	*p-value*	*ηp²*	*F (1,82)*	*p-value*	*ηp²*
Inhibition	0.275	0.601	0.003	0.894	0.347	0.011
Working Memory	0.402	0.528	0.005	2.99	0.088	0.035
Inhibition*Working Memory	0.269	0.605	0.003	0.003	0.957	0.000
Group	2.134	0.148	0.025	**6.159**	**0.015**	**0.070**
Inhibition*Group	**5.396**	**0.023**	**0.062**	1.685	0.198	0.020
Working Memory*Group	0.105	0.747	0.001	0.025	0.875	0.000
Inhibition*Working Memory*Group	0.747	0.39	0.009	0.355	0.553	0.004

Note: Partial-Eta Squared (ηp^2) effect sizes are interpreted as follows: 0.01 = small, 0.06 = medium, >0.14 = large.

interaction ($p = .015$, $\eta^2 p = .144$). This indicates that the effect of inhibition varied by group across multiple cognitive measures. The working memory × group interaction was also significant ($p = .040$), suggesting group differences were most pronounced under higher working memory load. These findings support a multi-component impact of cognitive load on executive functioning in NF1 (see Tables 2–4, Figures 3–5).

Supplementary univariate analyses

Analysis of working memory load effects on accuracy revealed a significant WM × group interaction ($F(1,82) = 4.34$, $p = .040$, $\eta^2 p = .050$). NF1 participants performed significantly worse than controls under 1-back conditions (mean difference = 6.38, $p = .004$), whereas no significant difference was observed under 0-back conditions ($p = .341$). The main effect of group was also significant ($F(1,82) = 5.27$, $p = .024$, $\eta^2 p = .060$), with NF1 participants showing worse performance compared to controls.

A univariate repeated- measures ANOVA focused on Target RT did not yield a significant inhibition × group interaction ($F(1,82) = 2.30$, $p = .134$, $\eta^2 p = .027$). Pairwise comparisons showed no significant differences between NF1 and controls in RT under either inhibition or no-inhibition (all $p > .40$). This suggests that the multivariate interaction was not driven solely by RT performance.

Analysis of Response Time Standard Deviation (RTSD) revealed a significant inhibition × group interaction ($F(1,82) = 4.81$, $p = .031$, $\eta^2 p = .055$). NF1 participants had significantly higher RTSD than controls under inhibition (mean difference = 4.52, $p = .040$), whereas no significant difference was observed under no-inhibition conditions ($p = 800$). The main effect of group was not significant ($F(1,82) = 1.46$, $p = .231$, $\eta^2 p = .017$).

Simple effects and pairwise comparisons

A significant difference was found between the NF1 and control group on 1-Back Standard Response Accuracy with and without inhibition ($F(1,82) = 8.602$, $p = .004$, $\eta^2 = .095$; $F(1,82) = 8.692$, $p = .004$, $\eta^2 = .096$, respectively). There were no simple effects differences between the groups on 0-Back standard response accuracy (Figure 3). Regarding Target Response Time, there were no statistically significant differences between groups for either level of working memory (Figure 4). A statistically significant group difference was found for 0-Back Standard RTSD with inhibition ($F(1,82) = 4.34$, $p = .040$, $\eta^2 = .050$), with no other significant simple effects (Figure 5). All relationships were in the direction of NF1 participants performing worse than controls.

Association with real-world executive dysfunction

A full examination of the association between performance on the TEC and real-world executive dysfunction was beyond the scope of this study. However, we did explore the relationship between performance on the TEC and parent-rated executive functioning on the BRIEF-2 for the participants with NF1. Our analysis showed a significant positive relationship between the BRIEF-2 Global Executive Composite (GEC) and the TEC Target Correct ($r2 = .319$, $p = 0.39$), Standard Correct ($r2 = .348$, $p = .024$), and Standard Intraindividual Coefficient of Variation (ICV) ($r2 = .309$, $p = .046$), supporting the clinical relevance of laboratory-based cognitive load effects. There was no significant relationship between the GEC and Commission Errors on the TEC ($r2 = .101$, $p = .493$).

Conclusions

This study demonstrates that children with NF1 experience unique and pronounced decrements in executive function performance in response to increased cognitive load compared to typically developing peers. Specifically, as working memory and inhibitory demands increase, children with NF1 show declines in accuracy, consistency, and efficiency, whereas controls maintain or improve their performance under similar conditions. These findings extend the previous work by Rowbotham et al. (2009), highlighting that increased cognitive load disproportionately impacts children with NF1.

The observed deficits in NF1 appear to be complex as the children with NF1 appear less able to flexibly adapt to increased inhibitory and working memory demands, as evidenced by slower and more inconsistent responses to infrequent stimuli under higher inhibitory load and poorer accuracy with higher working memory load (Beaussart et al., 2018; Smith et al., 2020). This aligns with prior reports of executive dysfunction in NF1 and supports cognitive load theory, which posits that working memory capacity is a bottleneck for learning and performance (Schnotz & Kürschner, 2007; Sweller, 1988).

While typically developing children may benefit from increased cognitive load, children with NF1 are more likely to experience performance decrements, underscoring their vulnerability to cognitive load. This distinction highlights the need for tailored interventions that specifically address cognitive overload in NF1, as

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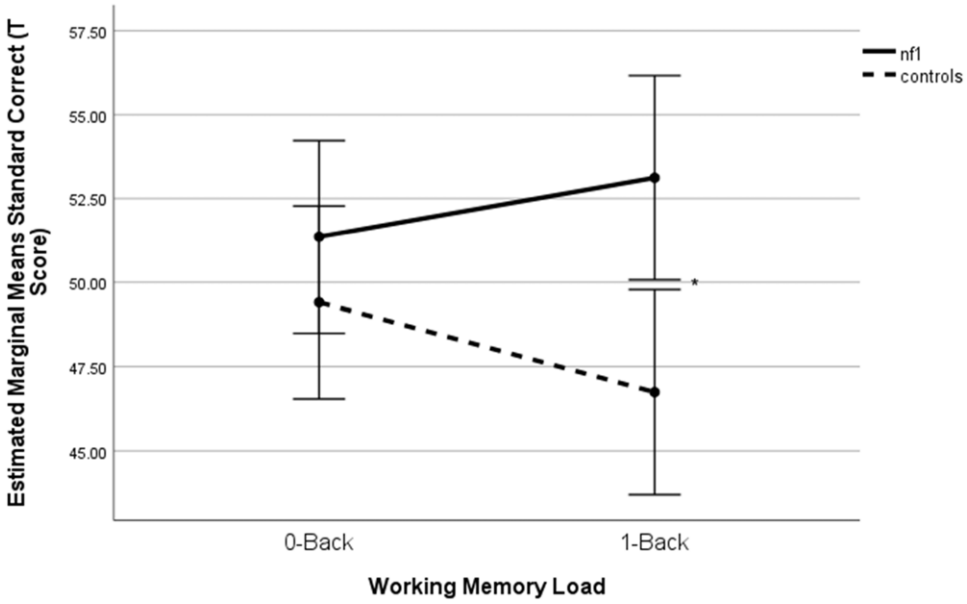


Figure 3. Working memory × group interaction on standard response accuracy[+]. *Notes.*[+]Higher scores denote poorer performance. *Note:* $^{*}p < .01$.

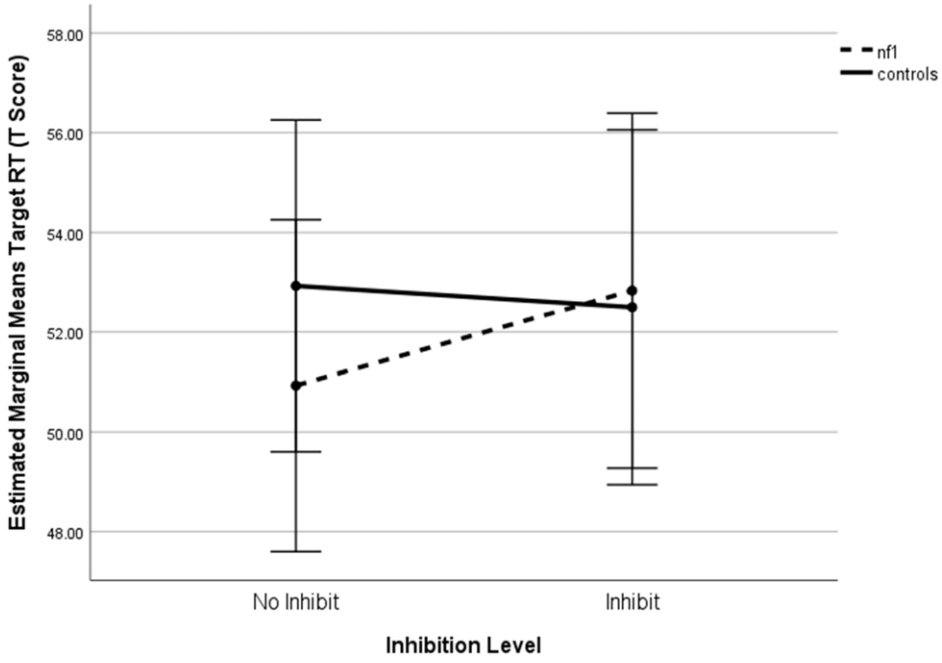


Figure 4. Inhibition × group interaction on target response time[+]. *Notes.*[+]Higher scores denote poorer performance.

advocated in educational research applying cognitive load theory (Castro-Alonso et al., 2021).

The significant associations between laboratory-based cognitive load effects and parent-reported executive dysfunction in NF1 provide preliminary evidence that these deficits may translate to everyday functioning. The underscores the clinical relevance of targeting working memory and inhibitory control in intervention for NF1 (Hardy et al., 2021; Hou et al., 2023).

The educational implications are substantial given the high incidence of learning problems in NF1 (Hyman et al., 2006; Torres et al., 2017). Working memory is strongly associated with academic achievement (Spiegel et al., 2021), and children with NF1 exhibit the most significant impairments and morbidity in this domain (Beaussart et al., 2018). These impairments are linked to deficits in reading, writing, and math (Hou et al., 2023) and worsen with age (Hou et al., 2020). Instructional techniques that minimize cognitive overload and promote self-directed strategies may be particularly useful for this population (Castro-Alonso et al., 2021; Schnotz & Kürschner, 2007).

Beyond academics, impaired response to cognitive load may also affect social functioning. Evidence from ADHD literature implicates working memory, impulsivity, and inconsistent focus as contributors to social difficulties (Kofler et al., 2011; Kofler et al., 2018). Given the high rates of ADHD, executive dysfunction, and

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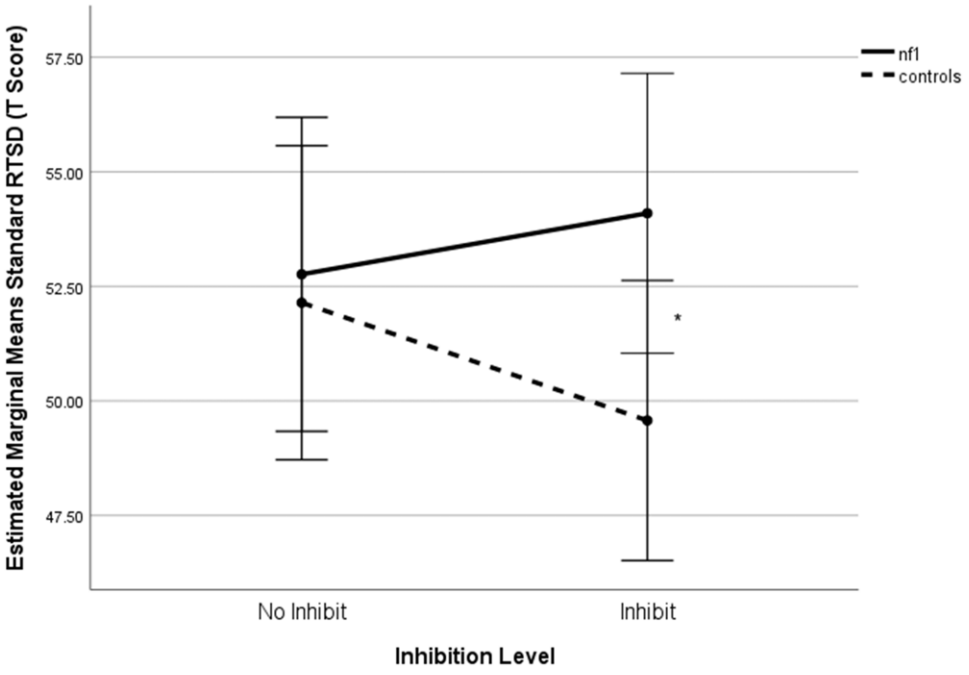


Figure 5. Inhibition × group interaction on standard response time standard deviation[+]. [+]Higher scores denote poorer performance. *Note:* $^{*}p < .05$.

social deficits in NF1, these impairments likely contribute to social challenges (Chisholm et al., 2018; Payne et al., 2020).

Limitations of this study include the lack of intellectual and EF data for the control group, modest and predominantly white sample, and cross-sectional design. Future research should evaluate the impact of cognitive load on daily life and academic functioning and explore physiological markers of cognitive load (Paas et al., 2003). Interventions targeting working memory, such as Cogmed, show promise for improving deficits in NF1 (Hardy et al., 2021), and combining these with broader EF strategies may further enhance learning and social development.

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