A Diffusion Decision Model Analysis of The Cognitive Effects of Neurofeedback Treatment For ADHD

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Citation

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Abstract

Objective: To examine cognitive effects of neurofeedback (NF) for attention-deficit hyperactivity disorder (ADHD) as a secondary outcome of a randomized clinical trial (RCT). **Method:** In a double-blind RCT (NCT02251743), 133 7-10-year-olds with ADHD received either 38 sessions of NF (n=78) or control treatment (n=55); and performed an Integrated Visual and Auditory Continuous Performance Test (IVA2-CPT) at baseline, mid- and end-treatment. We used the diffusion decision model (DDM) to decompose IVA2-CPT performance at each assessment into cognitive components: efficiency of integrating stimulus information (v), context sensitivity (c_v), response cautiousness (a), response bias (z/a), and non-decision time for perceptual encoding and response execution (T_{er}). Based on prior findings, we tested whether the components known to be deficient improved with NF and explored whether other cognitive components improved using linear mixed modeling. **Results:** Before NF, children with ADHD showed main deficits in integrating stimulus information (v) which led to less accurate and slower responses than healthy controls (p=0.008). The NF group showed significantly more improvement in integrating auditory stimulus information (v) than control treatment (significant group-by-time-by-modality effect: p=0.044). **Conclusion:** NF seems to improve *v*, deficient in ADHD.

Keywords: diffusion decision model, adhd, neurofeedback, asd, computational psychiatry

Key Points: This study suggests that neurofeedback improves underlying deficient cognitive processes in attention-deficit hyperactivity disorder. It highlights the importance of studying treatment effects on neurocognitive testing together with process-oriented computational modeling analyses.

Introduction

Attention-deficit hyperactivity disorder (ADHD) is a common neurodevelopmental disorder characterized by inattention and hyperactivity-impulsivity (DSM-5; American Psychiatric Association [APA], 2013). To date, the efficacy of pharmacological treatments for ADHD is limited (Kofler et al., 2017; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). For instance, a third of individuals diagnosed with ADHD are non-responders to the first stimulant tried, and up to 10% are non-responders to any pharmacological treatments (Adler, Reingold, Morrill, and Wilens, 2006; Bhandary et al., 1997). Therefore, a persistent need exists for alternative, non-pharmacological, treatments.

Neurofeedback treatment (NF) has been considered a promising alternative or complementary therapy for ADHD (e.g., Arns, Heinrich, and Strehl, 2014; Sherlin, Arns, Lubar, and Sokhadze, 2010). In NF, individuals learn to modify their brain activity (i.e., power of specific bandwidths measured with an electroencephalogram [EEG]; for a review see Sherlin et al., 2010). To date, it is unclear how NF affects an individual's cognition. For instance, which, if any, underlying cognitive components (e.g., relative emphasis of fast versus accurate decisions, tendency towards premature decisions, efficiency of integrating presented information) change in response to NF? Are these cognitive components also those that are characteristically deficient in ADHD? Are improvements also noticeable in cognitive tasks that are not part of the NF training? Addressing these questions helps to build an understanding about NF mechanisms. This understanding is important to personalize treatment. For instance, existing treatment procedures can be refined to focus on the underlying components that are deficient and that need to be targeted.

This study comprises a secondary analysis of one of the largest double-blind randomized clinical trials (RCT) on NF treatment (The Neurofeedback Collaborative Group, 2021). The RCT

did not find a significant overall difference on the primary outcome (parent- & teacher-rated inattentiveness) between those who receive NF and those who received control treatment that differed only in not having deliberate down-training of the theta-beta power ratio (TBR). Specifically, both groups had large significant inattentive symptom improvements that were sustained for 21 months following treatment (The Neurofeedback Collaborative Group, 2023), without a significant difference between treatments. However, moderator analyses showed significant effects in comorbidity subgroups; at treatment end, those with comorbid anxiety without oppositional-defiant disorder (ODD) improved significantly more with the control treatment than with NF, and at 13-month follow-up, those with ODD without anxiety were significantly more improved with NF than control treatment (Roley-Roberts et al., 2022). Moreover, baseline cognitive signatures, quantified by applying computational modeling to the baseline neurocognitive data, indicated children who benefitted from receiving NF treatment (Ging-Jehli et al., 2023). Due to these primary and secondary findings and given the circumstance that NF can be seen as a reinforcement learning intervention (Lubianiker, Paret, Dayan, & Hendler, 2022), it is critical to understand the cognitive mechanisms of action for personalized treatment planning.

Continuous performance tests (CPTs), also known as go/no-go tasks, have been among the most popular assessments of the cognitive characteristics of ADHD (for review see: Ging-Jehli, Ratcliff, & Arnold, 2021; see also: Gomez, Ratcliff, & Perea, 2007; Huang-Pollock, Karalunas, Tam, & Moore, 2012; Ratcliff, Huang-Pollock, & McKoon, 2018). In classical CPT, a series of go trials and no-go trials is presented. Participants are instructed to respond to go trials by key presses and to respond to no-go trials by withholding key presses. There are two types of CPTs that differ in their proportion of go versus no-go trials (Edwards et al., 2007). The CPT version

with frequent go trials (e.g., 10% no-go trials) is presumed to tap into cognitive concepts such as "impulsivity" (Conners, 2002; Parsons, Duffield, & Asbee, 2019). The CPT version with rare go trials (e.g., 90% no-go trials) is presumed to tap into cognitive concepts such as "sustained attention" or vigilance (e.g., Robertson et al., 1997). Many studies used CPTs to test for performance (i.e., accuracy or mean reaction times [RTs] or both) differences between individuals with and without ADHD (for review see Ging-Jehli et al., 2021; see also: Huang-Pollock, Nigg, & Halperin, 2006; Sergeant et al., 1999). They found that children and adults with ADHD have slower mean RTs, greater RT variability (and sometimes increased error rates) than those without ADHD. The underlying sources of slower mean RTs, which seem to represent an ADHD characteristic, remain inconclusive when using summary statistics (e.g., mean RTs). This is because multiple underlying mechanisms can lead to slower mean RTs: e.g., difficulty getting started with a task; difficulty processing information that is presented during the task; or more cautious response strategies to avoid mistakes.

Computational psychiatry utilizes modeling to better understand which underlying cognitive components are affected by mental-health disorders such as ADHD and to explain the underlying components that result in slower mean RTs (e.g., Ging-Jehli et al., 2021; Frank et al., 2016). The diffusion decision model (Ratcliff, 1978; henceforth DDM) is a commonly applied model that describes how people make decisions (for review see: Ging-Jehli et al., 2021). Below we briefly describe the DDM (for a detailed description and graphical illustration see Ging-Jehli et al., 2021).

The DDM provides a simplified blueprint of the decision-making process. Specifically, the DDM assumes that individuals gradually integrate increments of information about the presented stimulus until a threshold is reached. The decision is made when the threshold is reached,

producing a RT and an accuracy value. Compared to other computational models (e.g., models based on signal-detection theory, ex-Gaussian distribution models), the DDM simultaneously considers the frequency of each response option as well as their corresponding RT distributions (rather than only mean RTs). It decomposes performance into components (quantified by model parameters) that have established psychological interpretation and that can be studied separately: general response cautiousness (boundary separation, *a*), start of decision processes (starting point, *z*), the tendency towards premature decisions (starting point bias, *z/a*), the efficiency of integrating stimulus information (drift rate, *v*), context sensitivity (drift bias, c_v), and the time for task preparation, forming a neural representation, and response execution (nondecision time component, T_{er}) (Forstmann, Ratcliff, & Wagenmakers, 2016; Ratcliff, 1978; Ratcliff & McKoon, 2008). The DDM is attractive because it is a well-established model with clinical populations (Forstmann et al., 2016; Ging-Jehli, Arnold, Roley-Roberts, & DeBeus, 2022; Caulfield & Myers, 2018; Pe, Vandekerckhove, & Kuppens, 2013; Weigard & Sripada, 2021; Wiecki, Poland, & Frank, 2015; Zeguers et al., 2011), utilizes more information than conventional performance measures, and it is theoretically founded in decision theory that integrates our current understanding of brain dynamics and functioning (Cohen & Kohn, 2011; Forstmann, Ratcliff, & Wagenmakers, 2016; Gold & Shadlen, 2001; 2007; Hanes & Schall, 1996; Philiastides, Ratcliff, & Sajda, 2006; Ratcliff, Cherian, & Segraves, 2003; Wong, Huk, Shadlen, & Wang, 2007).

Research (see for a review: Ging-Jehli et al., 2021; Weigard & Sripada, 2021) that uses DDM analyses consistently found that children with ADHD have a poorer efficiency of integrating stimulus information (lower drift rate, *v*) than those without ADHD across different tasks intended to assess cognitive concepts including: cognitive flexibility (e.g., Metin et al., 2013; Salum et al., 2014); inhibitory control (e.g., Huang-Pollock, Ratcliff, & McKoon, 2017; Mowinckel et al.,

2015); selective attention (e.g., Merkt et al., 2013; Mulder et al., 2010; Weigard & Huang-Pollock, 2014); sustained attention (e.g., Huang-Pollock et al., 2012); time perception (e.g., Shapiro & Huang-Pollock, 2019); working memory (e.g., Weigard & Huang-Pollock, 2017); and reinforcement learning paradigm (e.g., Fosco, White, & Hawk, 2017). Moreover, recent DDM applications (Ging-Jehli et al., 2022) to the data of CPTs have shown that ADHD is not only associated with lower drift rates but also with deficient drift biases (c_v) and longer nondecision times (T_{er}). However, these differences were only detectable when considering comorbidities and DSM-defined presentations. Some researchers have already emphasized the importance of drift bias (c_v) and that it is a neglected parameter (Ging-Jehli et al., 2023; Starns et al., 2012; Kloosterman et al., 2019).

There is preliminary evidence (e.g., Ging-Jehli et al., 2022; Merkt et al., 2013; Salum et al., 2014) that the severity of ADHD symptoms is positively associated with lower drift rate (v). Studies that applied neurocognitive tests, together with a DDM analysis, examined the cognitive characteristics of ADHD by comparing performance between children with and without ADHD. However, we will show that the information from a DDM analysis could also be used to determine whether a therapeutic intervention, such as NF treatment, targets the cognitive components (e.g., drift rate, v) found to be deficient in ADHD.

Present Study

The primary purpose of this DDM analysis was to assess whether low drift rate (v), a purported characteristic of ADHD in prior research (Ging-Jehli et al., 2021; 2022; see also: Sripada & Weigard, 2021), improves in response to NF treatment for ADHD. Specifically, the hypothesis was that *phi v* (introduced in the Method section: Diffusion Decision Model, subsection: Model parameters under examination) would improve significantly more with NF than with control

treatment. To show that *phi v* was indeed a dysfunctional cognitive component in the ADHD sample before the start of NF treatment, we compared the baseline (BL) data with the data of a healthy sample (children without ADHD). A more thorough comparison of the cognitive differences between children with and without ADHD (including an accountant of comorbidities and DSM-defined presentations) can be found in Ging-Jehli et al. (2022). Specifically, Ging-Jehli et al. also found drift biases (c_v) and starting point biases (z/a) that depended on DSM-defined presentations and comorbidities, respectively. We therefore also tested in secondary analyses the effects of NF on these other model parameters. To our knowledge, this is the first study that used such a computational model-based analysis to quantify the cognitive effects of NF treatment for ADHD on underlying cognitive components.

Methods

Transparency and Openness

We report all data exclusions (if any), all manipulations, and all measures in this study and the primary study (The Neurofeedback Collaborative Group, 2021). Additional data is available in the Supplemental Material and all data has already or will be uploaded to NDAR as part of the RCT. The primary analysis of this RCT was pre-registered (RCT; NCT02251743). However, secondary analyses were not pre-registered. All analyses were conducted using SAS (v9.4, SAS Inst. Inc, NC) and R (v4.0, R Core Team, 2017).

Participants

This report examines how NF treatment for ADHD affects underlying cognitive components. Another secondary study of this RCT already compared the baseline cognitive characteristics of this ADHD sample to those of the healthy control sample (Ging-Jehli et al., 2022). However, that study did so by focusing on comorbidities and DSM-defined presentations

rather than the entire ADHD sample. We therefore report group-specific differences between the entire ADHD sample (before the start of the treatment) and the healthy controls in the Supplement. We did so to independently establish for this study whether NF improves deficient cognitive components.

ADHD sample. This study included 133 children with ADHD, aged 7 to 10, in the International Collaborative ADHD NF (ICAN) randomized clinical trial (RCT; NCT02251743) who completed the IVA2-CPT at baseline, mid-treatment, and end-treatment[1]. The primary objective of the ICAN RCT was to test for a specific effect of TBR electroencephalographic biofeedback for ADHD beyond any nonspecific benefits. In a double-blinded procedure, children with ADHD were randomized to either NF or the control treatment. The active treatment group obtained actual NF training to downregulate theta power and upregulate beta power. The control group received an identical-appearing treatment that did not deliberately reinforce theta decreases or beta increases of their brain wave power; instead, their reinforcements were based on a prerecorded EEG from another child (for details see: Neurofeedback Collaborative Group, 2021). To be included in the ICAN RCT, children had to be diagnosed with ADHD (either combined or inattentive presentation; based on the DSM-5; APA, 2013) by doctoral clinicians and the Children's Interview for Psychiatric Syndromes (Weller et al., 1999a; see Measures: ChIPS) and have a T-score of 65 or more on inattention ratings by both parent and teacher. They also had to have an IQ $\geq$ 80 (assessed with the Wechsler Abbreviated Scale of Intelligence; WASI, Wechsler, 1999), and an electroencephalographic TBR $\geq$ 4.5, and were required to stop medication five days

[1] Of the 144 participants who met the study criteria and who were therefore initially randomized into the ICAN study, we excluded 11 participants (2 participants prematurely opted out, 5 participants were uncooperative by aborting the IVA2-CPT or by not responding at all, 4 participants' data were lost due to computer crashes). Therefore, the modified intention to treat (ITT) participant pool included all randomized participants with at least one IVA2-CPT.

prior to each assessment. For full inclusion/exclusion criteria see The Collaborative Neurofeedback Group (2021).

Healthy Control sample. The healthy controls were 57 children without any mental-health disorders who completed the IVA2-CPT in one session. To be included, children had to meet the following criteria: no diagnosis of any DSM-5 defined disorders, no head injury with loss of consciousness, no current psychotherapy or physical or occupational therapy, and no medication for seizures. Inclusion criteria were assessed in a pre-screening telephone interview with parents by a master's-level graduate student. In addition, we administered a parent-rated Conners-3rd edition rating scale to screen for undiagnosed ADHD and symptom severity of inattention and hyperactivity-impulsivity (see Instruments: C-3:P).

Recruitment Procedure. This RCT was conducted at two sites, namely: a university in a midwestern metropolitan area of 1.5million people and a private neurofeedback clinic (in association with a local university) in a southeastern city of 100,000 people. Prospective participants (ADHD and control sample) were recruited through flyers and phone calls in local communities, schools, and through online recruiting platforms (e.g., researchmatch.org). Pre-screening telephone interviews were conducted to assess study eligibility.

Measures

Children's Interview for Psychiatric Syndromes -child (ChIPS) and -parent (P-ChIPS; Weller et al., 1999). The ChIPS/P-ChIPS is a structured diagnostic interview that was administered /reviewed at baseline by doctoral-level clinicians to assess the presence of 20 mental-health disorders defined by DSM-5 (APA, 2013). Specifically, the ChIPS/P-ChIPS screens for: ADHD, ODD, CD, Substance Abuse, Specific Phobia, Social Phobia, Separation Anxiety Disorder, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, Stress Disorders

[ASD/PTSD], Anorexia, Bulimia, Depression/Dysthymia [MDD/DD], Mania/Hypomania, Enuresis, Encopresis, and Schizophrenia/Psychosis.

Conners-3rd edition: Parent Report Long Version (C-3; Conners, 2008). This questionnaire was completed by parents and teachers of children with ADHD at baseline, mid- and end-treatment and by parents of control children at their one-time assessment. The questionnaire has 108 questions (11 related to hyperactivity-impulsivity and 10 related to inattentiveness) and item ratings range from 0 (no problem) to 3 (severe problems).

Demographic Questionnaire. All parents completed a demographic questionnaire at baseline. Table 1 provides an overview of the sample characteristics for each participant group (Healthy Controls (HC), ADHD Control treatment, ADHD NF).

The Integrated Visual and Auditory Continuous Performance Test (IVA2-CPT). The IVA2-CPT (Sandford & Turner, 2000) involves 500 trials (with additional 10 pre- and post-practice trials). Participants are presented with either the number "1" or the number "2." If a "1" is presented, participants are instructed to click the button of a computer mouse (go trials). If a "2" is presented, participants are instructed to withhold any button presses (no-go trials). The IVA2-CPT includes two types of blocks: blocks with frequent go trials (84% go trials, henceforth FreqGo) and those with rare go trials (16% go trials, henceforth RareGo). Moreover, the numbers are either visually or auditorily presented. Therefore, the IVA2-CPT involves eight conditions: two block types (FreqGo, RareGo) times two trial types (go trials, no-go trials) times two modality types (visual stimulus, auditory stimulus). RTs as well as accuracy are recorded for each trial. We provide additional details about the task in the online Supplement.

Study Procedure

Both the healthy control research study and the ICAN RCT were approved by The Ohio State University Institutional Review Board (IRB), and written informed consent and assent were obtained. Parents of children without ADHD completed the C-3 questionnaire (among others), while children performed the IVA2-CPT on a computer in a separate room. A research assistant accompanied the child in the separate room. Instructions on the IVA2-CPT were standardized and computerized (Sandford & Turner, 2000). IVA2-CPT, and other measures outlined previously were utilized from the ICAN RCT for the present study.

Diffusion Decision Modeling (DDM)

Model Specification. We used the same model specification as in Ging-Jehli et al. (2022) to allow for comparisons across studies (for details see also Supplemental Material). To fit the DDM to the IVA2-CPT data, we parameterized the model as follows: two starting point biases (one for each block type [FreqGo, RareGo]); one boundary separation (one combined for all eight conditions); eight drift rates (one for each stimulus type [go, no-go] by each modality [visual, auditory] by each block type [FreqGo, RareGo]); and four nondecision time components (one for each modality by each block type). We provide additional details in the Supplement.

Model Parameters Under Examination. The DDM includes the following four main parameters: drift rate (v), drift criterion (cv), nondecision time component (T_{er}), response cautiousness (a) and response bias (z/a). Past research consistently showed (across a range of cognitive tasks) that faster and more accurate processing of information are associated with larger drift rates (for a review see: Ging-Jehli et al., 2021). To focus on how information processing is affected by changes in modality (visual vs. auditory) and by changes in target frequency (frequent vs. rare go trials), we focused on two transformed measures of drift rate and drift criterion

subsequently referred to as *phi v* and c_v. *Phi v* (also known as drift criterion; Ging-Jehli et al., 2022; White, Skokin, Carlos, & Weaver, 2016) represents the summed drift rate of go and no-go trials for each block type and each modality[2]. The larger *phi v*, the better (i.e., faster and more accurate) information processing. Hence, *phi v* indexes the general *efficiency of processes associated with integrating stimulus information irrespective of the stimulus type*. Moreover, the IVA2-CPT represents a go/no-go task which means that participants who are continuously responding "Go" to all trials accomplish high accuracy on go trials (large drift rates for go trials) but low accuracy on no-go trials (low drift rates for no-go trials). Therefore, we calculated the difference between drift rates for go and no-go trials for each block type and for each modality (subsequently referred to as c_v). The higher c_v, the more information processing is dependent upon the stimulus type. Hence, we refer to c_v as a measure for *context sensitivity of processes involved with information integration* (with c_v = zero representing the optimal level). In so doing, we follow the practice of recent studies (Ging-Jehli et al., 2022; 2023).

Fitting Procedure. We estimated a separate set of model parameters for each child for each assessment (baseline, mid- and end-treatment). For the controls we only had baseline data. The DDM was fit to the data by using a standard method procedure (Ratcliff, Huang-Pollock, & McKoon, 2018; Ratcliff & Tuerlinckx, 2002) in which model parameters were iteratively adjusted to minimize a chi-square value.

Goodness Of Fit. We assessed goodness of fits in two ways (as suggested by Ging-Jehli et al., 2021) and we discuss them in detail in the Supplemental Material. First, the Supplemental

[2] It is common to represent drift rates for go trials with positive signs (as they represent diffusion processes towards the upper boundary) and drift rates for no-go trials with negative signs (as they represent diffusion processes towards the lower boundary). We therefore multiplied all drift rates for no-go trials by minus one before summing them (for phi v) or subtracting them from the drift rates for go trials (for c_v).

Table S1 shows the mean chi-square goodness of fit values and the degrees of freedom. All the chi-square values were below the critical chi-square values, which suggests that the model fit the data well. Moreover, the fits are quite reasonable compared to previous studies that applied the DDM to other CPTs (e.g., Ratcliff et al., 2018; Gomez et al., 2007). Second, Supplemental Figure S1 illustrate plots of model predictions against data for response proportions (i.e., accuracy) and the .1, .5, and .9 quantile RTs for each condition (averaged over participants and assessment points). Supplemental Figure S1 supports that the model fit the data well.

Statistical Analysis

All analyses were conducted using SAS (v9.4, SAS Inst. Inc, NC) and R (v4.0, R Core Team, 2017). Following an "intention to treat" analysis, we used violin plots of main DDM parameters (Supplemental Figure S2) to confirm that the ADHD population had deficits in specific cognitive components at baseline. To test for statistical significance between the healthy controls and the ADHD group, we performed an analysis of variance (ANOVA) for *phi v* after accounting for gender and age differences. The ADHD treatment-assignment group represented the between-subject factor. For the key analysis (i.e., investigating the effects of NF treatment on the efficiency of information processing), we estimated a linear mixed model (LMM) for *phi v.* For other secondary analyses (i.e., investigating the effects of NF treatment on other cognitive components that seemed to change over time), we estimated separate linear mixed models (LMM) for c_v and T_{er}. We did not run LMM on the other two DDM parameters (*a*, *z/a*) since the values did not change much over time (Table 2). We used LMMs to account for repeated measures and missing data. Due to repeated measurements, the error terms within each subject were correlated. Therefore, we specified an unstructured covariance matrix for the residual errors. For the LMMs, the following terms served as fixed effects: time, group, block type, modality, and the interaction of i) time by group; ii) time by group by block type; iii) time by group by modality. For each

participant, we included a separate intercept as a random effect. We used the Kenward-Roger Degrees of Freedom Approximation to adjust for multiple tests within each LMM.

We also performed follow-up analyses by calculating (for each treatment group separately) the changes of the two main model parameters (v, c_v), introduced in the Introduction (subsection: Present Study), for: i) baseline to mid-treatment; ii) mid- to end-treatment; and iii) baseline to end-treatment. We then compared the treatment effect by comparing the change of the control group to those of the NF group (using hedges' g for reporting effect sizes).

Results

Table 1 summarizes the sample characteristics of the three participant groups (healthy controls, ADHD control treatment, and ADHD NF). We did not find any statistically significant differences between the ADHD control treatment and NF groups on any demographic or clinical characteristics at baseline. However, the healthy controls were on average one year older and included significantly more females than each of the two ADHD groups.

Which cognitive components are deficient in ADHD before treatment?

To identify the cognitive components deficient in ADHD, we compared the baseline data of children with ADHD with those of healthy controls (Table 1). The data in Table 1 come from another secondary study of this RCT that has already examined cognitive similarities and differences between the healthy controls and the ADDH sample at baseline (Ging-Jehli et al., 2022). However, that prior study decomposed behavior additionally by comorbidities and DSM-defined presentations. We therefore independently provide all model parameters averaged across the entire ADHD sample in the Supplemental Figure S1, which illustrates that children with ADHD had significantly poorer efficiency of information integration (lower *phi v*) than the controls (M_{ADHD}=0.196, SD=0.091; $M_{controls}$=0.261, SD=0.114; 95% CI [0.031, 0.098],

df(1,185)=7.254, *p*=0.008). We did not find any other significant differences in main model parameters (c_v, T_{er}, *a*, *z/a*) between controls and the ADHD group (Supplemental Figure S1). However, we acknowledge that Ging-Jehli et al. (2022) found additional differences in other model parameters (c_v, *z/a*) when accounting for comorbidities and DSM-defined presentations.

Key Analysis (Primary analysis of this study): does NF treatment for ADHD improve the deficient component *phi v*?

The results from the LMMs suggested a significant time-by-group-by-modality fixed effect for *phi v* [*df*(5,1161)=2.29, *p*=0.044]. Specifically, the efficiency of processes associated with integrating *auditory* information of the ADHD NF group improved significantly more than that of the ADHD control treatment group. Figure 1 lists *phi v* for each modality, assessment point, and treatment group (see also Supplemental Figure S9).

Secondary analyses: does NF treatment for ADHD affect other cognitive components?

We also found a significant time-by-group-by-modality fixed effect for c_v [*df*(5,1161)=3.80, *p*=0.002]. Specifically, the context sensitivity of processes involved with auditory information integration of the ADHD NF group improved more than that of the ADHD control-treatment group (particularly when considering BL to treatment end). The ADHD NF group also improved on visual trials in blocks with rare go-trials. Figure 1 lists c_v for each modality, assessment point, and treatment group (see also Supplemental Figure S10).

Table 2 provides the values of other parameters (e.g., *a*, *z/a*, T_{er}) for each group at baseline, mid- and end-treatment. We found no significant group differences in how response cautiousness (*a*) and the tendency toward premature decisions (*z/a*) changed over time (all $p>0.05$). However, for the latency of processes involved in task preparation and response execution (T_{er}), we found a significant time-by-group interaction [*df*(2,129.3)=4.64, *p*=0.011]. Specifically, T_{er} remained stable over time for the ADHD control group, whereas it decreased for the ADHD NF group (Table

2). Due to the possibility that model parameters may trade-off against each other, we conducted a correlational analysis between model parameters. Changes in *phi v* across task conditions were not related to either changes in T_{er} across task conditions (r=-0.020, p<0.819) or, changes in c_v across task conditions (r=0.106, p<0.224). For sensitivity analyses, we also provide separate analyses for drift rates for go- and no-go trials in online Supplemental Tables S2 and S3. We also provide correlational analyses for all parameters and assessment points in the online Supplement.

Discussion

One way to find and tailor effective treatments is to build a deeper understanding of the effects of therapeutic interventions on cognition. To our knowledge, this is the first study that used computational modeling (i.e., DDM analysis) to study the effects of NF on distinct cognitive components. Our cognitive component correlational results also highlight the importance of studying cognitive components separately. Comparing the IVA2-CPT performance at baseline of children with ADHD to those of children without ADHD showed that the cognitive component most deficient in our ADHD sample was a poor integration of presented stimulus information (*phi v*). This result is consistent with prior research findings, and we refer for a more detailed analysis between healthy controls and different ADHD endophenotypes to Ging-Jehli et al. (2022).

We found support for our primary hypothesis, namely: *phi v* improved significantly more with NF than with double-blinded control treatment. Both primary and secondary analyses showed that NF treatment affects cognitive processing. Most importantly, cognitive processing was assessed in a cognitive task that was not part of the NF training. Moreover, NF improved the specific cognitive components identified to be deficient in ADHD. Compared to the ADHD control-treatment group, the ADHD NF group showed significant improvement in *phi v* and c_v, which led to faster and more accurate decisions, particularly on auditory trials. In contrast, the

ADHD control treatment group worsened in those components, especially from baseline to mid-treatment, with some recovery towards the baseline level by treatment end.

Our results further suggest that NF seems particularly to improve auditory processing. This is an interesting finding considering that auditory input is sequential, highly vulnerable to attention lapses, in contrast to visual input, which allows a "second look." Moreover, another secondary study of this RCT (i.e., Ging-Jehli et al., 2022) found that children with ADHD (particularly ADHD-Combined type) showed pronounced deficits in auditory processing compared to healthy controls. Hence, NF seems to exert benefits on cognitive components and sensory processing that seem deficient in ADHD. NF training improved the efficiency of integrating stimulus information to achieve faster and more accurate responses (as indexed by larger *phi v*); and made responses more consistent irrespective of the trial type (as indexed by lower c_v).

The "negative practice effect" for the ADHD placebo group in which *phi v* worsened from baseline to mid-treatment might be explained by boredom on repetition. Children with ADHD are motivated by novel situations but become bored and inattentive once novelty wears off. At baseline the IVA2 was novel; later assessments were "Been there; done that" situations. Interestingly, children in the ADHD NF group were nevertheless able to improve in information processing. Further studies are needed to identify whether cognitive components assessed at baseline could serve as predictors of therapeutic outcomes.

Limitations of this study include that we restricted our analysis to the IVA2-CPT to assess the effect of NF on cognition. Future studies are needed to apply computational modeling to a battery of cognitive tasks to accomplish a more holistic description of cognitive characteristics of ADHD and how treatments affect those characteristics (see for further discussions: Ging-Jehli et al., 2021; Hitchcock, Fried, & Frank, 2022). We focused on immediate effects of NF on cognition

by concentrating on changes in cognition from baseline to treatment end. Future studies should investigate whether these effects last beyond completion of the intervention.

NF treatment includes components of reinforcement learning whose paradigms have also been used for cognitive behavioral therapies and for characterizing other mental health conditions (e.g., Brown et al., 2021; Lubianiker, et al., 2022; Queirazza, Fouragnan, Steele, Cavanagh, & Philiastides, 2019). It is therefore critical to understand the underlying cognitive effects of NF which can help to personalize treatment planning. This seems particularly relevant when considering that the primary outcome (symptom improvement in inattention rated by parents and teachers) of this RCT showed unspecific improvement for all children, irrespective of whether they received NF or control treatment (The Neurofeedback Collaborative Group, 2021; 2023). Moreover, a recent secondary moderator analysis of this RCT showed that baseline cognitive characteristics identified children that benefitted more from NF than control treatment (Ging-Jehli et al., 2023). These findings therefore emphasize the importance of understanding the cognitive effects (i.e., active components) of NF as a cognitive reinforcement learning intervention.

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Figures

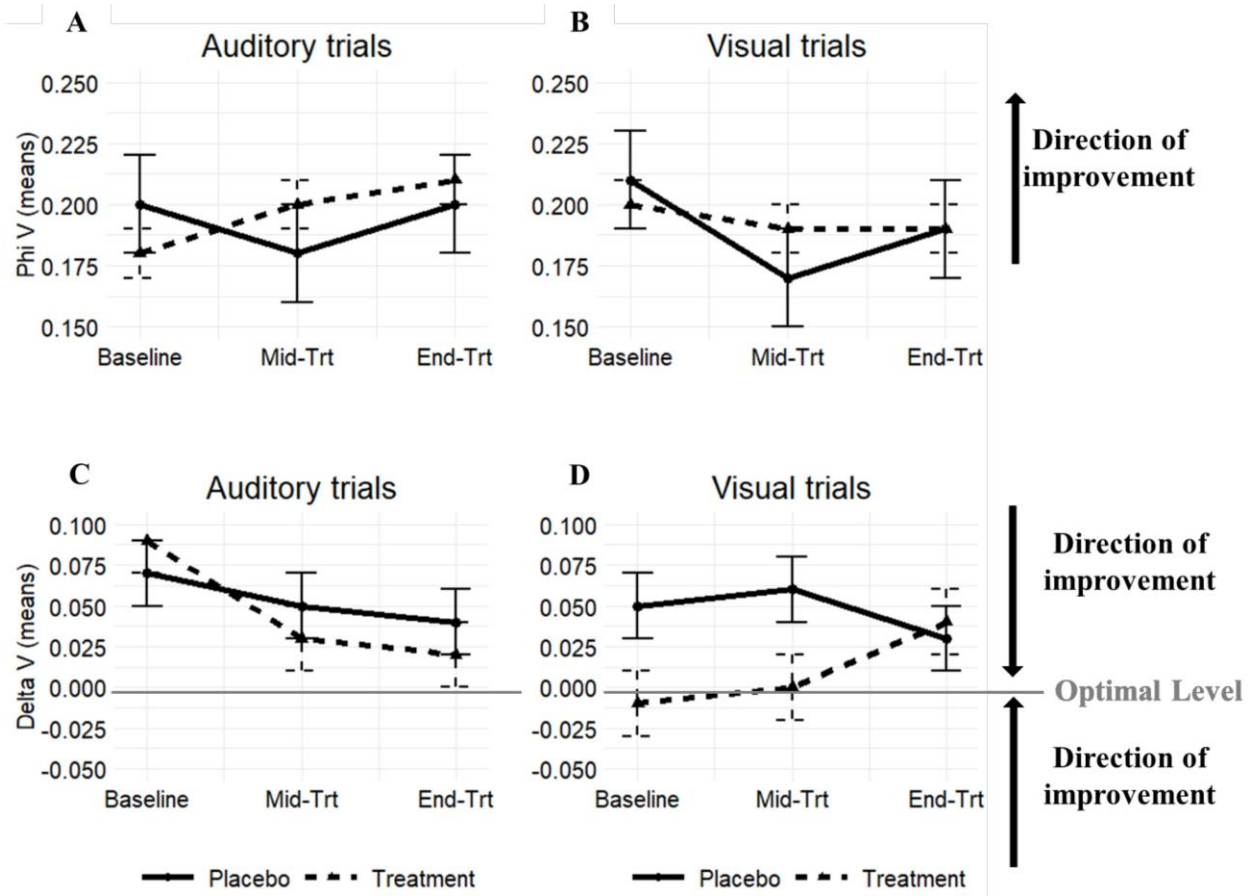

Figure 1. *Phi v (panels A and B) and delta v (panels C and D) for each ADHD group (control treatment, NF). Shown are estimated means (points) and vertical bars that represent +/- 1SE. Phi v refers to the averaged drift rates of go trials and no-go trials for each modality. Larger phi v indicates better (i.e., faster and more accurate) information processing. Delta v refers to drift rate of go trials minus drift rate of no-go trials for each modality. Positive delta drift rate means that go trials had higher (faster and more accurate) drift rate than no-go trials; negative delta drift means no-go trials had higher drift rate than go trials. Delta v equal to zero represents the optimal level, suggesting information processing to be context- independent. We also refer to Supplemental Table S4 for means and SDs by: parameter, block type, modality, time point, and group.*

Tables

Table 1. *Background characteristics for the subject groups.*

	Healthy Control (HC) Group (N = 57)	ADHD Control treatment (N = 55)	ADHD Neurofeedback treatment (N = 78)	Group comparisons
Mean (SD) age in years	9 (1)	8 (1)	8 (1)	HC vs. Control treatment: ** HC vs. NF: ** Control treatment vs. NF: ns
Number of females	26	11	19	HC vs. Control treatment: ** HC vs. NF: ** Control treatment vs. NF: ns
Co-morbid diagnoses[a]				HC vs. Control treatment: ** HC vs. NF: ** Control treatment vs. NF: ns
Neither ANX nor ODD	0	19	20	
ANX only	0	7	20	
ODD only	0	11	20	
Both (ANX and ODD)	0	18	18	
Mean (SD) T-scores on inattention	48 (6)	78 (10)	80 (9)	HC vs. Control treatment: ** HC vs. NF: ** Control treatment vs. NF: ns
Mean (SD) T-scores on hyperactivity-impulsivity	49 (7)	77 (12)	73 (14)	HC vs. Control treatment: ** HC vs. NF: ** Control treatment vs. NF: ns
Child's Educational setting				HC vs. Control treatment: ** HC vs. NF: ** Control treatment vs. NF: ns
Regular public school (N)	54	25	31	
Regular public school with some special classes (N)	1	26	26	
Regular private/parochial school (N)	2	1	5	
Home school (N)	0	0	3	
Charter school (N)	0	3	12	
Special school for children with developmental disabilities (N)	0	0	0	
Primary Caregiver's Education				HC vs. Control treatment: ns HC vs. NF: ns
High school/GED or less (N)	2	5	6	
Some college (N)	12	11	18	

College (N)	21	19	33	Control treatment vs. NF:
Advanced degree (N)	22	20	21	ns
Annual Household Income				HC vs. Control treatment:
Less than $23,850 (N)	2	4	8	**
$23,851-$50,000 (N)	5	8	14	HC vs. NF: **
$50,001-$100,000 (N)	13	27	31	Control treatment vs. NF:
More than $100,000 (N)	37	16	25	ns

Note. Group comparisons for age and T-scores are based on contrast tests. Group comparisons for number of females, co-morbid diagnoses, child's educational setting, primary caregiver's education, and annual household income are based on chi-square tests. Numbers in brackets refer to SD. ns=nonsignificant. [a]Comorbidity group classification based on ChIPS (see Measures): neither=neither anxiety disorders nor oppositional defiant disorder, ANX = anxiety disorders only, ODD=oppositional defiant disorder only, Both=ANX and ODD. T-scores on parent-rated inattention (AN) and hyperactivity-impulsivity (AH) (both are DSM-5 scales).
**$p < .01$ after correction for multiple tests.

Table 2. *Summary of performance data for each task condition and time point.*

Variable	IVA2-CPT condition	ADHD Control Treatment			ADHD NF Treatment		
		Baseline (N=55)	Mid-Trt (N=54)	End-Trt (N=51)	Baseline (N=78)	Mid-Trt (N=77)	End-Trt (N=76)
%Commissions	FreqGo,Aud	0.348	0.312	0.291	0.376	0.3	0.291
	FreqGo,Vis	0.311	0.31	0.276	0.298	0.292	0.296
	RareGo,Aud	0.134	0.107	0.09	0.14	0.098	0.077
	RareGo,Vis	0.108	0.095	0.083	0.088	0.074	0.081
%Omissions	FreqGo,Aud	0.134	0.183	0.199	0.155	0.207	0.208
	FreqGo,Vis	0.218	0.249	0.247	0.244	0.272	0.248
	RareGo,Aud	0.175	0.265	0.265	0.21	0.243	0.244
	RareGo,Vis	0.279	0.359	0.336	0.353	0.372	0.352
Mean RT (ms) (correct responses)	FreqGo,Aud	716.7	728	724.8	712.5	740.2	737
	FreqGo,Vis	569.2	566.8	570.9	595.6	584.3	578
	RareGo,Aud	807.4	811.4	817.4	832.5	807.4	814.2
	RareGo,Vis	658.5	642.3	663.4	694.8	687.6	668.5
SD RT (ms) (correct responses)	FreqGo,Aud	251.4	257.6	246.1	254.8	256.8	252.4
	FreqGo,Vis	233.1	222.3	216.5	232.1	226.7	237.9
	RareGo,Aud	234.9	234.4	222.1	243.5	239.4	306.5
	RareGo,Vis	216.2	225	208.5	219.6	231	313.8
Mean RT (ms) (error responses)	FreqGo,Aud	588.2	593.4	614.9	568.7	630.8	639
	FreqGo,Vis	497.7	498.9	485.2	489.7	503.6	490
	RareGo,Aud	641.1	668.8	740.5	642.5	717.4	704.8
	RareGo,Vis	559.1	588.3	608.9	536.4	580	591.5
SD RT (ms) (error responses)	FreqGo,Aud	268.2	280.3	273.8	249.8	279	304.7
	FreqGo,Vis	255.9	262.2	236.4	244.5	252.9	237.9
	RareGo,Aud	288.6	305.2	292.3	269.7	309	306.5
	RareGo,Vis	301.5	315.5	326	258.2	310.2	313.8
T_{er}	FreqGo,Aud	0.470	0.437	0.456	0.460	0.482	0.476
	FreqGo,Vis	0.356	0.334	0.343	0.369	0.370	0.361
	RareGo,Aud	0.517	0.443	0.507	0.511	0.525	0.526
	RareGo,Vis	0.403	0.361	0.383	0.421	0.422	0.390
v_{Go}	FreqGo,Aud	0.208	0.194	0.210	0.214	0.171	0.194
	FreqGo,Vis	0.202	0.202	0.210	0.187	0.176	0.209
	RareGo,Aud	0.258	0.201	0.236	0.239	0.249	0.250
	RareGo,Vis	0.247	0.175	0.203	0.189	0.160	0.194
$v_{No\text{-}Go}$	FreqGo,Aud	0.095	0.096	0.114	0.047	0.129	0.140
	FreqGo,Vis	0.122	0.096	0.098	0.116	0.127	0.104
	RareGo,Aud	0.229	0.222	0.267	0.230	0.257	0.276
	RareGo,Vis	0.250	0.233	0.273	0.319	0.293	0.272
a	Constant	0.142	0.149	0.142	0.142	0.142	0.142
z/a	Constant	0.500	0.460	0.450	0.500	0.470	0.460

Note. IVA2-CPT conditions: first expression in second column refers to block type, whereas second expression in second column refers to modality. T_{er} = nondecision time component (in ms), v_{Go} = drift rate for go trials, $v_{No\text{-}Go}$ = drift rate for no-go trials (absolute values, see footnote 2), a = boundary separation, z/a = starting point bias.