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Behavioral and neurofunctional profiles of delay aversion in children with attention-deficit hyperactivity disorder

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Despite substantial efforts to unravel cognitive heterogeneity in ADHD, the examination of motivational variability, particularly delay aversion, remains limited. This study aimed to identify homogeneous delay-averse profiles in children with ADHD to understand motivational deficits. Delay-averse profiles were examined in a clinically well-characterized sample of 43 children with ADHD and 47 control participants using cluster analyses on an experiential delay discounting task. External validation analyses included parents' and teachers' clinical ratings, and fNIRS-based resting-state functional connectivity (rsFC) from the frontoparietal (FPN) and the default mode (DMN) networks. A five-profile solution best fit the data. Two clusters, labeled *Conventional* and *Conventional-steeper*, exhibited a conventional reward discount with increased delay but differed in the discounting slope. Three clusters demonstrated altered discounting: *Steep discounting* (abrupt devaluation of the reward), *Shallow discounting* (shallow discounting), and *Zero discounting* (no devaluation across delay durations). 77.78% of ADHD-C children clustered into steep discounting profiles, while 41.67% of ADHD-IN children were found in Shallow and Zero profiles, showing a significant disparity in the distribution of categorical presentations. External validation showed no differences in clinical ratings. However, clusters showing *Zero* and *Shallow discounting* demonstrated hypoconnectivity within and between FPN and DMN nodes. Delay aversion in ADHD spans a continuum from decreased to increased discounting rather than being solely defined by steeper discounting. These findings highlight the relevance of dimensional approaches in capturing ADHD's motivational heterogeneity and identifying distinct neurobiological substrates, with implications for improving diagnostic protocols and intervention strategies through the incorporation of behavioral measures of reward processing.

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INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neuropsychological heterogeneous condition in which multiple causal pathways [1, 2] contribute to behavioral deficits manifesting as inattention, impulsivity, and hyperactivity symptoms [3]. This heterogeneity persists even within each clinical subtype of the disorder, complicating the process of differential diagnosis and effective treatment guidance [4, 5]. In response, over the last decade, clinicians and researchers have increasingly adopted data-driven approaches to unravel novel neurocognitive phenotypes of the disorder [6]. The ultimate goal is to identify and characterize new ADHD subgroups that may enhance current nosology and diagnosis, and clarify etiological pathways to the disorder [5].

Despite substantial efforts to unravel cognitive heterogeneity in ADHD [6], the exploration of motivational styles, particularly the phenomenon of delay aversion, remains comparatively limited. Prominent theoretical models of ADHD have acknowledged the unique and independent contribution of delay aversion to prototypical ADHD symptoms [1, 7, 8]. These theories support that some individuals with ADHD exhibit "delay-averse" behaviors (e.g. fidgeting, excessive talking, distractibility) to avoid the negative feelings associated with waiting. Meta-analyses indicate that, compared to control participants, individuals with ADHD show a steeper decline in the value of delayed reinforcers (i.e. steeper discounting behavior), demonstrating a greater preference for immediate rewards even if they are less valuable [9, 10]. However, similar to the great variability observed in executive functioning [4, 11] and temperamental traits [12], only a subset of ADHD individuals is estimated to present delay-aversion deficits [13].

Delay aversion is commonly assessed using Delay Discounting Tasks (DDTs), where participants choose between smaller-immediate and larger-delayed rewards. Given that the waiting period is the primary factor exacerbating delay discounting in ADHD [10], DDTs employing real delays hold greater ecological validity and better discriminate between ADHD and non-ADHD children [14–16]. Nonetheless, paradigms employing real waiting periods are infrequent, and findings are often inconsistent due to the heterogeneity of categorical ADHD presentations, particularly within the inattentive presentation (ADHD-IN) [16, 17]. This subgroup includes children exhibiting subthreshold hyperactive-impulsive symptoms as well as those with purely inattentive profiles, who do not experience impairments in inhibitory control [18–21], and theoretically, are not expected to exhibit delay aversion deficits.

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Therefore, in line with current initiatives [22, 23], adopting a dimensional approach to investigate experiential delay discounting behavior in ADHD may prove useful in understanding motivational deficits in this population, and explaining mixed findings in delay aversion research. Dimensional analyses have revealed that the steepness of delay discounting is associated with the severity of ADHD symptoms, surpassing the arbitrary cut-offs for ADHD presentations [14, 24, 25]. Additionally, from a clinical perspective, this approach may identify clinically relevant phenotypes to guide nosology reframing, and, by incorporating neurobiological levels of analyses, advance the understanding of ADHD pathogenesis.

In this study, we aimed to examine delay-averse motivational profiles among children with and without ADHD. To justify our data-driven approach, we first demonstrated the inability of traditional presentations to parse motivational heterogeneity and elucidate neurobiological correlates in ADHD [26]. We evidenced no differences between ADHD presentations in experiential delay discounting (Fig. S1), nor resting-state functional connectivity (rsFC) (Fig. S2). Subsequently, we applied clustering procedures to DDT performance to identify homogeneous subgroups. Although this study is primarily exploratory, drawing parallels with cognitive [27–29] and temperamental [12] subtyping studies, we hypothesized the identification of three to five subgroups. Then, we further characterized these subgroups using clinical ratings from parents and teachers, and fNIRS-based brain functional connectivity. We expected that these profiles would contribute to a comprehensive understanding of the motivational pathways involved in childhood ADHD.

MATERIALS AND METHODS

Participants

The sample comprised 90 participants aged 7–16 years: 43 children diagnosed with ADHD according to DSM-5 criteria and 47 age- and IQ-matched control participants (Table 1). Participants were recruited for a comprehensive executive functions project at the University of Almeria (Spain) through public and private health and education services in this province. Diagnostic assignment to ADHD or control groups was conducted by an experienced psychologist based on the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS-PL-5) [30], and parents' and teachers' ratings of current ADHD symptoms (ADHD Rating Scale- fifth edition —ADHD-RS-5— [31], Strengths and Difficulties Questionnaire —SDQ— [32]). Exclusion criteria included neurological or genetic disease, traumatic brain injury, serious comorbidities (e.g. intellectual disability, psychosis, ASD), or IQ < 70 (two-subtest short-form [33] of the WISC-V [34]). ADHD children discontinued medication at least 24 h before measurements. Control children had no neurological or psychiatric history.

All parents/legal guardians and children over 12 years of age provided verbal and written informed consent. Ethical approval was obtained from the Bioethics Committee of the University of Almería [UALBIO2017/018] and the Torrecárdenas University Hospital [PSI2015-70037-R]. The study was performed under the ethical standards of the World Medical Association Declaration of Helsinki. Personal information was treated in compliance with current EU and Spanish General Data Protection Regulations.

Measures

Experiential delay discounting task. We developed an experiential DDT in E-Prime 3.0 using well-established parameters from prior research on delay aversion in ADHD [15, 25, 35]. The task consisted of 40 experimental trials in which children pressed a button to choose between a small immediate reward (2, 4, 6, 8 coins) and a larger but delayed one (10 coins delivered after 5, 10, 20, 30, or 60 s). Each trial began with a jittered fixation point (1000–2000 ms). Next, choices were visually represented by two treasure islands. Children had to decide whether to stay on an island with a small treasure (Fig. 1A) or navigate to a distant island for a larger treasure (Fig. 1B). If they decided to navigate, a black screen with a white clock appeared to represent the waiting time (Fig. 1B). Delays were visually represented by vertical lines indicating the distance between the two islands, with greater distances corresponding to longer delay durations. However, children were not informed about the exact duration of each delay. Two feedback screens followed each choice, displaying the number of coins earned in the trial (1500 ms), and the updated total coins earned in the task (2000 ms).

The task also included 5 'catch' trials to verify whether children were paying attention and making choices as expected [35, 36]. In catch trials, children had to choose between receiving 10 coins immediately (optimal decision) or 10 coins after a delay (Fig. 1C). Experimental and 'catch' trials were randomly presented, and the right-left positioning for immediate and

Table 1. Demographic and clinical characteristics of ADHD and control participants.

Characteristic	ADHD	Control
Demographics		
n	43	47
Mean (SD) age (years)	11.32 (3.18)	10.68 (2.44)
Mean (SD) IQ	93.79 (16.39)	100.21 (15.52)
n (%) of girls	12 (27.91)*	25 (53.19)
n (%) of European origins	40 (93.02)	42 (89.36)
n of C:I:H presentations[a]	18:24:1	-
n (%) on medication[b]	24 (55.81)	-
Mean (SD) hours in wash-out	89.04 (116.97)	-
n (%) of Learning Disorder	12 (27.91)	2 (4.26)
n (%) of Mood Disorder	1 (2.33)	2 (4.26)
n (%) of ODD	7 (16.28)	-
n (%) of Language Disorder	-	1 (2.13)
Mean (SD) ADHD-RS-5-Parents		
Inattention	19.51 (5.59)**	6.29 (6.52)
Hyperactivity-Impulsivity	13.12 (7.02)***	5.36 (5.42)
Total scale	32.63 (10.96)***	11.65 (9.92)
Mean (SD) ADHD-RS-5-Teachers (SD)[c]		
Inattention	15.83 (6.74)***	6.79 (7.35)
Hyperactivity-Impulsivity	6.66 (5.94)	3.26 (4.85)
Total scale	22.49 (10.65)*	10.05 (10.88)
Mean (SD) SDQ Subscales–Parents		
Emotional symptoms	4.67 (2.30)**	2.67 (2.30)
Conduct problems	3.33 (2.24)**	1.44 (1.65)
Inattention/Hyperactivity	6.88 (2.06)***	3.28 (2.51)
Peer problems	3.47 (2.35)**	1.82 (1.88)
Prosocial behavior	7.61 (2.27)	8.65 (1.36)
Total difficulties	18.35 (6.48)***	9.20 (6.10)
Mean (SD) SDQ Subscales–Teachers[c]		
Emotional symptoms	3.10 (1.71)**	1.61 (1.76)
Conduct problems	2.12 (2.35)	0.95 (1.71)
Inattention/Hyperactivity	5.64 (2.08)**	2.82 (2.59)
Peer problems	1.82 (2.03)	1.24 (1.62)
Prosocial behavior	6.72 (2.45)	7.55 (2.55)
Total difficulties	12.67 (5.88)*	6.61 (6.47)

No significant differences in age ($W = 802.50$, $p = 0.094$) or IQ ($t(86.21) = -1.91$, $p < 0.060$) but sex ($\chi^2(1) = 5.93$, $p = 0.015$) and comorbidity ($\chi^2(1) = 11.50$, $p < 0.001$). ADHD-RS and SDQ comparisons were performed using General Linear Models, with sex and comorbidities as covariates. ***$p < 0.001$, **$p < 0.01$, *$p < 0.05$.

[a]One participant had a subthreshold ADHD-IN profile (three to five criteria leading to incapacitating symptoms [94]).

[b]91.67% Methylphenidate, 8.33% Lisdexamfetamine, 4.17% Atomoxetine, 20.83% Guanfacine.

[c]Information missing for 9 ADHD children and 9 controls.

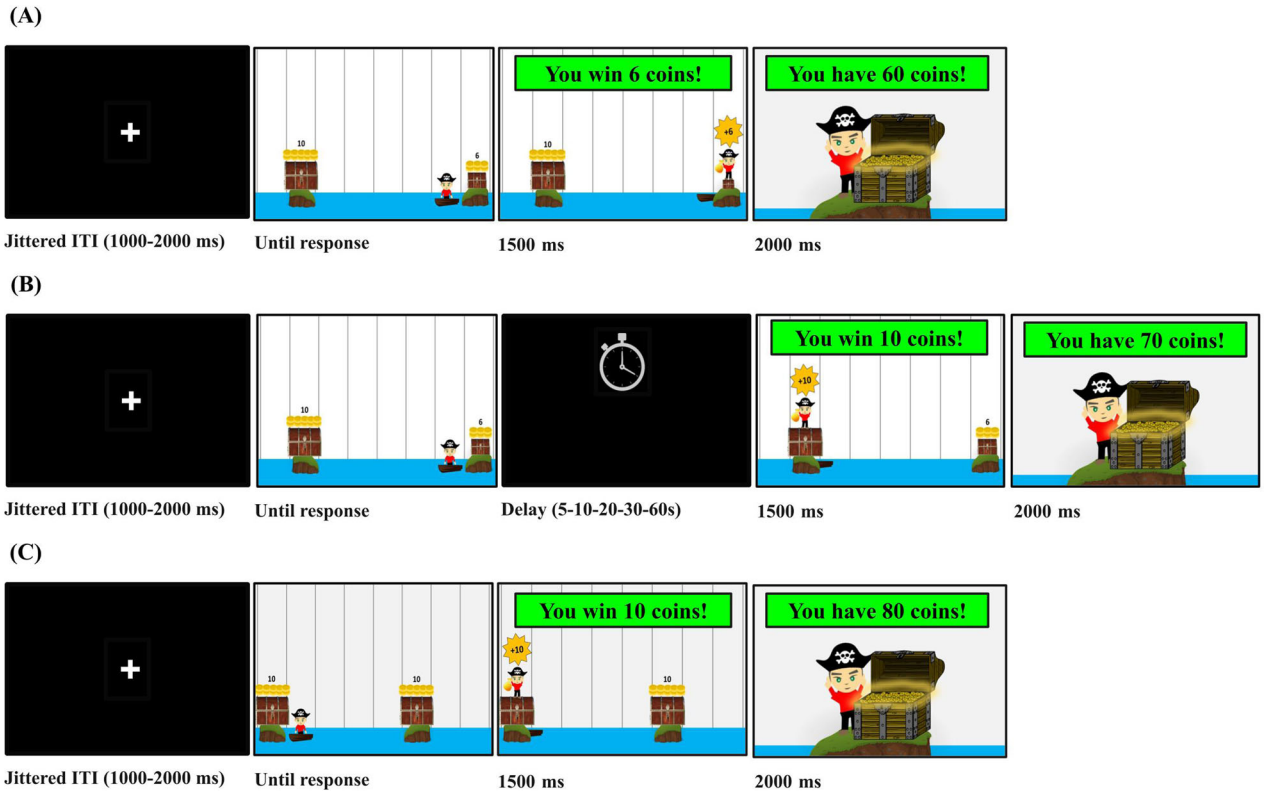

Fig. 1 Design of the experiential Delay Discounting Task. A Example of immediate choice. **B** Example of a 30-s delayed choice. **C** Example of a 'catch' trial.

delayed choices was counterbalanced. Participants were informed that there was a fixed number of trials and were encouraged to choose based on their preferences, as there were no right or wrong answers. Before performing the task, participants completed 10 practice trials to experience each level of delay. The "subjective value" (SV) of the delayed reward (i.e. the indifference point) at each delay was calculated as the main outcome measure using previously described procedures [17] (Table S1) The greater the SV, the greater the ability to wait for larger rewards.

Clinical and neurofunctional measures

Clinical ratings: Cluster profiles were characterized using parents' and teachers' reports of ADHD symptoms (ADHD-RS-5, SDQ), parents' reports of externalizing and internalizing behaviors (Child Behaviour Checklist — CBCL6-18— [37]), and parents' reports of behaviors associated with executive functions in daily routines (Behaviour Rating Inventory of Executive Function-2—BRIEF-2— [38]).

Resting-state functional connectivity: *fNIRS acquisition*: We used two portable continuous-wave fNIRS systems in tandem mode (NIRSport, NIRx Medical Technologies LLC) to record the relative changes in the concentration of oxyhemoglobin (HbO2) of the main cortical areas of the FPN and DMN during 8 min of resting state. The sampling rate was 3.41 Hz. We used a custom probe array of 31 optodes (16 light sources and 15 detectors at two wavelengths, 760 nm, and 850 nm) according to the International 10–10 montage system with an inter-optode distance of approximately 30 mm (Fig. 2A). The source-detector configuration was selected using the fNIRS Optodes' Location Decider tool [39] and resulted in 34 fNIRS measurement channels covering seven regions of interest (ROIs) from the FPN — dorsolateral prefrontal cortex (DLPFC), superior parietal lobe (SPL), premotor and supplementary motor area (preSMA), frontopolar cortex—, and six ROIs from the DMN —orbitofrontal cortex (OFC), inferior parietal lobe (IPL), middle temporal gyrus (MTG)—. Precise coordinates for each ROI are detailed in supplementary material (Table S2). Probe spatial sensitivity was evaluated using AtlasViewer software [40] (Fig. 2B).

Pre-processing and connectivity analyses: fNIRS data was missing for 1 ADHD child (poor signal due to afro-textured hair) and 1 control participant (missing data). fNIRS signals were pre-processed using MATLAB [41]. Saturated channels were replaced with high-variance noise and missing values were interpolated. Next, the signals were downsampled to 1 Hz and converted to optical density values. We applied the modified Beer-Lambert Law to derive the relative changes in the concentration of HbO2 [42]. To address potential confounding signals derived from systemic physiological noise and motion artifacts, we implemented pre-whitening and pre-weighting correction methods [43–45]. We computed rsFC in the time domain using a whole-brain correlation method. Pearson correlation analyses were conducted on the time series data for every pair of ROIs to determine the functional connectivity between the designated areas, resulting in 78 rsFC outcomes. In this context, functional connectivity was defined as the strength of the correlation in the hemodynamic activity for each pairwise comparison.

Procedure

Assessments took place in a well-acclimated and soundproofed experimental room and were always monitored by an experienced researcher. First, children completed the rsFC recording followed by the experiential DDT. During the fNIRS recording, children were instructed to sit in a relaxed position and stay as still as possible, keeping their eyes open and looking at a fixation point on the computer screen. Children were also directed not to fall asleep, touch the cap, or playfully move their eyebrows. The children's field of vision was free from any sources of distraction. The fNIRS recording continued during the execution of the DDT for additional research purposes. Before commencing the DDT, children were informed that if wearing the fNIRS cap caused any discomfort, they should promptly notify the experimenter for its removal, thereby avoiding potential biases in task performance (e.g., opting for immediate choices to complete the task quickly and remove the cap). Upon task completion, children responded to an ad-hoc questionnaire addressing sensations associated with the fNIRS recording. They were explicitly asked whether any sensation (e.g. itching, headache) affected their task performance. No participant reported such an influence. Therefore, no data from the DDT were excluded due to fNIRS-related discomfort affecting task performance.

Statistical analyses

Statistical analyses were conducted using R software [46]. We checked "catch" trials to exclude participants who did not choose as expected (i.e.

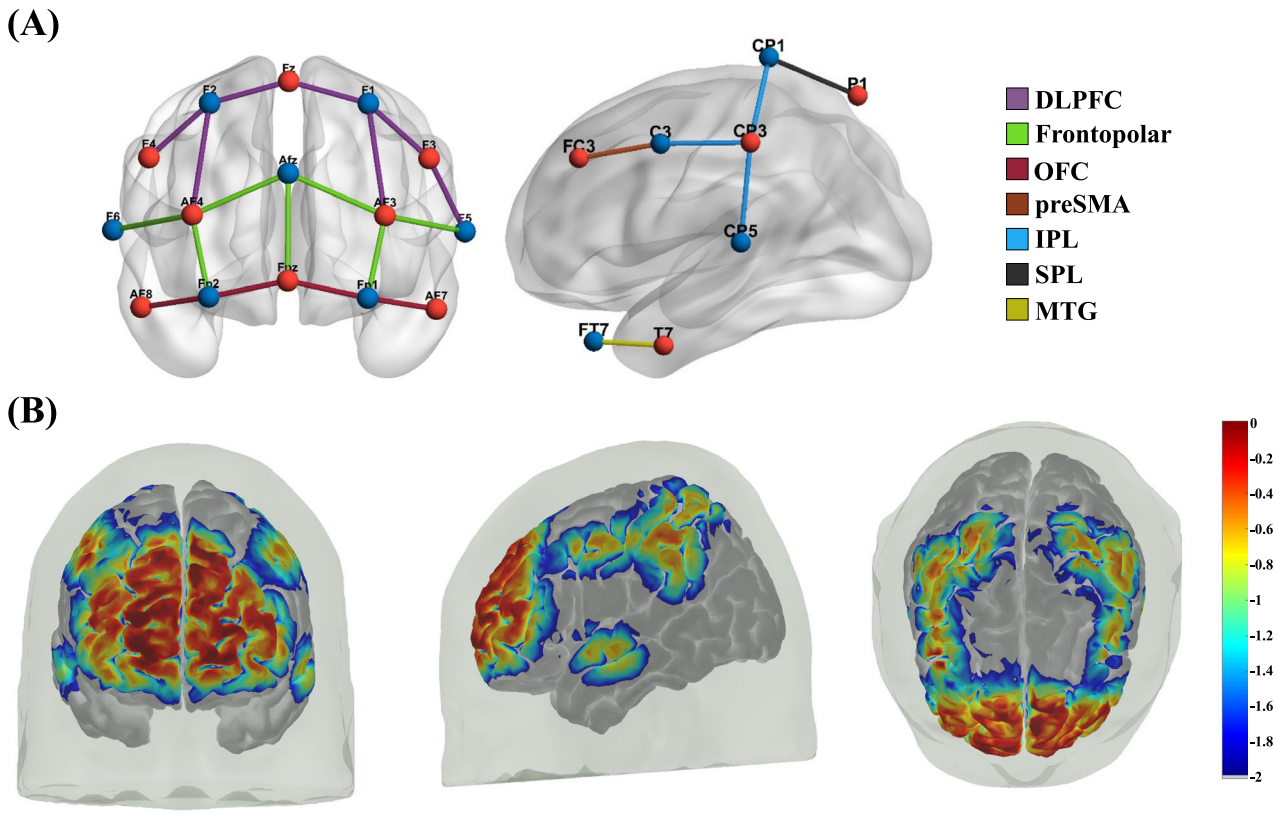

Fig. 2 Optode placement for the fNIRS assessment. A Red and blue dots represent sources and detectors respectively. Lines represent measurement channels covering the main cortical nodes of the FPN and DMN. **B** Spatial sensitivity profile, in log10(mm^{-1}), for each measurement channel on the cortical surface after performing a Monte Carlo photon migration. Greater sensitivity is indicated in red. DLPFC dorsolateral prefrontal cortex, OFC orbitofrontal cortex, SPL superior parietal lobe, IPL inferior parietal lobe, MTG middle temporal gyrus, preSMA premotor and supplementary motor area.

chose the immediate option) on more than half of the occasions (≥3). No participants were excluded for this reason.

We applied hybrid k-means clustering algorithms over the whole sample on the standardized SV of the delayed reward at each delay to identify novel delay-averse profiles. Hierarchical methods (Ward's method on Euclidean distance) selected a tentative number of cluster centroids minimizing within-cluster variance in each iterative step. Next, cluster membership was determined through k-means analysis using the previously defined cluster centroids. This combination of clustering methods overcomes the limitations of each [47] and has been applied in previous subtyping studies in children [28] and adults [48–50] with ADHD. We examined several cluster solutions (*k*) ranging from three to five subgroups [27, 28, 48, 49, 51–53]. We decided on the optimal cluster solution guided by the visual inspection of each cluster solution and the majority rule of thirty clustering validation indices [54].

Two-way (Cluster x Delay) ANOVA was performed to analyze DDT performance. One-way ANOVAs were performed to compare demographic features and clinical ratings. When data violated statistical assumptions, we applied robust statistics on 10% trimmed means and 2000 bootstrap samples for better control of type-I error [55–57]. Post-hoc tests were adjusted for multiple comparisons using Benjamini-Hochberg or Bonferroni corrections for robust and non-robust models respectively. The significance level was set at $p < 0.05$ (two-sided).

We developed a Bayesian model (supplementary material) [49] to compute the extensive number of mean comparisons among cluster profiles in each of the 78 rsFC outcomes [58]. Statistical decisions were made using the 95% Highest Density Intervals (HDIs) in conjunction with Regions of Practical Equivalence (ROPEs). These ROPEs establish a range around specific values of interest, such as zero in the case of estimating differences between means. If the HDI entirely falls outside the ROPE, we conclude that values within the ROPE are not credible [59]. When assessing mean differences in rsFC outcomes across various groups, we considered those differences where the 95% HDIs exclude the value of 0. Given that rsFC outcomes represent the correlation between two areas and considering our exploratory approach, we lack a priori knowledge about what extent of change would constitute a meaningful difference. Bayesian analyses were performed using the RStan package [60]. For each analysis, we extracted 12,000 samples using Markov Chain Monte Carlo sampling, each of the 4 chains having 4000 warmup samples and saving 8000 samples.

The required sample size, calculated using G*Power (v.3.1.9.7) [61], ranged from 69 to 90 participants, depending on the number of groups (three to five; categorical ADHD presentations/possible cluster solutions according to hypotheses). This calculation was based on a small effect size ($f = 0.15$), $\alpha = 0.05$, power = 0.80, and five repeated measures conditions (delay periods) with a correlation of 0.5.

RESULTS

Categorical analysis

Analyses between categorical ADHD presentations did not reveal significant differences in either DDT performance (Tables S3 and S4) or the rsFC of FPN and DMN networks (Table S5). Detailed results are provided in the supplementary material.

Cluster analysis and characterization

Clustering validation indices selected a five-cluster structure as the best cluster solution for explaining variability in DDT performance in our sample (Fig. S3, Table S6). This solution was also congruent with heterogeneity on discounting behavior in general and clinical populations (Fig. 3A) [62, 63]. Cluster profiles were labeled according to this literature. We identified two subgroups showing a conventional delay-reward trade-off: (1) *Conventional DD* ($n = 18$) included participants who exhibited a systematic discount of the reward with increased delay; (2) *Conventional-Steep DD* ($n = 35$) consisted of participants with a conventional DD profile but showing a slightly steeper devaluation of the reward. Three

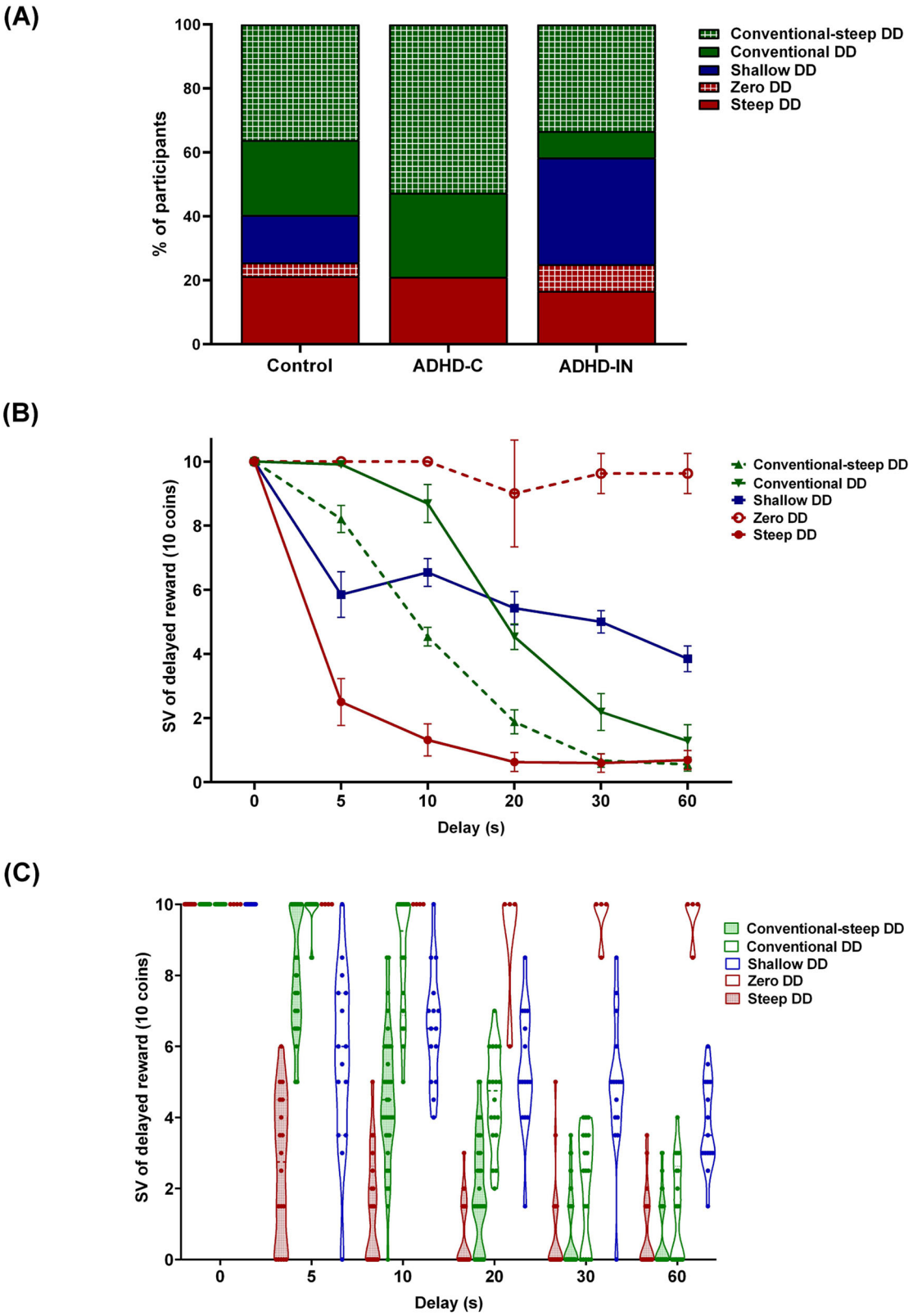

Fig. 3 Cluster profiles of experiential discounting behavior. A Percentage distribution of each delay-averse profile within ADHD and control groups. **B** 10% trimmed mean and 10% trimmed SEM values of the SV of the delayed reward in the DDT. **C** Individual SV values of the delayed reward for each cluster and each delay. Significant effects after adjustment for multiple comparisons are detailed in supplementary material (Tables S7 and S8).

additional subgroups demonstrated altered discounting: (3) *Steep DD* ($n = 18$) included participants characterized by an abrupt and significant devaluation of the reward. *Shallow DD* ($n = 15$) comprised participants displaying a shallow discounting of the reward. *Zero DD* ($n = 4$) included participants showing no systematic devaluation of the reward across delay durations. Graphical inspection of three- and four-cluster structures are presented in Fig. S4.

Robust two-way mixed ANOVA showed significant Cluster x Delay ($T_{WJ(16,\ 37.75)} = 63.08$; $p < 0.001$), Delay ($T_{WJ(4,29.37)} = 83.52$; $p < 0.001$) and Cluster profile ($T_{WJ(4,\ 17.24)} = 134.94$; $p < 0.001$) effects on the SV of delayed reward (Fig. 3B, C). Test statistics and adjusted post-hoc comparisons are provided in supplementary material (Tables S7 and S8). Analyses revealed significant differences among cluster profiles at each delay except for the 60-s delay interval, in which Steep DD, Conventional DD, Conventional-Steep DD, and Steep DD cluster profiles exhibited the same SV. The SV of delayed reward significantly decreased as a function of delay for Conventional DD and Conventional-Steep DD profiles, whereas no discounting was found for Steep, Zero, and Shallow participants.

Demographic characteristics are summarized in Table 2. Clusters significantly differed in age ($F(4) = 4.97$, $p = 0.001$, $\eta^2 = 0.19$) but not in IQ ($F(4)$ 1.91, p =0.012) sex ($p = 0.15$) or comorbidity ($p = 0.31$) distributions. Fisher's Exact Test ($p = 0.015$) revealed a significant disparity in the distribution of ADHD presentations across cluster profiles. ADHD-C clustered mainly in Conventional-steep and Steep profiles (77.78%), while ADHD-IN was more heterogeneous (Fig. 3A): 41.67% clustered in Zero/Shallow profiles and 50%, in Conventional-steep/Steep profiles. Notably, Zero/Shallow included only ADHD-IN children.

External validation

Clinical ratings. Cluster profiles did not exhibit significant differences in parents' or teachers' ratings of ADHD symptoms (ADHD-RS-5, SDQ), (Table 2) parents' ratings of internalizing and externalizing behaviors (CBCL/6-18) (Table S9), nor parents' ratings of behaviors associated with executive functions in daily routines (Table S10). Given the limited sample size of the Zero profile ($n = 4$), additional analyses were conducted excluding this group to address potential statistical biases. Upon excluding this group and repeating the analyses, no significant differences arose in any of the administered questionnaires.

Resting-state functional connectivity. Bayesian mean comparisons revealed credible differences in the rsFC among delay-averse profiles. Participants with Zero and Shallow DD exhibited decreased rsFC within the FPN and DMN, as well as between FPN-DMN nodes, compared to those with Conventional, Conventional-Steep, and Steep DD profiles.

Specifically, in the Shallow DD profile, credible differences involved DMN and FPN-DMN connections, peaking in the left DLPFC, right MTG, right IPL, left OFC, and frontopolar cortex (Fig. 4B). In the Zero DD group, reduced rsFC was primarily related to FPN-DMN interactions involving the left DLPFC and the right IPL. Furthermore, Zero DD participants showed reduced rsFC in these areas compared to Shallow DD participants (Fig. 4A). To ease the comprehension of results, the mean of the differences and 95% HDIs for credible comparisons are reported in the supplementary material (Tables S11 and S12).

To address the limited sample size of the Zero group, we conducted analyses excluding this subgroup (Table S13). These repeated analyses upheld the observed differences in reduced rsFC between participants with Shallow DD and those with Conventional and Conventional-Steep profiles. However, the differences with the Steep DD profile were no longer credible. Graphical visualizations of credible differences for Zero and Shallow DD participants are respectively presented in Fig. 4A, B.

DISCUSSION

Negative findings in delay aversion research [16, 17, 64, 65] are often attributed to the inability of categorical diagnostic models to capture the symptomatic variability inherent in the ADHD population [4]. Despite utilizing an ecological DDT featuring sufficiently extended delay intervals to elicit impulsive discounting behavior [15, 25], our study demonstrated that children diagnosed with ADHD-C, ADHD-IN, and control participants do not differ in delay discounting behavior. To address this issue, we employed clustering procedures to examine individual differences in delay discounting among children with ADHD and control participants. This approach identified five delay-averse motivational profiles that surpassed the arbitrary thresholds of DSM presentations. These findings offer several significant contributions to the understanding of delay aversion in ADHD.

Contrary to previous research consistently linking ADHD with heightened discounting [9, 10, 63], our results suggest that delay aversion in ADHD spans a continuum ranging from decreased to increased discounting. Specifically, we found that approximately 20% of ADHD children might exhibit a delay aversion deficit, aligning with previous estimates [13, 66], while another 20% displayed shallow discounting tendencies, and around 4% showed no discounting behavior at all. This continuum of delay discounting behaviors underscores the need to broaden our understanding of motivational deficits in ADHD, as decreased discounting has been previously associated with inflexible and obsessive traits characteristic of eating and obsessive-compulsive disorders [63, 67, 68], as well as mood symptoms such as anhedonia [69] and anxiety [70]. Importantly, these traits have also been identified in a small subset of individuals with ADHD [71]. These findings suggest that variability in delay aversion could be relevant to understanding not only ADHD heterogeneity but also its overlap with other psychopathological conditions characterized by impulsive-compulsive behaviors [63].

Remarkably, our data also reveal distinct patterns of delay aversion across ADHD presentations: most ADHD-C children (77.78%) clustered into steeper discounting profiles, while the ADHD-IN presentation exhibited a broader range of discounting behaviors—50% exhibited steeper discounting, whereas 41.67% displayed decreased or no discounting. This variability may explain inconsistencies in prior delay aversion research, particularly in studies including ADHD-IN children [16, 17]. These findings align with ADHD's heterogeneity across personality, temperament and cognitive domains. Around 85% of ADHD-C children cluster into personality ("high extraversion" or "low conscientiousness" [71]) and temperamental ("high surgency" or "high negative affect" [12, 72]) profiles linked to hyperactivity-impulsivity symptoms [73]. In contrast, half of ADHD-IN children exhibit these traits, while the other half (≈43–56%) align with introversion [71] or mild temperament [12, 72], correlating with inattention symptoms and supporting ADHD-IN heterogeneity [18–21], where subthreshold hyperactivity-impulsivity symptoms coexist with restrictive inattention, often associated to cognitive disengagement syndrome (CDS) [74]. Prior research on attentional performance might support these results [28], showing that while 83.87% of ADHD-C children cluster into two ADHD-like profiles exhibiting high rates of inattention and motor activity, ADHD-IN children are split—half (42.31%) align with these groups, while the other half (42.31%) resemble a CDS profile. Moreover, this recent study suggests that objective measures of hyperactivity may dissociate from impulsivity-related constructs such as response inhibition, as only one ADHD-like hyperactive profile exhibited clinically significant response disinhibition. Taken together, these findings underscore the importance of considering individual differences in reward processing when conceptualizing ADHD-related motivational deficits, reflecting underlying subgroups varying in personality traits, temperament, and cognitive processing. Further research distinguishing ADHD-

Table 2. Demographic and clinical characteristics of delay-averse profiles.

Characteristic	Steep DD ($n = 18$)	Zero DD ($n = 4$)	Shallow DD ($n = 15$)	Conventional-steep DD ($n = 35$)	Conventional DD ($n = 18$)
Demographics					
Age (years), *mean (SD)*	8.86 (2.69)**	12.04 (2.52)	12.36 (2.76)	10.84 (2.43)	12.01 (2.70)
IQ, *mean (SD)*	93.83 (17.97)	86.25 (9.91)	96.27 (17.65)	102.43 (15.84)	93.33 (12.57)
n (%) of girls	11 (61.11)	3 (75.00)	6 (40.00)	12 (34.29)	5 (27.78)
n (%) of European origins	16 (88.89)	4 (100)	15 (100)	29 (82.86)	18 (100)
n of C:I:H presentations*	4:4:0	0:2:0	0:8:0	10:8:0	4:2:1
n (%) on medication	4 (22.22)	2 (50.00)	6 (40.00)	9 (25.71)	3 (16.67)
hours in wash-out, *mean (SD)*	51.75 (45.70)	43.00 (19.80)	75.67 (57.30)	143.56 (174.79)	32.67 (4.16)
n (%) of Learning Disorder	4	-	3 (20.00)	3 (8.57)	4 (22.22)
n (%) of Mood Disorder	-	-	-	1 (2.86)	2 (11.11)
n (%) of ODD	-	-	1 (6.67)	4 (11.43)	2 (11.11)
n (%) of Language Disorder	-	-	-	1 (2.86)	-
ADHD-RS-5-Parents, mean (SD)					
Inattention	10.72 (9.42)	12.75 (10.50)	15.10 (8.45)	13.63 (9.17)	10.39 (8.45)
Hyperactivity-Impulsivity	9.17 (7.16)	5.50 (4.66)	9.40 (8.01)	10.26 (7.35)	7.17 (7.42)
Total scale	19.89 (15.43)	18.25 (14.59)	24.50 (13.55)	23.89 (15.19)	17.56 (14.66)
ADHD-RS-5-Teachers, mean (SD)[a]					
Inattention	11.93 (8.02)	9.00 (12.73)	10.67 (8.53)	12.01 (8.96)	8.25 (6.94)
Hyperactivity-Impulsivity	3.14 (4.52)	2.50 (3.54)	4.08 (4.38)	6.17 (6.75)	4.58 (4.52)
Total scale	15.07 (10.85)	11.50 (16.26)	14.75 (10.67)	18.18 (14.29)	12.83 (10.35)
SDQ Subscales–Parents, mean (SD)					
Emotional symptoms	3.67 (2.57)	3.75 (2.50)	3.43 (1.88)	3.57 (2.52)	3.83 (3.03)
Conduct problems	2.06 (1.98)	1.50 (1.29)	3.03 (2.04)	2.31 (2.22)	2.28 (2.52)
Inattention/ Hyperactivity	4.78 (2.78)	4.75 (2.50)	5.47 (2.23)	5.26 (3.18)	4.39 (3.27)
Peer problems	1.83 (1.54)	2.00 (1.63)	2.63 (1.80)	2.94 (2.71)	2.83 (2.36)
Prosocial behavior	8.17 (1.79)	8.50 (1.73)	8.10 (1.56)	8.06 (2.24)	8.28 (1.84)
Total difficulties	12.33 (7.15)	12.00 (5.83)	14.57 (5.27)	14.09 (8.38)	13.33 (9.54)
SDQ Subscales–Teachers, mean (SD)[a]					
Emotional symptoms	2.21 (2.29)	2.00 (2.83)	1.92 (2.11)	2.57 (1.68)	2.17 (1.75)
Conduct problems	1.43 (2.24)	0.50 (0.71)	0.75 (1.14)	1.91 (2.31)	1.42 (2.23)
Inattention/ Hyperactivity	4.36 (2.65)	3.00 (4.24)	3.92 (2.39)	4.27 (2.93)	4.00 (2.86)
Peer problems	1.00 (1.52)	1.00 (1.41)	0.83 (1.34)	2.18 (2.14)	1.08 (1.31)
Prosocial behavior	7.36 (2.34)	8.50 (2.12)	7.17 (1.85)	6.83 (2.90)	7.58 (2.50)
Total difficulties	9.00 (7.46)	6.50 (9.19)	7.42 (5.05)	10.93 (7.21)	8.67 (6.75)

**Bonferroni adjusted post-hoc comparisons revealed that Steep DD participants were significantly younger than Conventional and Shallow DD participants ($p < 0.01$).
*Significant differences were found in the distribution of ADHD-C and ADHD-IN children across cluster profiles ($p < 0.05$).
[a]Information missing for 9 ADHD children and 9 controls.

IN and CDS traits could provide critical insights into these variations.

From a theoretical perspective, the bifactorial model of ADHD, which accounts for both specific symptom dimensions and a general ADHD factor, may provide a useful framework for understanding this heterogeneity [75]. In this context, our findings suggest that delay aversion may be more closely related to the hyperactivity-impulsivity ("s") factor [1], given its connection to reactive control, immediate reward-seeking tendencies, negative emotionality, and low agreeableness [73, 76, 77]. Thus, while inattentiveness (e.g. reaction time variability [78], susceptibility to distractions [28, 79]), working memory impairments [80] and overactivity [28, 81] are widely recognized as core features of ADHD ("g" factor), difficulty in waiting for valued outcomes may

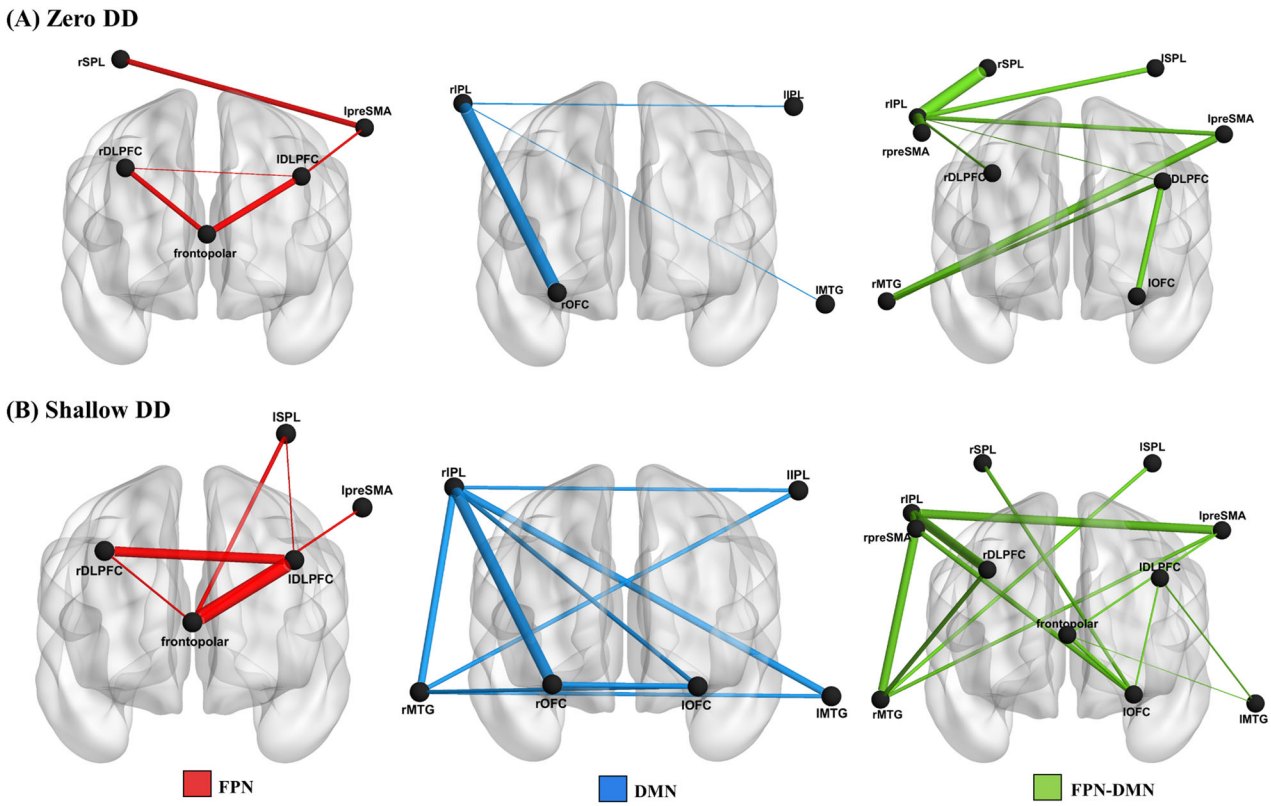

Fig. 4 Graphical visualization of credible differences in rsFC within and between FPN and DMN networks for delay-averse profiles. A Zero DD participants showed reduced rsFC than the rest of the cluster profiles (Table S11). **B** Shallow DD participants showed reduced rsFC than Conventional, Conventional-steep, and Steep DD participants, but increased rsFC than Zero DD participants (Table S12). lDLPFC left dorsolateral prefrontal cortex, rDLPFC right dorsolateral prefrontal cortex, lOFC left orbitofrontal cortex, rOFC right orbitofrontal cortex, lSPL left superior parietal lobe, rSPL right superior parietal lobe, lIPL left inferior parietal lobe, rIPL right inferior parietal lobe, lMTG left middle temporal gyrus, rMTG right middle temporal gyrus, lpreSMA left premotor and supplementary motor area, rpreSMA right premotor and supplementary motor area. frontopolar frontopolar cortex.

be present in ADHD children with significant hyperactivity-impulsivity traits. Further research into these delay-averse profiles, particularly considering their potential association with the hyperactive-impulsive "s" factor, could deepen our understanding of motivational deficits in ADHD and refine theoretical models that acknowledge their role in behavioral symptom manifestation [1, 66].

From a clinical perspective, our findings suggest that while delay-averse profiles do not differ in third-party clinical ratings of ADHD symptoms, externalizing and internalizing behaviors, or executive functions, these profiles could still hold important implications for diagnosis and treatment. Previous studies have similarly failed to establish significant associations between behavioral cluster profiles and clinical ratings of depressive symptoms [50], externalizing behaviors, and functional correlates [51]. These results reinforce the notion that rating scales, which rely on parental reports, provide complementary yet distinct information compared to performance-based tests [82–84]. Consequently, our findings emphasize the importance of integrating multimodal assessment methods in ADHD diagnosis, incorporating direct neuropsychological and behavioral evaluations [85]. Furthermore, although performance-based measures of reward processes are seldom standardized in neuropsychological assessments, our findings suggest they may be valuable in identifying children with difficulties in delaying valued outcomes or those exhibiting rare perfectionistic/rigid traits. This, in turn, can inform the design of tailored and effective interventions addressing these specific motivational challenges.

Finally, in line with the RDoC initiative, our study demonstrates that categorical ADHD-C and ADHD-IN presentations hamper the identification of neurobiological differences within ADHD. While ADHD children exhibited hypoconnectivity within and between the FPN and DMN compared to control participants, no credible differences were observed between the traditional ADHD-C and ADHD-IN presentations [6]. In contrast, we linked cluster profiles to neurofunctional correlates and found credible differences in fNIRS-based rsFC related to cluster profiles exhibiting no discounting or shallow discounting. Participants with Zero and Shallow DD were characterized by hypoconnectivity within and between FPN and DMN nodes compared to other cluster profiles. Moreover, Zero DD participants displayed even more reduced rsFC than Shallow DD participants, peaking in connections of the right IPL.

Initially, these results may appear inconsistent with research linking increased DLPFC activity to a preference for delayed rewards [86], and reduced rsFC to steep discounting behavior [87], and hyperactive-impulsive symptoms [88], especially considering the absence of such correlates in participants with Steep DD. However, considering the hypothesis that Zero and Shallow DD subgroups might present restrictive inattentive symptoms, as described above, or inflexible traits [63], our findings align with previous research associating reduced DMN connectivity in ADHD with mind-wandering [89], and suggesting that within- and between-network hypoconnectivity among FPN and DMN areas underlies OCD symptomatology [90, 91]. Additionally, these findings might be consistent with research supporting that inattentive profiles without core inhibitory control deficits might be biologically distinct [19, 92]. However, as previously mentioned, future studies should include a restrictive ADHD-IN subgroup to examine this interpretation, as well as significantly increase the sample size.

In summary, our study highlights the importance of adopting dimensional approaches to comprehensively understand atypical discounting behavior in ADHD. Through cluster analyses, we identified five distinct delay-discounting profiles, which broadened our understanding of ADHD variability across a spectrum of motivational traits, from impulsive to inflexible behaviors. Despite the lack of associations with clinical ratings, promising neurobiological substrates were identified by fNIRS. Nonetheless, it is important to interpret our conclusions cautiously due to the limited sample size, particularly in the Zero DD profile, which constrained robust statistical analyses. Future research with larger samples should delve into neuropsychological factors underlying delay discounting behavior, including temporal processing (timing), which could not be included in the current study and merit consideration [66]. The inclusion of medication-naïve participants and an extensive representation of the ADHD-HI subtype would enhance external validation, potentially leveraging functional connectivity measures [93]. Furthermore, longitudinal studies across different age cohorts might clarify developmental trajectories of temporal discounting in ADHD, offering insights with distinct implications for childhood and adult diagnostic practices, which may have different implications for diagnosing the disorder in both children and adults [86].

DATA AVAILABILITY

The data and source code that support the findings of this study are openly available at osf.io/qxe9h.

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AUTHOR CONTRIBUTIONS

PFM Conceptualization, Methodology, Investigation, Formal analysis, Data curation, Writing – Original Draft, Writing – Review & Editing, Visualization. DTS Investigation, Data curation. RRH Investigation, Data curation, Writing – Original Draft, JL Formal analysis, Writing – Review & Editing, RC Conceptualization, Methodology, Resources, Supervision, Writing – Review & Editing. PF Conceptualization, Methodology, Funding acquisition, Supervision, Writing – Review & Editing. All authors contributed to and approved the final manuscript.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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