Neurophysiology of perceptual decision-making and its alterations in attention deficit hyperactivity disorder (ADHD)

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Abstract

ADHD is a prevalent neurodevelopmental disorder associated with adverse outcomes and significant social and economic cost. However, efforts to develop a more detailed understanding of the neuropsychology of ADHD are complicated by the diversity of interindividual presentation and the inability of current clinical tests to distinguish between its sensory, attentional, arousal or motoric contributions. Identifying objective methods that can decompose the clinical heterogeneity of ADHD is a long-held goal that will advance our understanding of aetiological processes and aid the development of personalised treatment approaches. Here, we examine key neuropsychological components of ADHD with an electrophysiological (EEG) decision making paradigm capable of isolating distinct neural signals of several key information processing stages necessary for sensory-guided actions from initial sensory processing to motor responses. We show that compared to typically developing children, children with ADHD display slower and less accurate behavioural performance driven by the atypical dynamics of discrete electrophysiological signatures of attentional allocation, the accumulation of sensory evidence, and strategic adjustments reflecting urgency of response. These findings offer an integrated account of impairments in ADHD and establish neural signals that can serve as critical guidance in constructing or constraining mechanistic accounts in future research, as well as in ADHD diagnosis and treatment tailoring.

Keywords: ADHD, Perceptual Decision-Making, Evidence Accumulation, Drift Diffusion Model, Attention

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a prevalent childhood-onset condition characterised by persistent inattention, impulsivity, and hyperactivity. These symptoms undermine the ability to adapt behaviour to social and environmental demands and are associated with negative lifetime health, social, occupational and economic outcomes (Barkley, 1997; Nigg, 2013; Sciberras et al., 2022). While early attempts to identify the neuropsychological impairments underlying ADHD focused primarily on reliable deficits in higher-level cognitive processes associated with attentional control and executive functioning (e.g. response inhibition, working memory, cognitive flexibility (Barkley, 1997; Pennington and Ozonoff, 1996; Sergeant et al., 2002)), ADHD is now recognised as a complex, heterogeneous, and multifactorial condition with contributions spanning multiple levels of processing (Coghill et al., 2014; Sergeant et al., 2003; Willcutt et al., 2005), including basic perceptual processes (Gonen-Yaacovi et al., 2016; Kim et al., 2014; Mihali et al., 2018; Mulder et al., 2010; Nazari et al., 2010; Panagiotidi et al., 2018), reward sensitivity (Ma et al., 2016; Tripp and Wickens, 2009), arousal (Sergeant, 2005; Sonuga-Barke and Castellanos, 2007), evidence accumulation (Karalunas and Huang-Pollock, 2013), and neuromotor processing (Goulardins et al., 2017; Hurks et al., 2005; Kaiser et al., 2015; Rommelse et al., 2008; Steger et al., 2001). This raises the possibility that deficits detected in higher-level processes may actually be inherited from dysfunction in more basic mechanisms that support downstream faculties (Rommelse et al., 2007).

Many of these component processes have been identified based on hallmark differences in performance on decision making reaction time tasks, where ADHD participants exhibit reduced accuracy and slower, more variable reaction times (Alderson et al., 2007; Castellanos and Tannock, 2002; Johnson et al., 2007; Karalunas and Huang-Pollock, 2013). These reliable effects represent promising avenues in the search for the fundamental cognitive mechanisms underlying dysfunction in ADHD (Alderson et al., 2007; Castellanos et al., 2005; Castellanos and Tannock, 2002). However, since they are the product of various interacting processes (e.g., sensory encoding, signal processing, motor preparation, urgency, decision bias and strategy), it is difficult to develop mechanistic accounts based on reaction times alone. Computational models, like the Drift Diffusion Model (DDM; (Ratcliff et al., 2016; Ratcliff and McKoon, 2008)), provide powerful theoretical frameworks that can help to disentangle these influences

by recovering latent psychological processes from their behavioural output (Forstmann et al., 2016; O'Connell et al., 2018).

The DDM conceptualises decision-making as a dynamic process of accumulation of noisy sensory evidence over time until a decision threshold is reached, and a response is initiated. Efforts to interpret the neuropsychology of ADHD within this principled framework suggest that children with ADHD exhibit slower, less efficient accumulation of sensory evidence (reflected in a reduced drift rate parameter), and standard speed-accuracy relationships (reflected in typical decision boundary separation; but see (Mulder et al., 2010)); with mixed evidence for impairment of non-decision processes such as stimulus encoding and motor execution (Huang-Pollock et al., 2017, 2012; Karalunas et al., 2018, 2014, 2012; Karalunas and Huang-Pollock, 2013). While these are valuable insights pointing to specific decision making mechanisms that may operate differently in ADHD, they are primarily derived from neurocognitive tasks designed to assess special cases of decision making that isolate executive inhibition or vigilance specifically (e.g. Stop-Signal, Go/No-Go, Stroop, Continuous Performance Test (Nigg, 2001)), so they may not capture dynamics that easily generalise to cognition more broadly. In addition, computational models offer little insight into the underlying neurophysiological processes that support these mechanisms. Perceptual decision-making paradigms can address each of these issues.

Perceptual decision making is a fundamental neural process through which ambiguous sensory information is translated into meaningful interpretations of the environment and adaptive action. Research on perceptual decisions has led to critical advances in cognitive science specifically because they expose basic mechanisms that are essential for higher-cognitive functions (Shadlen and Kiani, 2013) and much of this progress has been forged through the application of sequential sampling models (Forstmann et al., 2016; Gold and Shadlen, 2007; O'Connell et al., 2018; Smith and Ratcliff, 2004), such as the DDM. Building on foundational work in monkey neurophysiology (Gold and Shadlen, 2007; Hanks and Summerfield, 2017), research in humans now capitalises on paradigms with the capacity to decompose these simple decisions into their constituent processes (e.g. Steinemann et al., 2018) and the ability to non-invasively capture electrophysiological signatures of several key information processing stages in decision formation (O'Connell and Kelly, 2021).

Some of these signals index processes that are candidate sources of decision making deficits in ADHD, including attentional selection (N2c/i) and strategic adjustments to the level of evidence required for decision commitment (i.e. dynamic urgency; frontocentral negativity or FCN). However, because inefficient evidence accumulation has been consistently identified as a marker of ADHD in modelling research (Weigard and Sripada, 2021), of central interest in this study is an abstract evidence accumulation signal called the centroparietal positivity (CPP (O'connell et al., 2012)). These processes can be measured simultaneously and independently within a single paradigm and each of these EEG signals have been shown to predict the timing and accuracy of behavioural responses in a manner that can be reconciled within a mechanistic, computational framework (O'Connell et al., 2018). Here, we sought to develop an integrated account of the neurophysiology of ADHD by linking these distinct EEG signals to computational mechanisms associated with characteristic performance deficits in perceptual decision-making tasks.

The aims of our study were twofold. First, we aimed to characterise the dynamical properties of decision-making signals in individuals with ADHD and develop a mechanistic account that captures individual variations in performance. Second, we sought to establish the link between EEG data and DDM parameters to bridge the existing gap between the neurophysiological signals and computational modelling in humans with a particular focus on ADHD. This comprehensive analysis allowed us to explore the neural mechanisms, cognitive dynamics, and computational principles that govern decision making in the context of ADHD.

Methods

Participants. The study recruited a total of 79 right-handed individuals with normal or corrected-to-normal vision who were aged between 8 and 17 years, including 37 participants with ADHD (13 female; $Mean_{age}$ = 13.45 years ± SD_{age} = 2.026) and 42 healthy controls (18 female; $Mean_{age}$ = 13.46 years ± SD_{age} = 1.93). Data were collected at the University of Queensland (UQ) (n = 58) and Monash University (n = 21) of Australia following identical experimental protocols. Ethical approval was obtained from the human research ethics committees of both universities, and the study was conducted in accordance with approved guidelines. For the ADHD group, inclusion criteria required previous diagnosis by a specialist (e.g., psychiatrist or paediatrician), confirmed using the the Anxiety Disorders Interview Schedule for DSM-IV (Child Version) (A-DISC) (for UQ participants) or The Development and Well-being Assessment (DAWBA, for Monash participants) and the Global Index on the

Conners' Parent Rating Scale-Revised: Long Version (CPRS-R: L; T-Score > 65 from the mean). The participants with ADHD completed a 48-hour washout of ADHD-related medications before testing. Typically developing children, who were free of clinical diagnoses and had Conners' Global Index T-Scores < 65 from the mean, were subject to the same inclusion criteria, except for the ADHD diagnosis. Informed consent from parents or guardians and assent was obtained from the participant prior to testing. Figure 1 presents the clinical characteristics of the individuals in the two groups.

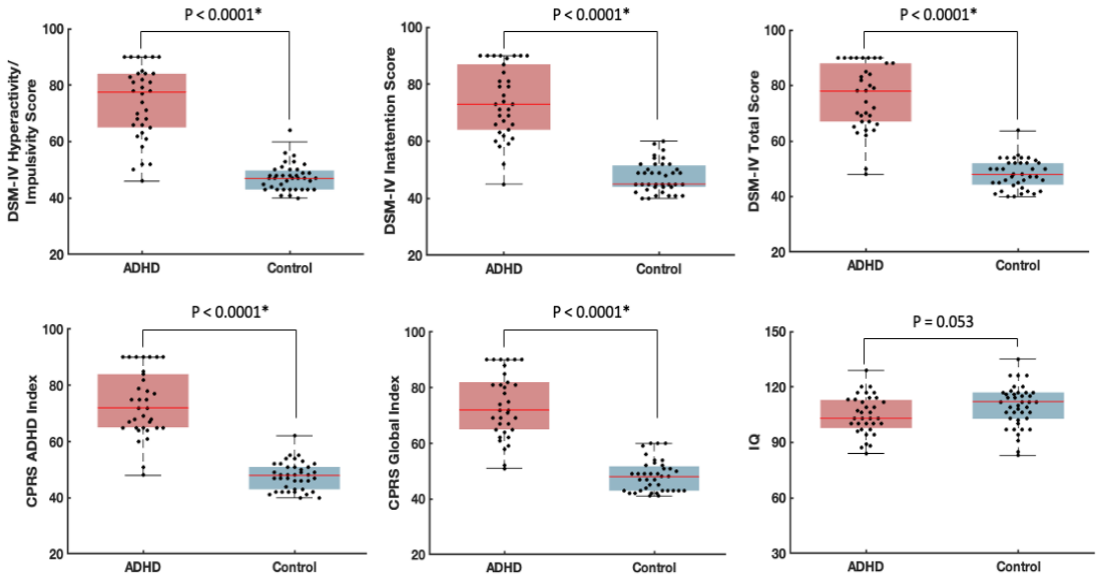

Figure 1. Clinical characteristics of participants in each group. P: P-Value from Wilcoxon-Signed Rank Test comparing the two groups. Every data point in the box and whisker plots corresponds to the clinical score for each person. The shaded boxes indicate the range between the 25th and 75th percentiles of the scores, while the red horizontal lines inside the boxes represent the median score. * Indicates statistically significant differences between groups.

Experimental Protocol

Participants were seated in a darkened sound-attenuated room positioned at a viewing distance of approximately 56 cm from a 21-inch CRT monitor (resolution: 1024 x 768, refresh rate: 85 Hz) and instructed to perform a bilateral random dot motion perceptual decision-making task. The task was run through MATLAB's psychophysics toolbox extension on a 32-bit Windows XP computer(Brainard and Vision, 1997). A chin rest was employed to stabilise participants' heads and maintain a constant visual angle throughout the task. An Eyelink 1000 eye tracking system (SR Research, Ottawa, ON, Canada) was used to monitor gaze at fixation.

In this task, participants fixated on a centrally presented 5 x 5-pixel square dot while simultaneously monitoring two circular patches, one per hemifield. These peripheral patches were eight degrees in diameter and contained 150 randomly moving six by six-pixel dots. The centre of each patch was situated 4º below and 10º to the left or right of the central fixation dot to maintain an optimum visual angle for both hemifields. At pseudo-random intervals of either 3.06, 5.17 or 7.29 seconds, a subset of the dots transitioned to coherent downward motion for 1.88 seconds (Stefanac et al., 2021). These dots were randomly selected to move downwards by 0.282 degrees per frame (6 degrees per second). The coherent downward motion occurred once per trial and appeared with equal probability in either the left or right hemifield patch. Participants were instructed to respond promptly by pressing both mouse buttons simultaneously with their thumbs upon detecting the downward motion, employing a double thumb click to mitigate potential bias associated with the Simon effect (Simon and Rudell, 1967). To capture robust accuracy and reaction time contrasts between groups, a relatively low coherence level of 30% was chosen for this task, deviating from previous studies using the same paradigm (Loughnane et al., 2016; Stefanac et al., 2021). Participants completed 200 trials of the task, divided into 10 blocks of 20 coherent motion trials each, with intermittent breaks to reduce fatigue (Figure 2)

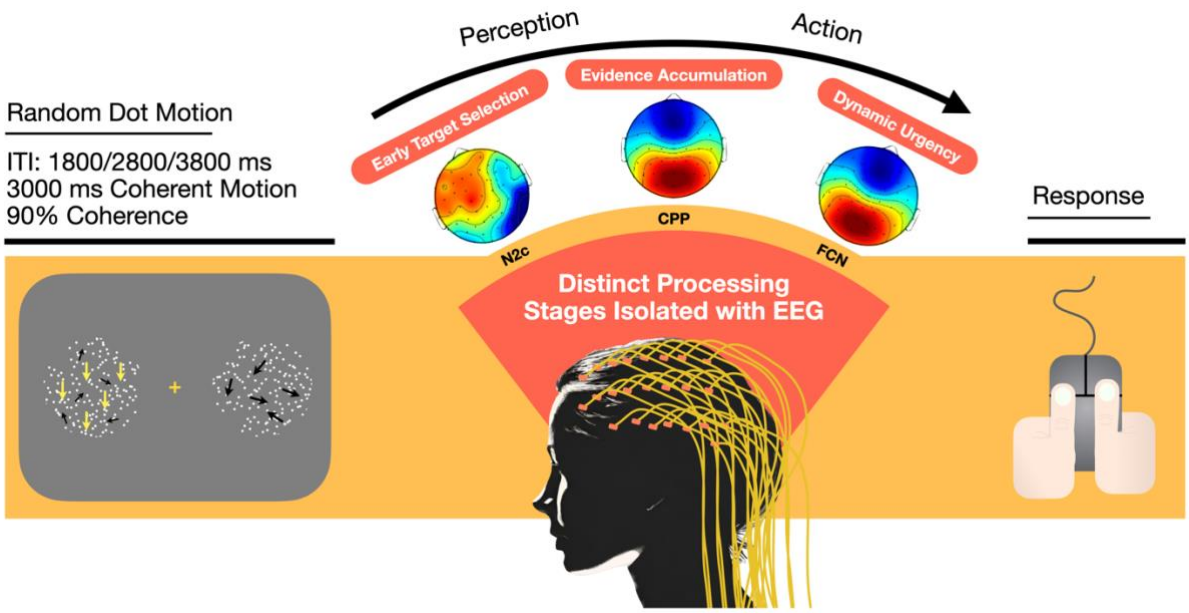

Figure 2. Depiction of the random-dot motion detection task and the obtained measures.

EEG acquisition and pre-processing

Continuous EEG data were acquired from 64 scalp electrodes (10-10 layout) using BioSemi Active II (Neurospec, Switzerland) digitised at 1024 Hz (at the University of Queensland) or a

BrainAmp DC system (BrainProducts, Germany) digitised at 500 Hz (at Monash University). Data were analysed using custom scripts in MATLAB (The MathWorks, Inc.) and EEGLAB toolbox (Delorme and Makeig, 2004). EEG recordings from the two locations were combined by down-sampling the data collected in Queensland to 500 Hz. Signals were low-pass filtered up to a 35 Hz cut-off using Hamming windowed-sinc FIR filter and no high pass filter was applied. Noisy channels were then interpolated using spherical spline interpolation and the data were re-referenced to the common average. Target epochs were extracted using a window of -800 ms to 1880 ms around the onset of the target stimulus (coherent motion) and baseline corrected at -100 to 0 ms. The behavioural measures of reaction time (RT) and accuracy were extracted as the average time to respond to a target in milliseconds (ms) and the percentage of correctly identified targets, respectively. Trials were rejected if any of the following occurred: 1) the central gaze fixation was broken by blinking or vertical/horizontal eye movement greater than three degrees; 2) recordings from any electrode exceeded ±100 μV; 3) RTs were faster than 200 ms (pre-emptive responses) or slower than 1880 ms (responses after the offset of coherent motion). Missed targets were defined as either responses that took longer than 1880 ms or complete absence of responses. Hit rate was measured as the percentage of trials with valid responses.

ERPs for each individual were extracted from the average of single-trial epochs. For each individual, we isolated four distinct and previously validated EEG signatures of decision-making processes (Brosnan et al., 2020): pre-target attentional engagement (Alpha power), early target selection (N2c peak latency and amplitude Loughnane et al., 2016), evidence accumulation (CPP onset latency, slope, and amplitude (McGovern et al., 2018; O'connell et al., 2012; Steinemann et al., 2018), and dynamic urgency (FCN slope and amplitude (Devine et al., 2019)). Pre-target alpha power was computed using the temporal spectral evolution approach. In which, all epochs were bandpass filtered at 8–14 Hz, rectified, and trimmed by 200ms at both ends of the epoch to eliminate filter warm-up artefacts. Subsequently, the data were smoothed by averaging within a 100-ms moving window shifting forward in 50-ms steps throughout each epoch. Mean alpha power was extracted bilaterally at peak electrodes - PO3/PO7 (left hemisphere) and PO4/PO8 (right hemisphere) - from 400 to 0 ms prior to the target onset and was baseline-corrected using the period of 700 to 400 ms before the target onset. For N2c components, peak negative amplitude was measured contralateral to the target hemifield, from electrodes P7 and P8 between 150ms and 400ms post target onset (Stefanac et al., 2021). CPP was extracted from the average of the potentials recorded at Pz and POz

electrodes and FCN was measured at the peak electrode FCz. The amplitude of each component was determined by calculating the mean signal amplitude within the same 100-ms window preceding the response. CPP onset latency was derived by performing point-by-point one-sample t-tests against zero over stimulus-locked trials for each individual. The onset was defined as the first point in time when the amplitude reached the significance level of 0.05 for 25 consecutive samples. The build-up rate of the CPP was measured as the slope of a straight line fitted to the response-locked signal at -450ms to -50ms. The variation of the task used in this study, involving double thumb clicks, did not allow us to extract lateralized beta activity.

Drift Diffusion Modelling

The drift diffusion model was fitted to the behaviour of all participants (both ADHD and typically developing children) at an individual level (Ratcliff et al., 2018)(Ratcliffe et al., 2018). In this case, the RT distribution for the response and no-response choices of each individual was first split into five bins according to the quantiles 0.1, 0.3, 0.5, 0.7 and 0.9. Together with a single bin containing the number of missed responses, these six bins were then used to fit the drift diffusion model using the G-square method of the hDDM package (de Gee et al., 2020; Ratcliff et al., 2016; Wiecki et al., 2013). This method is a variant of the chi-squared method and was chosen for its efficiency, the availability of significant trial data for each individual, its robustness to outliers and its success in previous experiments (Myers et al., 2022; Ratcliff et al., 2016). The proportion of responses within each bin was determined by subtracting the cumulative probabilities for each successive quantile from the next highest quantile. These proportions were then multiplied by the number of observations to obtain the expected frequencies $E \in R$. Next, the observed proportions, determined from the data (in this case: 0.1, 0.2, 0.2, 0.2, 0.2 and 0.1), were also multiplied by the number of observations to obtain the observed frequencies $O \in R$. G-square, calculated for each quantile $i \in N$, defined as:

$$G^2 = 2\sum_{i=1}^{6} O_i \ln\left(\frac{O_i}{E_i}\right)$$

where $i \in N$ represents the quantile number. The drift-diffusion model parameters a, v, and t, were then determined by minimising the G-square statistic using the modified Powell method (Powell, 1964). The fitted drift-diffusion model assumed that the response caution, mean drift

rate across trials, and non-decision time varied with the response bins. Further details can be found in (Ratcliff et al., 2018).

Statistical Analysis

Significant outliers were winsorised to the 5th percentile (for the lower outliers) and the 95th percentile (for the upper outliers) in each participant group to improve normality of RT distributions. CPP onset could not be detected for five individuals in each group due to noisy signals, so the missing values were replaced with the group median. A multivariate analysis of covariance (MANCOVA) was conducted to compare the two groups in behavioural measures (RT, Hit Rate and Miss Rate), EEG signatures including Alpha (Power), N2c (peak latency, amplitude), CPP (onset, amplitude, slope) and FCN (amplitude, slope), and DDM parameters (drift rate, Decision Threshold, Non-Decision Time), while controlling for the effect of recruitment site (Monash, UQ).

Next, we adopted a hierarchical regression approach to investigate the association between the EEG components and both behavioural and DDM outcomes. Separate hierarchical linear regression models were applied for each behavioural and DDM measure as a function of the EEG signals that showed significant differences between groups in at least one feature. EEG components were sequentially entered into the regression models, following the temporal order of the perceptual decision-making processes: pre-target attentional engagement (Alpha power), early target selection (N2c peak latency and amplitude), evidence accumulation (CPP onset, slope and amplitude) and dynamic urgency (FCN slope and amplitude). This hierarchical entry method allowed us to evaluate whether each individual neurophysiological signal contributed to the model fit for behavioural performance or DDM parameter beyond the preceding signals in the temporal sequence. Any neurophysiological signals that improved the model fit were incorporated into a separate regression model to obtain accurate parameter estimates. Subsequently, Spearman's partial correlation analyses were employed, controlling for the effects of group and location, to evaluate the magnitude and orientation of the relationship between EEG components and both behavioural outcomes and DDM parameters.

Finally, we examined whether the EEG signals could predict ADHD symptom scores while accounting for the effects of Group and Site. We employed five separate linear regression models, each with one ADHD symptom score as the dependent variable (i.e., DSM-IV

hyperactivity/impulsivity score, DSM-IV inattention score, DSM-IV total score, CPRS ADHD Index, CPRS Global Index), while EEG signatures were entered into the model hierarchically.

Results

To examine the differences between the ADHD and control groups on the behavioural measures, DDM parameters, and electrophysiological signals, we conducted MANCOVA with site as a covariate. As the Box's M test score was significant ($p = 0.04$), we used Pillai's trace statistic, which is considered to be most robust against type I error in MANCOVA (Olson, 1976; Scheiner, 2020). The results revealed significant main effects of both Group (Pillai's Trace = 0.31, $F(14, 63) = 2.05$, $p = 0.02$, partial $\eta 2 = 0.31$) and Site (Pillai's Trace = 0.46, $F(14, 63) = 3.90$, $p < 0.001$, partial $\eta 2 = 0.46$). Table 1 summarises pairwise comparisons of all the measures between the two groups. Significant differences were observed between the two groups in all behavioural and EEG signals (in at least one feature), with the exception of Alpha power. Despite individuals with ADHD showing a notable reduction in the DDM drift rate parameter, none of the parameters estimates showed significant differences between the two groups.

Table 1. Pairwise comparisons of measures between the ADHD and typically developing groups

Measures	Mean Difference ADHD - Control	Std. Error	Sig.	95% Confidence Interval	
RT	58.57	28.45	0.04*	1.91	115.24
Hit Rate	-3.70	1.73	0.03*	-7.14	-0.26
Miss Rate	7.70	2.91	0.01*	1.90	13.51
Pre-target Alpha†	-0.36	0.05	0.51	-0.14	0.72
N2c Latency	-24.93	10.95	0.02*	-46.75	-3.13
N2c Amplitude	0.75	0.32	0.02*	0.11	1.40
CPP Onset	25.50	16.55	0.13	-7.46	58.45
CPP Slope	-0.01	0.002	0.02*	-0.01	-0.001
CPP Amplitude	-2.36	0.88	0.009*	-4.12	-0.61
FCN Amplitude	1.29	1.04	0.21	-0.78	3.37
FCN Slope	0.02	0.01	0.03*	0.002	0.04
Drift Rate	-0.24	0.13	0.07	-0.50	0.03
Non-Decision Time	0.26	0.23	0.38	-0.19	0.72
Response Threshold	0.33	0.28	0.24	-0.23	0.89

* The mean difference is significant at alpha level of 0.05. † Mean values are multiplied by 10^{15}.

Next, we examined the relationship between each EEG signal that showed significant difference between groups (N2c, CPP and FCN; Figure 3) and variations in behaviour using a hierarchical regression model including group and location as nuisance variables in the initial step of the model. In the subsequent steps, we sequentially incorporated the neural markers of target selection (N2c: peak latency and amplitude), evidence accumulation (CPP: onset, amplitude, and slope), and dynamic urgency (FCN: amplitude and slope) into the model.

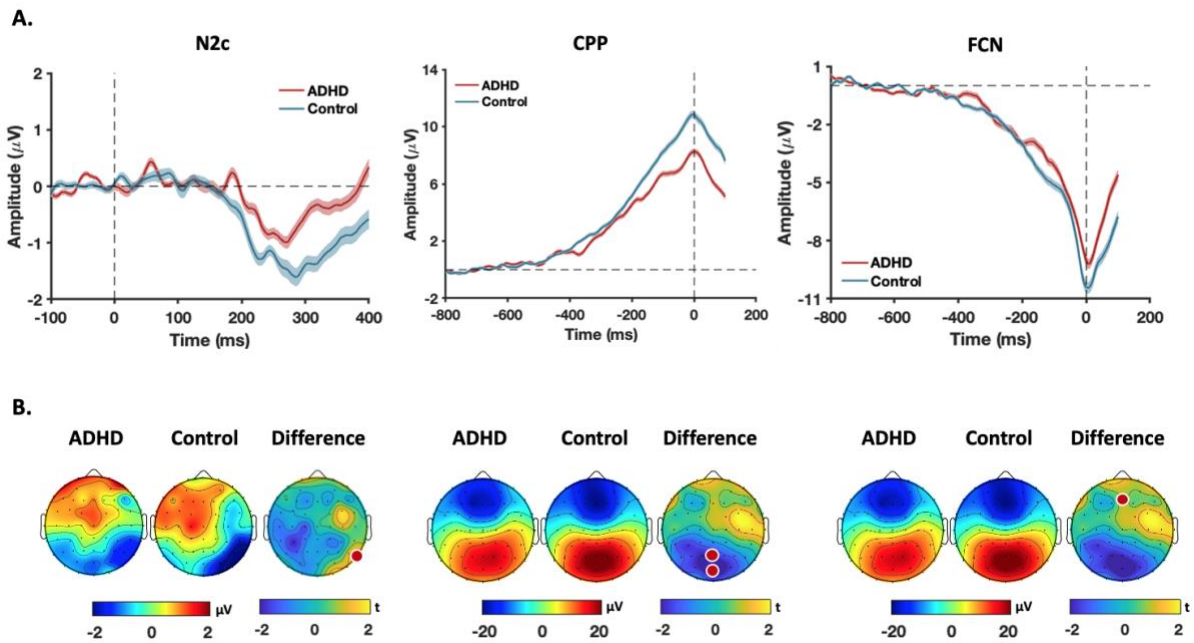

Figure 3. EEG signals of decision making in ADHD and typically developing groups. A. Group average EEG signal waveforms for each neural signature of the decision process. The thick line in the graph is the group-averaged waveform and the shaded areas represent the standard error of mean at each point of time. The vertical dashed line at the zero point indicates the onset of target for N2c and represents the response time for CPP and FCN. The horizontal lines represent the baseline level of EEG activity. **B.** The scalp maps depict the potential distribution at the time of peak amplitude for each group, and difference maps demonstrate the distribution of t-values resulting from t-tests comparing the two groups. The electrodes highlighted in red indicate the specific electrodes used for the line graphs and subsequent analysis.

Neural metrics of sensory evidence accumulation capture variations in Reaction time

The neural signatures of the decision process collectively accounted for a substantial 43% of the variance in RT. Adding each of the neural components resulted in a significant improvement in the model fit (N2c latency: R^2_{adj} = 0.16, F (3,75) = 5.98, p <0.001; N2c amplitude: R^2_{adj} = 0.16, F (4,74) = 4.61, p = 0.002; CPP onset: R^2_{adj} = 0.27, F (5,73) = 6.84, p <0.001; CPP slope: R^2_{adj} = 0.35, F (6,72) = 7.99, p <0.001; CPP amplitude: R^2_{adj} = 0.44, F (7,71) = 9.88, p <0.001; FCN slope: R^2_{adj} = 0.44, F (8,70) = 8.64, p <0.001; FCN amplitude: R^2_{adj} = 0.43, F (9,69) = 7.63, p <0.001). The analysis of coefficients revealed that only CPP components (onset: stand. β = 0.44, t = 4.51, p < 0.001; slope: stand. β = -0.81, t = -4.12, p <

0.001; amplitude: stand. $\beta = 0.60$, $t = 2.74$, $p = 0.008$) had independent predictive power for RT.

To further establish the utility of these decision signals as specifically sensitive to individual differences in ADHD, we repeated this linear regression model just for the ADHD group. Adding each CPP and FCN component led to a significant improvement in the model's overall fit (CPP onset: $R^2_{adj} = 0.21$, $F(4,32) = 3.31$, $p = 0.02$; CPP slope: $R^2_{adj} = 0.26$, $F(5,31) = 3.22$, $p = 0.02$; CPP amplitude: $R^2_{adj} = 0.47$, $F(6,30) = 6.30$, $p < 0.001$; FCN slope: $R^2_{adj} = 0.47$, $F(7,29) = 5.51$, $p < 0.001$; FCN amplitude: $R^2_{adj} = 0.47$, $F(8,28) = 5.11$, $p < 0.001$). However, N2c measures did not substantially improve the model (N2c latency: $R^2_{adj} = 0.095$, $F(2,34) = 2.9$, $p = 0.07$; N2c amplitude: $R^2_{adj} = 0.08$, $F(3,33) = 2.01$, $p = 0.13$).

Next, we incorporated the variables that explained independent variance in RT (CPP and FCN dynamics) into a single model and excluded non-informative signals. These variables collectively accounted for a notable 50% of the variance in RT ($F(6,30) = 4.74$, $p = 0.007$) in the ADHD group. CPP measures (onset: stand. $\beta = 0.53$, $p < 0.001$; slope: stand. $\beta = -0.74$, $p < 0.001$; amplitude: stand. $\beta = 0.77$, $p < 0.001$), but not FCN components (slope: stand. $\beta = -0.013$, $p = 0.94$; amplitude: stand. $\beta = 0.22$, $p = 0.20$), showed strong independent predictive power for RT for the ADHD group. Together, these findings highlight the significance of CPP dynamics as a robust indicator of RT variations in decision making processes, particularly in relation to individual differences observed in ADHD.

Neural metrics of sensory evidence accumulation capture variations in Miss Rate

The second model examined the neural predictors of Miss Rate, which is indicative of inattention, and yielded comparable outcomes to those from RT. Again, all neural components significantly improved the model's fit (N2c latency: $R^2_{adj} = 0.16$, $F(3,75) = 5.48$, $p = 0.002$; N2c amplitude: $R^2_{adj} = 0.15$, $F(4,74) = 4.46$, $p = 0.003$; CPP onset: $R^2_{adj} = 0.26$, $F(5,73) = 6.60$, $p < 0.001$; CPP slope: $R^2_{adj} = 0.27$, $F(6,72) = 5.47$, $p < 0.001$; CPP amplitude: $R^2_{adj} = 0.25$, $F(7,71) = 4.65$, $p < 0.001$; FCN slope: $R^2_{adj} = 0.25$, $F(8,70) = 4.24$, $p < 0.001$; FCN amplitude: $R^2_{adj} = 0.27$, $F(9,69) = 4.17$, $p < 0.001$) but only CPP onset demonstrated independent predictive power for Miss Rate (stand. $\beta = 0.45$, $t = 3.95$, $p < 0.001$). Repeating the analysis separately for the ADHD group alone yielded similar results, indicating that the

collective contribution of these measures to Miss Rate variations in the ADHD group (29%, $F(8,28) = 2.85$, $p = 0.02$) and CPP onset retained its independent predictive power (stand. $\beta = 0.40$, $t = 2.42$, $p = 0.022$). These findings further confirm the collective power of the N2c, CPP and FCN signals to predict performance in perceptual decision-making. They also establish CPP onset as a robust predictor of Miss Rate which can independently explain individual variation in ADHD.

Neural metrics of sensory evidence accumulation capture variations in Hit Rate

In the third model we explored the neural predictors of Hit Rate. In line with the results from RT and Miss Rate, adding each neural component substantially enhanced the model's overall fit (N2c latency: $R^2_{adj} = 0.14$, $F(3,75) = 5.16$, $p = 0.003$; N2c amplitude: $R^2_{adj} = 0.14$, $F(4,74) = 4.20$, $p = 0.004$; CPP onset: $R^2_{adj} = 0.23$, $F(5,73) = 5.80$, $p < 0.001$; CPP slope: $R^2_{adj} = 0.23$, $F(6,72) = 4.82$, $p < 0.001$; CPP amplitude: $R^2_{adj} = 0.22$, $F(7,71) = 4.17$, $p < 0.001$; FCN slope: $R^2_{adj} = 0.22$, $F(8,70) = 3.72$, $p = 0.001$; FCN amplitude: $R^2_{adj} = 0.26$, $F(9,69) = 4.06$, $p < 0.001$), and CPP onset (stand. $\beta = -0.44$, $t = -3.90$, $p < 0.001$) and FCN amplitude (stand. $\beta = -0.34$, $t = -2.24$, $p = 0.03$) made significant individual contributions to the prediction of Hit Rate. When analysing the ADHD group, the examined neural components collectively explained 32% ($F(8,28) = 3.07$, $p = 0.01$) of the variance in Hit Rate with significant contributions of each component to the model's overall fit except for FCN slope ($F(7,29) = 2.43$, $p = 0.06$). When removing the non-informative variable (FCN slope) from the model, only CPP onset ($\beta = -0.33$, $p = 0.04$) showed independent power in predicting performance, consistent with the Miss Rate findings.

Together, these results provide compelling evidence that the EEG metrics of target selection (N2c), evidence accumulation (CPP), and dynamic urgency (FCN) collectively exhibit predictive power for decision-making performance across the three behavioural measures (RT, Miss Rate, Hit Rate). This underscores the importance of considering multiple neural components when investigating and interpreting decision-making processes. Importantly, it is worth noting that the contribution of the neural predictors to decision-making performance did not appear to substantially change in ADHD. While the neural signals showed varying contribution to each behavioural measure, CPP onset emerged as an independent predictor for performance variations across all the three measures (RT, Miss and Hit Rates). Figure 4

illustrates the relationships between CPP onset and performance, along with the correlation coefficients from Pearson's correlation tests.

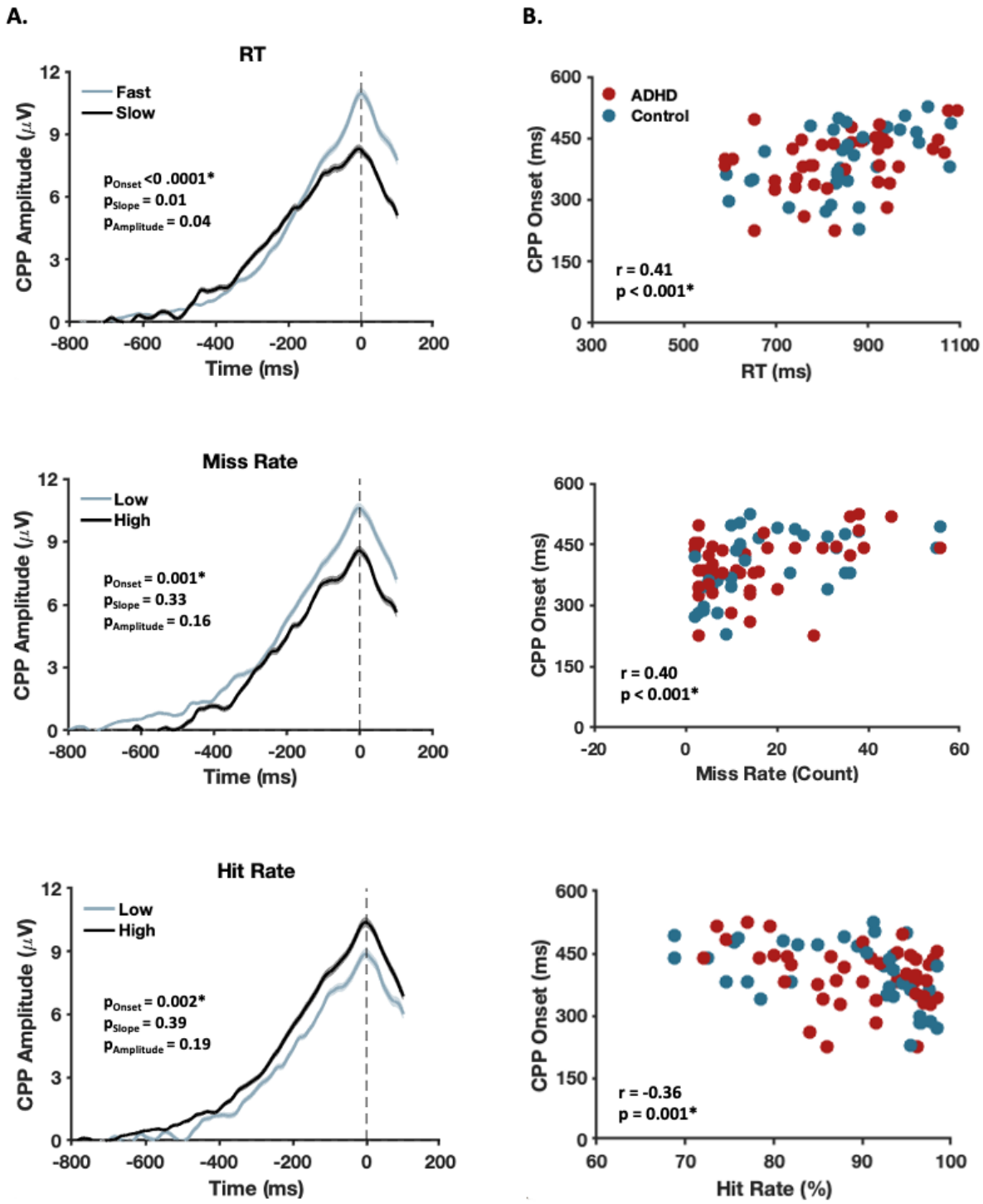

Figure 4. Relationship between CPP dynamics and performance. A. differences in CPP signal between the individuals with different levels of performance. Participants are binned by the behavioural measure using a median split of the data. P: P-Value from Wilcoxon-Signed Rank Test comparing the

two groups. **B.** The relationship between CPP onset and behaviour for both groups. P and rho: p-value and coefficient from Partial Correlation analysis using Pearson's correlation while controlling for Group and Site.

Group difference in Reaction Time is mediated by variations of evidence accumulation

These findings highlight the strong predictive power of the CPP for variations in behaviour during perceptual decision making. To further explore the importance of the group differences in evidence accumulation dynamics and shed light on the neurophysiological basis of perceptual decision making, we tested whether the differences between ADHD and control groups in task performance (RT, Hit Rate, Miss Rate) was mediated by the CPP (onset, slope and amplitude). Because the mediation effects of the different CPP metrics were likely related, for each behavioural measure, we jointly tested all the three mediators in one model to assess simultaneous effects more accurately (MacKinnon et al., 2007, 2000). Bootstrapped mediation analyses with 5000 samples (bias-corrected percentile; with site as confounding factor) revealed that the variability in ADHD-related deficits in RT, at least in part, depends on individual differences in the strength and rate at which sensory information is accumulated (Table 2).

Table 2. Mediation of CPP components on the impact of group on behaviour

									95% Confidence Interval	
		CPP Components			**Estimate**	**Std. Error**	**z-value**	**p**	**Lower**	**Upper**
Group	→	**Onset**	→	**RT**	-0.08	0.05	-1.51	0.13	-0.21	0.02
Group	→	**Slope**	→	**RT**	-0.24	0.10	-2.31	**0.02***	-0.45	-0.07
Group	→	**Amplitude**	→	**RT**	0.19	0.08	2.21	**0.02***	0.04	0.42
Group	→	**Onset**	→	**MR**	-0.06	0.04	-1.45	0.14	-0.21	0.01
Group	→	**Slope**	→	**MR**	-0.03	0.05	-0.57	0.56	-0.22	0.10
Group	→	**Amplitude**	→	**MR**	0.02	0.06	0.43	0.66	-0.13	0.20
Group	→	**Onset**	→	**HR**	0.06	0.04	1.43	0.15	-0.01	0.21
Group	→	**Slope**	→	**HR**	0.05	0.06	0.86	0.38	-0.06	0.22
Group	→	**Amplitude**	→	**HR**	-0.04	0.06	-0.75	0.45	-0.22	0.08

RT: Reaction Time; MR: Miss Rate; HR: Hit Rate. * Statistical significance

Collective predictive power of neural metrics on DDM parameters

Finally, we examined whether the EEG signatures of decision-making processes were associated with DDM parameters fitted to the behavioural measures by modelling each DDM parameter as a function of the EEG signals. Group and Site were entered as nuisance factors at the first stage of each model.

Non-decision time (t)

All of the neural metrics of decision-making significantly improved the model fit for the Non-decision Time parameter of the DDM (Figure 5A) explaining 24% of the variance (N2c latency: $R^2_{adj} = 0.24$, $F(3,75) = 9.11$, $p < 0.001$; N2c amplitude: $R^2_{adj} = 0.24$, $F(4,74) = 7.10$, $p < 0.001$; CPP onset: $R^2_{adj} = 0.24$, $F(5,73) = 5.97$, $p < 0.001$; CPP slope: $R^2_{adj} = 0.24$, $F(6,72) = 5.03$, $p < 0.001$; CPP amplitude: $R^2_{adj} = 0.23$, $F(7,71) = 4.37$, $p < 0.001$; FCN slope: $R^2_{adj} = 0.23$, $F(8,70) = 3.80$, $p < 0.001$; FCN amplitude: $R^2_{adj} = 0.24$, $F(9,69) = 3.73$, $p < 0.001$). However, none of the EEG signals demonstrated independent predictive power for this DDM parameter. The results were replicated when testing the ADHD group in isolation.

Drift Rate (v)

Similarly, the model fit for the drift rate was significantly improved when each of the EEG metrics were added into the model (Figure 5A). This model explained 15% of the variance in drift rate (N2c latency: $R^2_{adj} = 0.09$, $F(3,75) = 9.11$, $p = 0.02$; N2c amplitude: $R^2_{adj} = 0.08$, $F(4,74) = 7.10$, $p = 0.04$; CPP onset: $R^2_{adj} = 0.17$, $F(5,73) = 5.97$, $p = 0.002$; CPP slope: $R^2_{adj} = 0.18$, $F(6,72) = 5.03$, $p = 0.002$; CPP amplitude: $R^2_{adj} = 0.17$, $F(7,71) = 4.37$, $p = 0.005$; FCN slope: $R^2_{adj} = 0.16$, $F(8,70) = 3.80$, $p = 0.01$; FCN amplitude: $R^2_{adj} = 0.15$, $F(9,69) = 3.73$, $p < 0.01$) and CPP onset accounted for independent variation in drift rate (stand. $\beta = -0.36$, $t = -2.95$, $p = 0.004$). Notably, when specifically testing the ADHD group, none of the EEG signals significantly improved the model for drift rate, and their collective contribution to the overall fit was <10%.

Response Threshold (a)

The model fit for the Response Threshold parameter was significantly improved by the inclusion of CPP slope ($R^2_{adj} = 0.14$, $F(6,72) = 3.70$, $p = 0.008$) and amplitude ($R^2_{adj} = 0.19$, $F(7,71) = 3.22$, $p = 0.002$) and FCN dynamics (slope: $R^2_{adj} = 0.19$, $F(8,70) = 3.22$, $p = 0.004$; amplitude: $R^2_{adj} = 0.18$, $F(9,69) = 2.87$, $p = 0.06$), but not CPP onset ($R^2_{adj} = 0.02$, $F(5,73) = 1.38$, $p = 0.24$) or N2c measures (latency: $R^2_{adj} = 0.05$, $F(3,75) = 2.4$, $p = 0.08$; amplitude: $R^2_{adj} = 0.04$, $F(4,74) = 1.75$, $p = 0.15$) (Figure 5A). To obtain accurate parameter estimates for the

association between Response Threshold and neural components, without the influence of the non-informative signals, CPP (slope and amplitude) and FCN measures were entered into a separate model with the nuisance factors included. This model explained 20% of the variation and CPP slope showed independent predictive power for Response Threshold (stand. $\beta = -0.12$, $t = -3.30$, $p = 0.002$). When testing ADHD individuals separately, no evidence was found for a clear relationship between the neural metrics and the Response Threshold parameter. EEG signals collectively contributed only 12% to the overall fit.

The results above collectively provide evidence supporting the associations between neural metrics of decision-making as measured with EEG and DDM parameters derived from behavioural outcomes. Nevertheless, it appears that this relationship is influenced by additional factors in individuals with ADHD. Among all EEG components, the dynamics of CPP emerged as the strongest contributor to the variations in DDM parameters. Figure 5B illustrates the relationship between CPP dynamics and DDM parameters employing Spearman's partial correlation analysis while controlling for Site and Group. Notably, a significant correlation was found between CPP onset and drift rate, as well as CPP slope and response threshold.

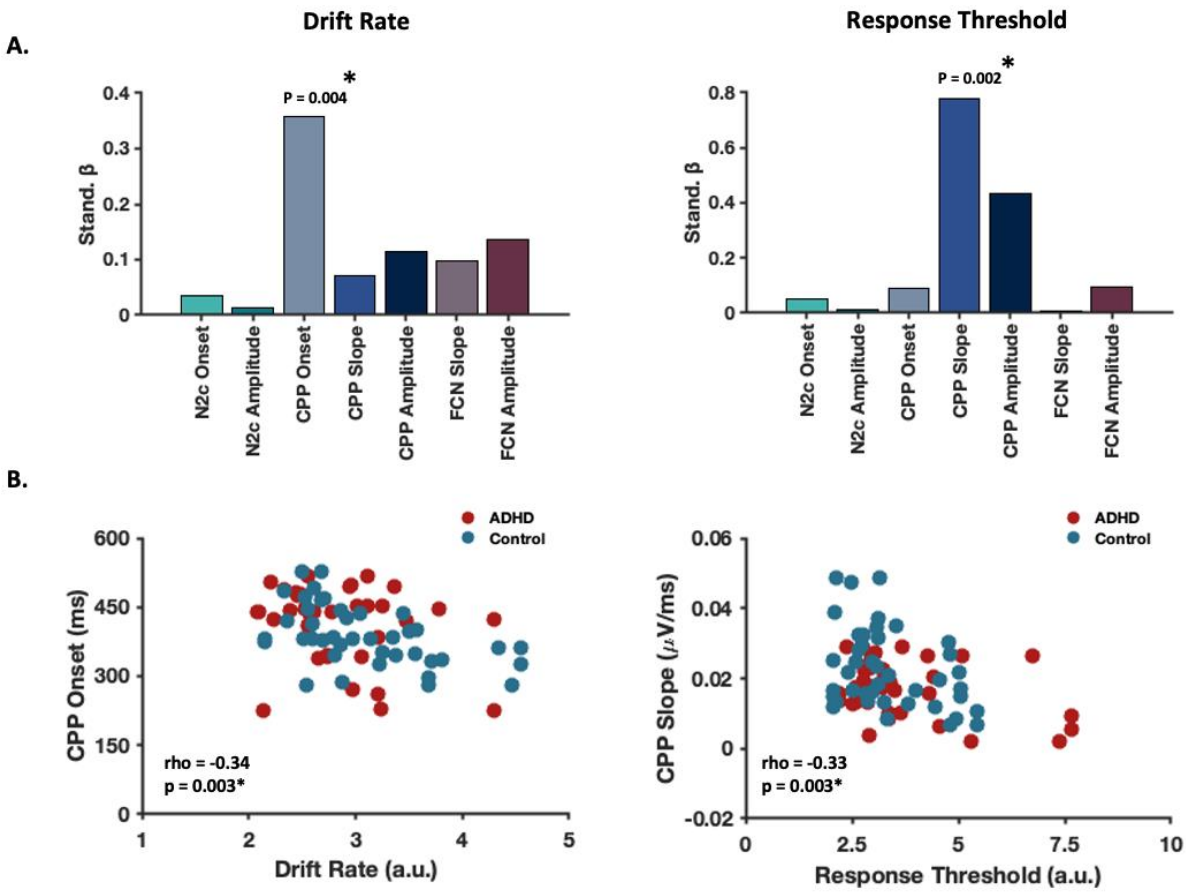

Figure 4. Relationships between DDM parameters and EEG signals. A. Results from the hierarchical regression models of DDM parameters indicating the predictive power of the EEG signals. * indicates that the EEG signal indicated independent predictive power for the DDM measure. Note that the standardised beta values are displayed as absolute values for the purpose of visualisation and the plots do not include the nuisance variables (Group and Site) that were entered into the model in the first step. **B.** Results from Spearman's partial correlation analysis depicting the relationship between the CPP component and DDM measures.

Clinical scores

Finally, we sought to determine whether the EEG signatures can serve as predictors for the clinical scores. Hierarchical regression models revealed that variations in the EEG signatures collectively accounted for a significant portion of ~70-80% (R^2_{adj}) of variance in each clinical score. The model fit for each score was significantly improved by adding each neural metric (all $p<0.001$). However, none of the EEG signals emerged as independent predictors for the clinical scores, in the general population or when specifically isolating the ADHD group.

Discussion

The prevalence of ADHD is among the highest of any neurodevelopmental disorder, but the efficacy of diagnostic and therapeutic pathways is limited by our incomplete understanding of its neuropsychological and neurobiological underpinnings. In addition, efforts to develop a unifying aetiological theory of ADHD have been complicated by its marked clinical heterogeneity (Coghill et al., 2014; Nigg et al., 2005) and high rates of concurrent comorbidity with other psychological disorders (da Silva et al., 2023; Jensen and Steinhausen, 2015). One promising avenue of research is the search for basic neural mechanisms that may contribute to the variety of higher cognitive deficits associated with the condition. In this study, we sought to develop a mechanistic account of differences in a fundamental cognitive process by integrating neurocognitive, neurophysiological, and computational levels of analysis. We detected several compelling signatures of phenotypic differences in ADHD.

Consistent with established effects in the literature (Karalunas and Huang-Pollock, 2013; Rommelse et al., 2007), the ADHD cohort demonstrated significantly slower RTs, a higher incidence of missed targets, and a reduced hit rate. In addition, children with ADHD exhibited altered dynamics in several neurophysiological signatures of the decision process. Specifically,

we detected differences in target selection as evidenced by the early and attenuated peak of N2c, dysfunction in evidence accumulation reflected in reduced slope and decreased amplitude of CPP and deficits in anticipation of volitional action as reflected in a reduced FCN slope. Critically, the interplay of these neural signals showed significant predictive power for inter-individual variations in both performance and ADHD clinical scores. The results specifically underscore the pivotal role of CPP onset, which emerged as a robust and independent neural predictor of decision-making performance across all the evaluated measures (RT, miss and hit rates). DDM parameters, however, did not show high sensitivity in distinguishing ADHD from typically developing children although they showed significant correlations with the dynamics of evidence accumulation as indexed by the CPP.

The N2c component is a key signature of early target selection mechanisms that support the decision process by facilitating enhanced processing of target features (Loughnane et al., 2016). While the marginally earlier latency of the N2c observed in children with ADHD could be interpreted as evidence of superior target detection, its diminished amplitude and the overall poorer performance of the ADHD group, including a higher rate of misses and fewer hits, is more consistent with immature processing of sensory information and/or insufficient allocation of attentional resources. Others have reported evidence of faster non-decisional processes in ADHD cohorts that compensate for slower rates of evidence accumulation, but have attributed this to inefficient processing, which may actually undermine performance (Karalunas and Huang-Pollock, 2013). N2c shares several key characteristics with the N2pc component elicited during visual search tasks, which has been shown to reflect attentional deficits in ADHD, as indicated by altered timing and reduced amplitude (Cross-Villasana et al., 2015; Luo et al., 2019; Wang et al., 2016). The N2pc is thought to reflect a combination of selection of attention and suppression of distractors, whereas the N2c is known as a more general target selection signal which emerges irrespective of the presence of distractors or the degree of spatiotemporal uncertainty (Loughnane et al., 2016). Although the N2c did not seem to act as an independent predictor of performance or clinical characteristics in ADHD, its significant contribution to models predicting both behavioural outcomes and DDM parameters suggests a partial link between ADHD-related decision-making deficits and the target selection mechanisms that support the decision process. N2c dynamics are relatively unexplored territory in the ADHD literature, but, given the centrality of attentional differences to ADHD, we believe it merits further investigation.

There is evidence that one of the key mechanisms that distinguishes cognitive processes in ADHD may be the rate of basic information processing(Alderson et al., 2007; Castellanos et al., 2005; Gonen-Yaacovi et al., 2016; Mihali et al., 2018; Rommelse et al., 2007; Salum et al., 2014b, 2014a) and efforts to model a variety of deficits associated with ADHD have consistently highlighted the rate of evidence accumulation as the core contributor to performance impairments, relative to typically-developing controls(Huang-Pollock et al., 2020, 2017; Karalunas et al., 2014, 2012; Shapiro and Huang-Pollock, 2019; Weigard and Huang-Pollock, 2017; Weigard and Sripada, 2021; Ziegler et al., 2016). Here, we sought to complement this work by tracing the dynamics of an established neurophysiological index of evidence accumulation, the CPP. CPP dynamics not only exhibited robust predictive power for all three measures of cognitive performance, but also differed significantly between ADHD and control groups. In line with claims of inefficient evidence accumulation in ADHD, we found a reduced CPP slope and a smaller CPP amplitude in the ADHD group. Consistent with previous research showing that steeper CPP slopes predict faster reaction time and CPP amplitude is reliably greater for hits than for misses (Kelly and O'Connell, 2015, 2013; O'connell et al., 2012), this result provides neurophysiological evidence to support the hypothesis that suboptimal evidence accumulation or poorer signal-to-noise ratios in the processing of task-relevant information may contribute to deficits in decision making in ADHD.

To our knowledge, this is the first study to investigate CPP dynamics in ADHD. However, the CPP is thought to be functionally equivalent to the P300 event-related potential[1] (O'Connell and Kelly, 2021; Twomey et al., 2015) and the P300 has received significant attention in ADHD research. In line with our results, reduced P300 amplitude and delayed onset in response to both auditory and visual targets have been consistently reported in research on ADHD across a variety of tasks (Hasler et al., 2016; Itagaki et al., 2011; Johnstone et al., 2013; Kaiser et al., 2020; Szuromi et al., 2011), including many of the tasks where modelling studies indicated reduced drift rates in ADHD groups. In fact, these effects are sufficiently robust that P300 dynamics have been proposed as potential ADHD biomarkers (Kaiser et al., 2020) and metrics for research on pharmacological treatment (Ogrim et al., 2016; Peisch et al., 2021; Yamamuro

[1] The CPP is typically observed during extended perceptual discrimination, while the P300 is evoked by discrete sensory events (e.g., an oddball stimulus).

et al., 2016). While the interpretation of these P300 effects varies across tasks, these results can be broadly characterised as reflecting suboptimal processing of task-relevant information.

The neural inefficiency of decision formation observed in ADHD aligns with the neurobiological changes associated with this condition. ADHD has been widely associated with an imbalance in dopamine and glutamate levels. The release of striatal dopamine into the prefrontal cortex suppresses the release of glutamate subsequently inhibiting the behavioural response to the stimuli (Russell, 2003). Consequently, decreased dopamine release triggers an overproduction of glutamate, amplifying responsiveness to stimuli and increasing distractibility, which may ultimately contribute to the observed inattention in ADHD (Purkayastha et al., 2015). Multiple lines of evidence support contributions of both dopamine and glutamate to the formation of perceptual decisions. Recent biophysical modelling of perceptual decisions suggests that NMDA receptors, that regulate the release of glutamate in the brain, are critical for integrating sensory information over time (Wang, 2008; Wong and Wang, 2006). It is hypothesised that NMDA receptors stabilise network dynamics and enable graded evidence accumulation, and therefore their impairment in ADHD may result in less efficient evidence accumulation, including slowed build-up rate. Moreover, recent computational models suggest that competition between the dopaminergic receptors D1 and D2 in cortical-basal ganglia-thalamic pathways may impact decision-making, with D1 activity linked to evidence accumulation rate and D2 activity to threshold-setting, and execution of the motor command (Dunovan et al., 2019). These notions align with research that indicates methylphenidate (MPH) enhances cognitive task performance by improving evidence accumulation. MPH is the mainstay of treatment for ADHD and works by inhibiting reuptake of catecholamines including dopamine. Specifically, DDM studies have shown that MPH normalises the lower drift rate in ADHD (Fosco et al., 2017), it realigns P300 features like onset, build-up rate, and amplitude in neurocognitive (Peisch et al., 2021) and perceptual decision making tasks (Loughnane et al., 2019), and there is preliminary evidence suggesting that MPH increases CPP slope in human EEG. The neural mechanisms by which MPH might enhance evidence accumulation are still largely unknown although some evidence from behavioural model studies suggests that it may regulate the suboptimal neural signal-to-noise ratios in children with ADHD (Loughnane et al., 2019; Pertermann et al., 2019; Ratcliff et al., 2009)suggested that an increase in neural gain may account for the effect they observed on the P300 . Future studies may yield a deeper understanding of the pharmacology of discrete processing stages underlying human choice behaviour by integrating the EEG paradigm and

computational modelling approach employed in the present study with pharmacological manipulation.

Despite a trend towards a reduced drift rate in ADHD, we did not observe strong sensitivity in DDM parameters to differentiate between the two groups. This stands in contrast to a substantial number of reports in the literature employing a range of neurocognitive paradigms (Weigard and Sripada, 2021). One possibility is that the more basic perceptual decision-making task used in this study did not dissociate drift rate differences as vividly as other tasks that place greater strain on cognitive resources (e.g., go/no-go or continuous performance task). However, it should be noted that other studies with similar numbers of participants and trials, requiring simple decisions about basic stimuli (e.g., arrows) have reported drift rate effects associated with ADHD (Metin et al., 2013). It is difficult to concretely attribute the absence of a drift rate effect to a particular aspect of the paradigm because there are myriad factors that shape the decision process and these often do not precisely map onto DDM parameters. Indeed, in the present study, the finding that collective variations of multiple neural signals predicted changes of each DDM parameter illustrates that these parameters are the product of multiple decision processes. In the context of the poorer overall performance of the ADHD group in our study, the diminished CPP slope in ADHD participants, and the consistent finding of drift rate effects in the ADHD literature, we are mindful that while the drift rate comparison was marginally short of statistical significance, this is not itself evidence that there is no drift rate difference. Alternatively, this may result from the inherent constraints of behavioural models to disentangle the discrete levels of neurocognitive processing. Unlike neural models, DDM does not specifically assess the efficiency of sensory encoding, integrity of evidence accumulation, or the involvement of additional processes that support perceptual decision-making such as attentional selection that are known to be affected in ADHD (Cross-Villasana et al., 2015; Luo et al., 2019; Wang et al., 2016). In general, a large body of research on non-human primates revealed complex neural computations underlying decision formation that may go undetected in the abstract decision models like DDM that are tailored only to behaviour. Among all the neural signatures examined, CPP dynamics demonstrated the strongest associations with variations in DDM parameters. Specifically, delayed CPP onset, which is an indicator of inefficient decision formation, independently predicted lower drift rate estimates. Furthermore, slower build-up rate in the CPP predicted lower response thresholds, which may hint at a compensatory strategy: accepting a lower, but achievable standard of evidence, given weaker evidence accumulation. However, it should be noted that studies fitting the DDM to data from

a variety of neurocognitive tasks have mostly reported no difference in response thresholds between ADHD and control groups (Huang-Pollock et al., 2012; Karalunas et al., 2018, 2014; Karalunas and Huang-Pollock, 2013; Weigard and Huang-Pollock, 2017) but see (Mulder et al., 2010). Given the unique value of computational models in bridging behaviour, neurophysiology and latent psychological processes, future studies employing joint neural-behavioural model fitting may offer deeper understanding of ADHD-related changes in decision-making.

Finally, our data revealed ADHD-related changes in FCN dynamics, which also contributed to the prediction of variations in performance. FCN corresponds to the contingent negative variation (CNV) signal commonly observed in target detection and choice response time tasks, which is associated with temporal preparation for anticipated events or volitional movements (Baker et al., 2012; Brunia and Van Boxtel, 2001; Van Rijn et al., 2011). The CNV is influenced by dopaminergic systems (Birbaumer et al., 1990) and its attenuation has been widely reported in children (Banaschewski et al., 2003; Doehnert et al., 2013; Kaiser et al., 2020; Perchet et al., 2001) and adults with ADHD (Hasler et al., 2016; Mayer et al., 2016; McLoughlin et al., 2011, 2010). Indeed, this frontocentral negative signal has been suggested as a robust neurophysiological marker of ADHD which effectively captures the underlying deficits in their preparatory and anticipatory motor processes (Doehnert et al., 2013; Kaiser et al., 2020). In the context of perceptual decision-making, the FCN is also described as a neural index of urgency which grows in a time-dependent but evidence-independent manner reflecting speed pressure in response. Therefore, a reduced amplitude and slope of FCN in ADHD is suggestive of deficient anticipatory processing and response preparation. Indeed, if the rate of evidence accumulation was slowed in the ADHD group, one might expect that increased, rather than decreased, urgency to reach decision commitment could be used to compensate. It appears that such strategic adjustment was not adaptive as the ADHD groups had slower and less accurate responses on average. Although numerous studies suggest that ADHD participants' decision boundaries do not differ from those of control groups, there is some evidence that they may have difficulty flexibly adjusting the inherent trade-off between speed and accuracy in response to task demands (Mulder et al., 2010). It is possible that dysregulation of response preparation, and specifically the timing mechanism associated with the CNV may contribute to this relative inflexibility.

Conclusions

The present study provides novel neurophysiological evidence linking decision-making deficits in ADHD to the alterations in the dynamics and interplay of the neural signals indexing three key cognitive processes: target selection, decision formation and dynamic urgency. Neurophysiology can complement and inform computational analysis of neurocognitive differences, which is increasingly popular in ADHD research. Here, we offer an integrated account of these differences in perceptual decision making and establish neural signals that can serve as critical guidance in constructing or constraining mechanistic accounts in future ADHD research.

Declaration of competing interests

The authors declare no conflicts of interest or competing interests.

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