BMC Psychiatry

SYSTEMATIC REVIEW Open Access

Brain–behavior relationships in task-based fMRI assessments of executive functions in children and adolescents with and without ADHD: a systematic review and ALE meta-analysis

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Abstract

Background Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder affecting about 5% of children and adolescents worldwide. ADHD symptoms often reflect impairments in executive functions (EFs). Functional magnetic resonance imaging (fMRI), particularly task-based fMRI (tb-fMRI), has been crucial in studying the neural mechanisms behind ADHD and its EF deficits. However, few studies have examined differences in brain activation and behavioral outcomes between children with and without ADHD using tb-fMRI, or the correlation between brain activation and behavior.

Methods This meta-analysis aimed to summarize evidence using activation likelihood estimation (ALE) analysis to identify differences in EFs and brain activation during tb-fMRI between children with and without ADHD. We retrieved published studies from PsychINFO, CINAHL Ultimate, Embase, MEDLINE, PubMed and Web of Science from inception through April 2024.

Results Following systematic review guidelines, 32 studies using tb-fMRI to compare children with and without ADHD during EF tasks were included in the ALE analysis. Children with ADHD showed significant differences in inhibitory control and working memory compared to controls. They exhibited reduced activation during EF tasks in areas such as the bilateral inferior frontal gyrus, supramarginal gyrus, inferior parietal gyrus, angular gyrus, caudate, occipital gyrus, and cerebellum. Correlation analyses revealed positive associations between brain activation and inhibitory control in the inferior frontal gyrus and anterior cingulate cortex in the ADHD group, suggesting a link between increased neural activity and better EF performance.

Conclusion The results of this study enhance our understanding of ADHDs pathophysiology and suggest potential pathways for developing interventions and therapies.

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Keywords ADHD, Task-based fMRI, Executive functions, ALE meta-analysis, Children and adolescents

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent childhood neurodevelopmental disorder characterized by inattention and hyperactivity/impulsivity. It has also emerged as a global public health concern [1]. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) defines three ADHD clinical presentations based on symptom count: predominantly hyperactive-impulsive subtype (ADHD-HI), predominantly inattentive subtype (ADHD-I), and a combined subtype (ADHD-C) [2]. The worldwide prevalence of ADHD is approximately 7.2% among all age groups [3] and 5% in school-age children [4]. ADHD has also been confirmed to be a lifelong disorder, with up to 70% of individuals with ADHD in childhood experiencing lasting symptoms into adulthood [4].

Compared with typically developing (TD) children, about 50% of children with ADHD show significantly impaired executive functions (EFs) [5]. EFs encompass top-down control processes, including automatic planning, organizing, and monitoring of complex goal-directed behaviours [6]. EFs comprise three core functions (inhibitory control [IC], working memory [WM], and cognitive flexibility) and three higher-level functions (reasoning, planning, and problem-solving) [6]. In children with ADHD, EF impairments often persist into adulthood and may worsen over time. These deficits are linked to substance abuse, antisocial behavior, and poor outcomes in education, career, and social life [7]. Vitamin D [8], Neurofeedback [8, 9], and various medications [10] have been used to treat children and adolescents with ADHD, but treatment effects are inconsistent. Some approaches have not demonstrated efficacy. Investigating how brain functional activity relates to EFs in ADHD may clarify the brain–behavior mechanisms. However, the interplay between abnormal brain functions and EF deficits remains unclear [11].

Functional magnetic resonance imaging (fMRI) is a non-invasive neuroimaging method that enables examining brain activity during rest and goal-oriented behaviors [12, 13]. Resting-state fMRI (rs-fMRI) [14] and task-based fMRI (tb-fMRI) [15] are among the most popular methods to explore the underlying mechanisms and pathophysiology associated with ADHD and EFs. Several rs-fMRI studies in a meta-review reported significant disruption in the connection between the default mode, cognitive control, and affective and salience networks in children and adolescents with ADHD [16]. Of the two, tb-fMRI, which evaluates brain functional activity during specific cognitive tasks, has demonstrated better predictive power for behavior and performance on EF-related tasks [17, 18].

Most tb-fMRI studies on ADHD have used cognitive paradigms related to EFs, such as IC, selective attention, and WM [19]. This focus is due to the prevailing belief that EFs are the primary etiological factors of ADHD. However, these neuroimaging studies have shown inconsistent findings, due to the heterogeneity of populations and the variability of task design. For example, the meta-analysis by McCarthy et al. [20] demonstrated that individuals with ADHD exhibit various levels of diminished activation in specific brain regions, contingent upon the type of cognitive tasks involved. During the N-back task (i.e., a WM test), reduced activation was observed in the bilateral superior frontal gyrus and the left medial frontal gyrus in both children and adults with ADHD. In the stop-signal task (i.e., inhibition test), decreased activation was noted in the bilateral inferior frontal gyrus (IFG), right medial frontal gyrus, right superior frontal gyrus, and right middle frontal gyrus in children with ADHD relative to the control children. During the go/no-go task, reduced activation was found in the left medial frontal gyrus and the right caudate in both children and adults with ADHD [20]. Another meta-analysis reported hypoactivation in the frontoparietal and ventral attention networks in ADHD children relative to control children, accompanied by hyperactivation in the default network, frontoparietal network, and dorsal attention network [21]. A recent meta-analysis [22] involving 1,914 children and adolescents with ADHD found little consensus among the studies. However, when categorized by stimulus type (emotion, reward, neutral), it did reveal a consistent pattern of dysfunction in tb-fMRI experiments with neutral stimuli. Specifically, there was convergence on dysfunction in the left pallidum/putamen and decreased activity (in male participants) in the left IFG. Several systematic reviews and meta-analyses have compared EF-related activation in ADHD and control groups using task-based paradigms [20–22]. For example, Samea et al. used activation likelihood estimation (ALE) meta-analysis with pooled structural and functional MRI data in adolescents. Their sub-analyses showed aberrant activation in the left pallidum/putamen and reduced activity in the left inferior frontal gyrus in males [22]. Another ALE meta-analysis including 55 studies (39 in children and 16 in adults) reported hypoactivation in the ADHD group within Yeo et al.'s seven-network framework [23], particularly in the frontoparietal (executive function) and ventral attention networks [21]. McCarthy et al. conducted a meta-analysis using Go/No-Go, Stop, and N-back tasks to explore the neurobiological basis of ADHD in both

adults and adolescents, finding that a higher proportion of combined-type ADHD was associated with reduced activation in the superior and inferior frontal gyri [20]. Unlike earlier work, our study focuses on brain–behavior relationships in a paediatric population. We apply the ALE method specifically to executive function-related tasks.

A quantitative meta-analysis using specific brain coordinates reported in a large number of studies is called for, especially as no meta-studies have explored the associations between brain activations and behavior in tb-fMRI studies of ADHD. Moreover, this method would help to filter out potential spurious activation [19]. Coordinate-based meta-analyses of neuroimaging studies can facilitate the integration of neural correlations and functional activities of brain regions identified in ADHD research. The activation likelihood estimation (ALE) method is considered one of the best meta-analytic approaches for identifying brain regions associated with behaviors of interest [24, 25]. By enabling the examination of differences in brain activations during various tasks in children and adolescents with ADHD, ALE provides a means to pinpoint specific regions implicated in EF deficits. A quantitative meta-analysis using specific brain coordinates reported across numerous studies is important, especially given the lack of meta-analyses using tb-fMRI in children and adolescents with ADHD to explore associations between brain activations and behavior. This approach helps to filter out potential spurious activations, enhancing the robustness of findings [19]. Therefore, our study aimed to conduct a quantitative, voxel-wise analysis based on ALE [24] of tb-fMRI studies involving children and adolescents with ADHD. This approach re-examines existing research to clarify how functional brain regions regulate EF and behaviour in this population.

Methodology

This study complied with the Preferred Reporting Items for Systematic Review and Meta-analyses Statement (PRISMA) [26]. The protocol is registered in PROSPERO (CRD42023467208). We conducted separate between-group ALE analyses on specific cognitive domains, ensuring a sufficient number of experiments (>17) for each analysis [27], such as overall ($N=32$) and IC ($N=22$). However, the experiments for WM ($N=8$) and reasoning ($N=2$) were too few in number to support a meaningful, comprehensive statistical analysis using the ALE method.

Search strategy

Electronic searches were conducted in six databases: APA PsychInfo (via Ovid), CINAHL Ultimate (via EBSCOhost), Embase (via Ovid), MEDLINE (via Ovid), PubMed, and Web of Science from inception through April 2024. The goal was to identify all of the relevant published articles regarding tb-fMRI and EFs in children and adolescents with and without ADHD. The search was limited to "English" and "human-related" articles. A snowball and citation search was performed to find additional relevant articles in the reference lists of the reviewed articles. The detailed keyword search strategy is presented in the Supplementary (Table S1).

Eligibility criteria

The inclusion criteria yielded studies that (1) included measurements of tb-fMRI to assess brain activity and EFs; (2) included observational research (i.e., cross-sectional, case-control, cohort, pre-test of the intervention research); (3) reported EFs measured by neurocognitive tasks during tb-fMRI (e.g., Inhibitory control assessed through Stroop task; working memory evaluated by n-back task; and cognitive flexibility measured by Wisconsin Card Sorting task); (4) were peer-reviewed articles with full-text available written in English; e) had participants aged 5–18 years and with diagnoses of ADHD confirmed by standardized diagnostic tools (e.g., DSM-III, -IV, and –5; ICD-10/and K-SADS-PL) or by parent and school reports as supplementary to standardised assessments and a healthy control (HC) group without ADHD; and f) provided complete research data where EFs and tb-fMRI paradigms could be computed.

The exclusion criteria excluded all articles that (1) collected data from rs-fMRI or subject reports of EFs; (2) only included participants with ADHD and no peers without ADHD; c) were written in a language other than English; d) reported intervention research without pre-test (e.g., clinical and field trials). Intervention studies that included baseline/pre-test data were retained, as our analysis only considered pre-intervention measures; e) were review studies, case/government reports, conference papers, book chapters, and policy documents; f) included preschool children (aged 0–4) or adults (aged 18 and older) as participants.

Study selection and data extraction

Two reviewers conducted independent, multi-step searching and screening and discussed discrepancies to reach a consensus. Between-reviewer consistency in abstract/full-text screening was measured with a Kappa value (none to slight agreement, 0×01–0×20; fair agreement, 0×21–0×40; moderate agreement, 0×41–0×60; substantial agreement 0×61–0×80; and almost perfect agreement 0×81–1×00) [28]. We developed a standardized data-extraction form to extract relevant study characteristics, including bibliographical details (author, year, and country/regions of the studies), participant characteristics (sample size, sex, age, medication status, IQ and diagnosis), measures of EFs, and major findings.

Quality assessment

Two independent reviewers evaluated the methodological quality of each included study. For every manual data entry, the second reviewer independently cross-checked the measures to ensure accuracy. The Newcastle–Ottawa Scale (NOS) was applied to both cross-sectional and cohort studies [29], with a maximum score of 7 for cross-sectional studies and 6 for the cohort studies. This scale has been adapted from the NOS for cohort and case-control studies to evaluate the methodological quality of cross-sectional studies included in this systematic review [29]. The original NOS was modified to suit the cross-sectional study design, a modification also employed by several other studies that recognized the need to adapt the NOS for appropriate assessment of such research. Studies were not excluded from the meta-analysis based on the quality score.

Data synthesis

A meta-analysis was conducted using Comprehensive Meta-analysis (v.3). EF performance (e.g., response time) was used to calculate the effect size to determine the differences in EFs between children and adolescents with ADHD and those without. Hedges' g was used to represent the effect size as this statistic addresses bias in small sample sizes [30]. We used a random-effects model to compute effect sizes with 95% confidence intervals (CI), accounting for potential heterogeneity between groups. The Hedges' g values were interpreted as small (≈0.2), moderate (≈0.5), or large (≥0.8) effect sizes [31]. Statistical heterogeneity was assessed (I^2),with a p-value calculated for Q statistics. Specifically, the I^2 values assessed whether heterogeneity was small (≤25%), medium (50%), or large (≥75%) [32].Furthermore, a sensitivity analysis was used to determine whether the elimination of any paper would influence the overall effect size [33]. A funnel plot and Egger's regression were applied to identify any publication bias. Statistical significance of $p < 0.05$ was set for all of the tests.

Activation likelihood estimation

The updated version of the ALE algorithm [34] was used to calculate the convergence of foci reported across various experiments. To test the significance of activation foci against a null hypothesis of random spatial association between experiments, the ALE meta-analysis followed a three-step process [22, 34, 35], as follows:

(1) The reported foci were treated as coordinate centers of 3D Gaussian probability distributions, reflecting spatial uncertainties due to sampling effects and methodological variations in data processing and analysis. Notably, this modelled uncertainty was adjusted based on the number of participants in the smaller study groups to account for the increased uncertainty inherent in smaller samples.

(2) The resulting "modelling activation" maps for each experiment were then integrated into an ALE map by calculating their agreement across all of the experiments.

(3) The peak foci of the distribution values that exceeded the threshold were reported and anatomically labelled, with a focus on clusters larger than 200 mm^3 and corrected for false discovery rate (FDR) at $p < 0.01$.

ALE-fMRI analysis

GingerALE 3.0.2 (https://www.brainmap.org/ale/) software was used for the systematic review of the neuroimaging data. We reorganized the extracted data, which included bibliographical information such as authors, title, publication year, and journal; age, gender, and number of participants; and the peak coordinates of group comparisons in either the Montreal Neurological Institute (MNI) [36] or Talairach [37] space. The Talairach coordinates were subsequently converted to the MNI space using the "convert foci" tool in GingerALE. We combined experiments that indicated changes in the same direction (increases or decreases) within patient groups. Each study contributed up to two contrasts: one for ADHD > HC contrast and another for HC > ADHD contrast. The coordinates for each study were then manually entered into GingerALE for these contrasts.

Results

Study identification

A total of 551 articles were identified through the initial six databases mentioned above. Figure 1 illustrates the number of articles that were screened and that met the inclusion criteria. After 315 duplicates were removed, 236 more were identified by title/abstract. Forty-five abstracts met the inclusion criteria, with an inter-rater reliability of $k = 0.81$ (almost perfect agreement) between the two reviewers. Additionally, 22 manually searched records that initially met the inclusion criteria were included for further full-text screening. Finally, 32 articles passed the inclusion criteria with an inter-rater reliability of $k = 0.91$ (almost perfect agreement). All 32 studies included were identified as suitable for meta-analysis, as they reported sufficient statistical data [38–69].

Descriptive characteristics of included studies

Table 1 summarizes the characteristics of the included studies. All of the studies were published between 2004 and 2022. In terms of geographic location, 15 were conducted in North America, 10 in Europe, four in Asia, and three in Australia. Eighteen of the studies utilized MNI space, while 14 employed Talairach space. The detailed

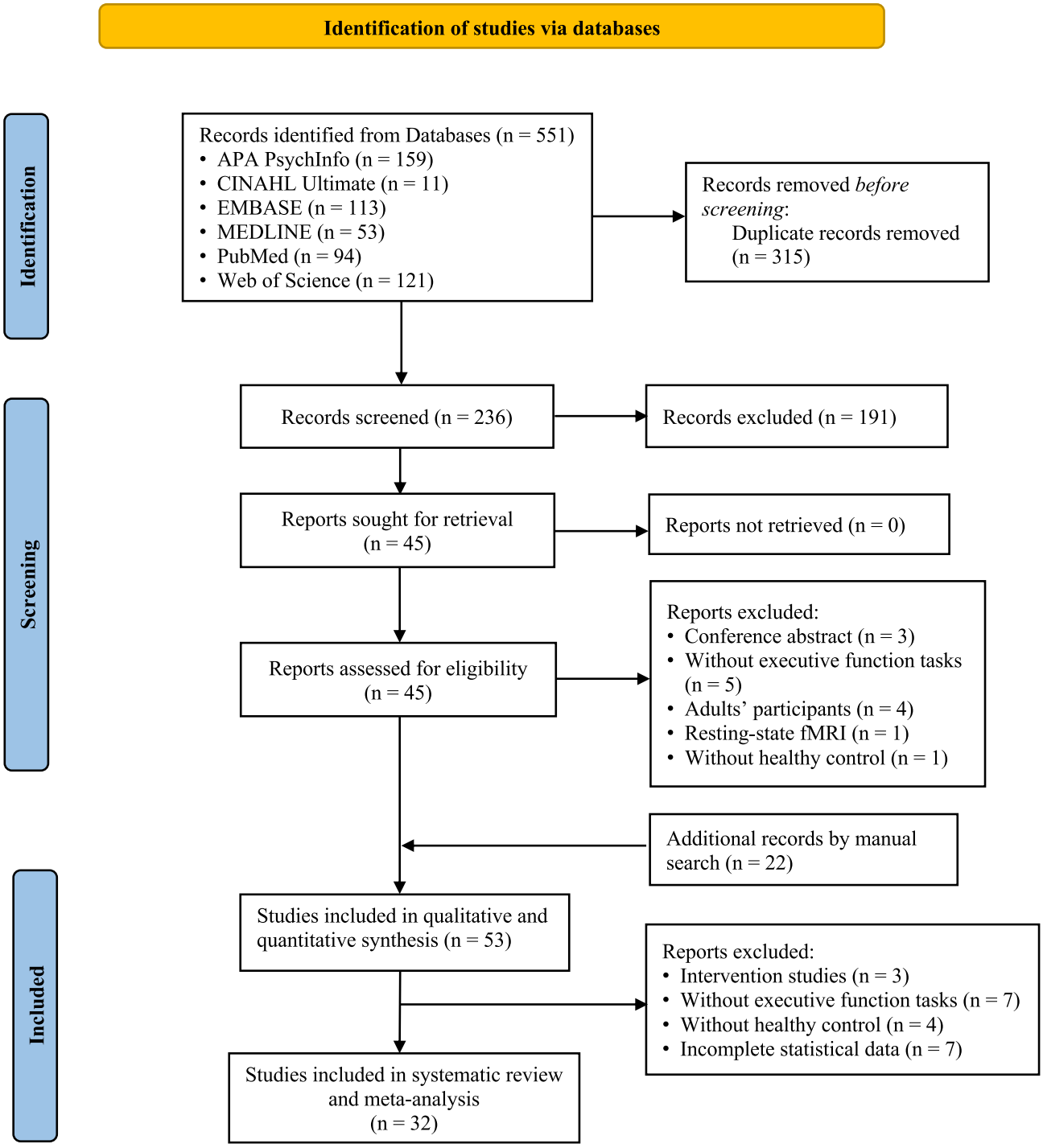

Fig. 1 Prisma flow diagram of the selection of studies

characteristics of brain imaging among the included studies are presented in Table 2. The 32 studies had a total of 586 participants with ADHD (82.0% boys) and 451 without ADHD (74.6% boys), aged 5 to 18 years. Of the reviewed studies, 31 (96.9%) reported the medication use status of the participants. Of these, 18 studies [38–41, 44–46, 49, 50, 53, 56, 57, 59, 62, 63, 65–67] involved children and adolescents with ADHD who were taking medication for ADHD. One of the studies [54] recruited both medicated and unmedicated participants. They were asked to stop taking medicine at least 18 hours before fMRI acquisition and psychological assessments. Furthermore, 30 of the studies (93.8%) measured IQ with a formal IQ test (e.g., Wechsler Intelligence Scale

Table 1 Summary of participants characteristics and executive functions of included studies (ADHD vs TD)

Study Name (Year, Country/ Region)	Sample size (ADHD vs TD)	Age (M±SD)	Sex (Male%)	Medication use[#] (ADHD)	IQ[#] (ADHD vs TD)	Diagnostic Methods (Classification of ADHD subtype)	Outcome measures (number of measures)	Major findings
Bechtel et al (2012, Switzerland)	15 ADHD 15 TD	11.03±1.35 11.49±1.82	M-100% M-100%	Yes	47.3±35.5 44.2±26.6 (Raven's progressive Matrices in percent ranks)	DSM-IV (ADHD-I (6); ADHD-C (9))	WM: N-Back Task	WM: **00**
Bhaijiwala et al (2014, Canada)	12 ADHD 12 TD	13.8±2.3 15.4±1.7	N/A	Yes (24 h)	No difference (WISC-IV)	DSM-IV& PICS-IV (ADHD-I (3); ADHD-C (9))	IC: Stop-Signal Task	IC: + (*t*=-2.217) faster RT in TD
Braet et al (2011, Ireland)	20 ADHD 38 TD	14.1±2.1 13.26±1.98	M-85% M-81.6%	Yes (24 h)	95.9±15.2 111.25±13.3	DSM-IV & PACS & CAPA	IC: SART	IC: + (*F*=18.48) more omission errors in ADHD
Chevrier & Schachar (2020, Canada)	14 ADHD 14 TD	13.7±2.1 15.4±1.6	M-50% M-64.3%	Yes (24 h)	NA	DSM-V & PICS-IV	IC: Stop-signal Task	IC: ($p = 0.039$) slower RT in ADHD
Fan et al (2014, Taiwan)	25 ADHD 23 TD	10.9±2.2 11.2±2.9	M-92% M-91.3%	None	107.2±10.8 109.1±7.5	DSM-IV & K-SADS-E	IC: Counting Stroop Task	IC: **00**
Fan et al (2018, Taiwan)	27 ADHD 27 TD	12.1±2.0 11.8±1.7	M-88.9% M-77.8%	None	105.2±13.7 110.4±10.2	DSM-IV & K-SADS-E	IC: Counting Stroop Task	IC: +(*t*=2.44) slower RT in ADHD
Fassbender et al (2011, United States of America)	13 ADHD 13 TD	10.7±4.13 10.6±1.8	M-84.6% M-61.5%	Yes	117.23±10.69 117.62±12.15	DSM-IV-TR, DICA,CPRS-R-L & CTRS-R-L	WM: Visual Serial Addition Task	WM: (F=7.8) more omission errors in ADHD
Hart et al (2014, United Kingdom)	30 ADHD 30 TD	13.9±2.0 14.1±2.5	M-100% M-100%	Yes (48 h)	92±11 109±12	SDQ, CPRS-R, SCQ & DSM-IV-TR	IC: Stop Task	IC: NS
Iannaccone et al (2015, Switzerland)	18 ADHD 18 TD	14.50±1.52 14.82±1.24	M-61.1% M-50.0%	Yes (24 h)	108.46±17.75 114.45±10.32	DSM-IV-TR,ICD-10,CPRS & KSADS	IC: Modified Speeded Flanker Task	IC: +(*t*=2.98) less conflict accuracy in ADHD
Karakaş et al (2015, Turkey)	53 ADHD 24 TD	9.73±3.53 9.97±1.68	M-100% M-100%	None	90-130	DSM-IV & KSADS (ADHD-AD (16); ADHD-C (37))	IC: Stroop Test	IC: +(*F*=4.83) higher correct responses in TD
Konrad et al (2005, Germany)	16 ADHD 16 TD	10.2±1.9 10.1±1.3	M-100% M-100%	None	103±12 105±10	DSM-IV & KSADS (ADHD-I (6); ADHD-HI (1); ADHD-C (9))	IC: Attention Network Test	IC: +(*t*=-2.9) slower conflicts RT in ADHD
Lemiere et al (2012, Belgium)	10 ADHD 10 TD	14.72±1.49 14.40±1.33	M-80% M-70%	Yes (48 h)	96.30±6.93 116.50±10.89	DSM-IV & KSADS	IC: Escape Delay Incentive Task	IC: **00**
Ma et al (2016, Netherlands)	25 ADHD 33 TD	15.36±1.08 15.30±1.05	M-76% M-66.7%	Yes (24 h)	98.28±16.26 108.94±12.81	DSM-IV-TR, DISC-IV-Parent version	IC: Rewarded Stroop Color-word Task	IC: +(*F*=7.92) slower and (*F*=5.11) less accurate in ADHD
Malisza et al (2012, Canada)	20 ADHD 21 TD	11.99±1.32 12.60±1.29	M-90% M-76.2%	None	96.55±16.87 107.81±13.08	Pediatric physician	WM: A Spatial Working Memory N-back Task	WM: + ($F = 3.98$) faster 0-Back RT in TD
Massat et al (2012, France)	19 ADHD 14 TD	10.75±1.31 10.05±1.28	M-47.4% M-57.1%	None	above 85 (WASI)	DSM-IV & KSADS	WM: A Verbal Working Memory N-back Task	WM: NS
Openneer et al (2021, Netherlands)	23 ADHD 52 TD	10.14±1.54 10.53±0.10	M-78.3% M-71.2%	Yes (non-stimulant medication)	103.62±12.35 108.72±11.02	DSM-IV-TR, K-SADS, CBCL & TRF	IC: Stop-signal Task	IC: + (*F*=4.75) faster RT in TD

Table 1 (continued)

Study Name (Year, Country/ Region)	Sample size (ADHD vs TD)	Age (M±SD)	Sex (Male%)	Medication use[#] (ADHD)	IQ[#] (ADHD vs TD)	Diagnostic Methods (Classification of ADHD subtype)	Outcome measures (number of measures)	Major findings
Pliszka et al (2006, United States of America)	9 ADHD (Med) 8 ADHD (Unmed) 15 TD	13.4±1.9 12.9±1.4 13.2±1.9	M-88.9% M-62.5% M-60.0%	Mix	108.8±11.2 107.9±16.1 115.4±11.7	DISC-IV-P	IC: Stop Signal Task	IC: +(*F*=2.6) reduced slop in TD
Poissant et al (2016, Canada)	23 ADHD 21 TD	7-15 7-15	M-69.6% M-42.9%	N/A	103-107(K-Bit) 103-114(K-Bit)	DSM-IV-TR & RFQ	IC: Auditory Consonant Trigrams Test	IC: **00**(*t*=1.15)
Posner et al (2011, United States of America)	15 ADHD 15 TD	13.5±1.2 13.4±1.2	M-86.7% M-86.7%	Yes	111.4±16 114.1±10 (WASI)	DISC-Parent versions	IC: The Cognitive Stroop Task and The Emotional Stroop Task	IC: **00**
Schulz et al (2004, United States of America)	10 ADHD 9 TD	17.9±1.6 17.5±1.2	M-100% M-100%	Yes (6 months)	88.4±15.7 91.9±16.0 (WISC-III/WAIS-III)	DSM-IV & DISC-IV (ADHD-C (10))	IC: Go/No-Go task	IC: +(*t*=-2.19) less commission errors in TD
Shang et al (2022, Taiwan)	28 ADHD 28 TD	10.29±2.17 11.35±2.61	M-78.6% M-78.6%	None	99.29±10.56 104.39±11.78	DSM-5 & K-SADS-E	IC: Counting Stroop task	IC: **00**
Sheridan et al (2007, United States of America)	10 ADHD 10 TD	15.2±2 14.9±1.3	M-0% M-0%	Yes (24 h)	Verbal IQ: 104±17.2 120.1±12.6 Performance IQ: 107.9±13.9 109.4±8.9	DSM-IV (ADHD-C (6); ADHD-I (4))	WM: Delayed Match-to-Sample Task	WM: NS
Silk et al (2005, Australia)	7 ADHD 7 TD	14.38±1.85 14.56±1.77	M-100% M-100%	None	Performance IQ: 109.29±12.30 113.00±8.34	A-DISC	WM: Mental Rotation Task	WM: (p<0.05) less accuracy in ADHD
Silk et al (2008, Australia)	12 ADHD 12 TD	11.15±1.53 11.09±1.50	M-100% M-100%	None	103.60±11.93 111.9±10.9	A-DISC & CGI (ADHD-C (12))	R: Adapted version of the Raven's Standard Progressive Matrices trial	R: NS
Spinelli et al (2011, United States of America)	13 ADHD 17 TD	10.6±1.4 10.5±1.2	M-69.2% M-47.1%	Yes (24 h)	109.2±5.2 108.8±15.4	DSM-IV-TR & CPRS-R/CTRS-R	IC: Go/NoGo Task	IC: NS
Steven et al (2007, United States of America)	23 ADHD 23 ADHD	14.7±1.85 15.1±1.94	M-100% M-100%	Yes (24 h)	101.8±9.70 107.1±11.8 (WRAT reading subtest)	KSADS	WM: Three-Stimulus Auditory Oddball Task	WM: (*t*=1.84) faster RT in TD
Tamm & Juranek (2012, United States of America)	12 ADHD 10 TD	9.0±1.3 10.0±1.4	M-54% M-80%	None	11.7±2.3 11.0±2.7 (WISC-IV-Matrix reasoning subtest)	DSM-4 & KSADS	R: Fluid Reasoning Task	R: +(*t*=-1.67) less control accuracy in ADHD
Tamm et al (2004, United States of America)	10 ADHD 12 TD	16.00±1.41 15.58±0.79	M-100% M-100%	Yes (18 h)	109.20±14.50 111.58±11.73 (WASI)	Parent Completed Diagnostic Interview for Children and Adolescents and Conners ADHD/DSM-4 Scale	IC: Modified Go/ NoGo task	IC: (t=2.74) more commission errors in ADHD
Tamm et al (2006, United States of America)	14 ADHD 12 TD	14-18 14-18	M-100% M-100%	Yes (18 h)	108.38±13.28 111.58±11.73 (WASI)	Conner ADHD/ DSM-IV scale-Parent Version & DICA-IV	IC: Oddball Task	IC: +(*t*=1.83) less commission errors in TD
Vaidya et al (2005, United States of America)	10 ADHD 10 TD	8.8±0.9 9.2±1.3	M-70% M-70%	Yes (36 h)	104.3±16.0 128.4±16.7 (WAIS-III)	DSM-IV & SNAP	IC: Modified Eriksen Flanker Task	IC: +(*t*=1.83) higher accuracy in TD

Table 1 (continued)

Study Name (Year, Country/ Region)	Sample size (ADHD vs TD)	Age (M±SD)	Sex (Male%)	Medication use[#] (ADHD)	IQ[#] (ADHD vs TD)	Diagnostic Methods (Classification of ADHD subtype)	Outcome measures (number of measures)	Major findings
Vance et al (2007, Australia)	12 ADHD 12 TD	11.1±1.5 10.2±1.3	M-100% M-100%	None	103.6±11.9 111.9±10.9	A-DISC	WM: Mental Rotation Task	WM: NS
Wang et al (2013, China)	28 ADHD 31 TD	9.60±1.61 10.23±1.83	M-89.3% M-51.6%	None	105.4±11.4 108.2±9.3 (WISCC-R)	DSM-IV & KSADS (ADHD-C (28))	IC: Numeral version of Cued Continuous Performance Task	IC: **00**(t=1.15)

Note: [#]: All children were free of stimulant medication (methylphenidate, dextroamphetamine, and caffeine) for at least 36 hours prior to the fMRI acquisition and for the psychological assessments. [#]: Full-Scale IQ (FIQ) score is measured by Wechsler Intelligence Scale for Children. A-DISC: Anxiety Disorders Interview Schedule for Children; ADHD-AD: Attention Deficit Hyperactivity Disorder Subtype Characterized Predominantly by Attention Deficit; ADHD-C: Attention Deficit Hyperactivity Disorder Combined Subtype; ADHD-I: Attention Deficit Hyperactivity Disorder Inattentive Subtype; CAPA Child and Adolescent Psychiatric Assessment; CBCL: Child Behaviour Checklist; CDIS: Clinical Diagnostic Interviewing Scales; CPRS-R:L: Parent Conners' Rating Scale-Revised Long Version; CTRS-R-L: the Conners' Teacher Rating Scale-Long Version; DICA-IV: Diagnostic Interview for Children and Adolescents–Version IV; DISC-IV: Diagnostic Interview Schedule for Children–Version IV; DISC-IV-P: Diagnostic Interview Schedule for Children–Version IV-Parent Version; DSM-IV and-V: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition and Fifth Edition; IC:Inhibitory Control; ICD-10: International Classification of Diseases; K-Bit: Kaufman Brief Intelligence Test; K-SADS: parent-administered Kiddie Schedule for Affective Disorders and Schizophrenia; KSADS: Kiddie Schedule for Affective Disorders and Schizophrenia for School-age Children; K-SADS-E: the Chinese Kiddie epidemiological version of the Schedule for Affective Disorders and Schizophrenia Interview; P-ChIPS: Children's Interview for Psychiatric Syndromes; PICS-IV: the Parent Interview for Child Symptoms; R: Reasoning; RFQ: Risk Factor Questionnaire; SART: Sustained attention to response task; SCQ: the Social Communication Questionnaire; SDQ: the Strengths and Difficulties Questionnaire for parents; TD: Typically Developing; WASI: Wechsler Abbreviated Intelligence Scale; WISCC-R: Wechsler Intelligence Scale for Chinese Children-Revised; WM: Working Memory; WRAT:Wide-Range Achievement Test

for Children, WISC). [38–40, 42–51, 53–69]. They found non-significant differences in IQ between children and adolescents with ADHD and those without, indicating normal intellectual development (i.e., IQ > 90) in all of the participants. Moreover, 31 of the studies (96.9%) confirmed ADHD diagnosis using standardized methods such as DSM-IV and –5, DISC, and K-SADS-PL. However, only seven of the studies (21.9%) provided a clear classification of the ADHD subtype [38, 39, 47, 48, 57, 59, 69]. Overall, the core EFs were assessed by 30 of the studies [38–60, 62, 63, 65–69]. IC was examined by 22 [39–43, 45–50, 53, 54, 56–58, 62, 63, 65–67, 69], and WM was measured by eight. [38, 44, 51, 52, 55, 59, 60, 68]. The studies that focused on higher-level EFs were limited, and reasoning was reported by only two [61, 64]. Moreover, for core EF outcomes, the Stroop and stop-signal tasks were frequently used to evaluate IC; the N-back task was the most commonly used to measure WM. Lastly, regarding higher-level EFs, reasoning was evaluated by Raven's Standard Progressive Matrices. In the quality assessment, 6 of the 32 articles scored 7, 21 scored 6, and 5 articles scored 5 based on the Newcastle–Ottawa Scales (Table S2). The detailed information on the included studies is also presented in Supplementary (Table S3).

Meta-analysis of behavioral performance of overall EFs

A significant and moderate-to-large difference in overall EFs was observed between children and adolescents with ADHD and those without ADHD (k = 32, g = –0.603, 95% CI [–0.779, –0.428]), with medium heterogeneity (Q = 64.493, I^2 = 51.9%, p < 0.001; Fig. 2). Furthermore, our sensitivity analysis revealed no significant changes in the effect size following the elimination of any individual paper (95% CI [–0.537, –0.639]). Additional sensitivity analysis was also performed by excluding studies with a quality score of 5. The results remained consistent (k = 27, g = –0.584, 95% CI [–0.779, –0.389]), indicating that the overall findings were robust to the exclusion of lower-quality studies. In the linear regression (Egger's) test, p = 0.041, indicating publication bias between the included papers. A trim-and-fill method (random effects model) was used to correct publication bias (g publication-bias-adjusted = –0.754, 95% CI [–0.943, –0.564], with six studies needed to balance the plot (Figure S1).

Meta-analysis of behavioral performance of core and higher-level EFs

A significant and moderate-to-large difference in core EFs was observed between individuals with ADHD and those without ADHD (k = 30, g = –0.626, 95% CI [–0.810, –0.442]), with medium heterogeneity (Q = 62.104, I^2 = 53.3%, p < 0.001; Fig. 3). Specifically, between-group differences in IC were statistically significant (k = 22, g = –0.608, 95% CI [–0.820, –0.396]) and showed medium heterogeneity (Q = 48.449, I^2 = 56.6%, p = 0.001). WM showed a moderate-to-large effect of poorer performance in children and adolescents with ADHD (k = 8, g = –0.682, 95% CI [–1.056, –0.309]) with medium heterogeneity (Q = 13.301, I^2 = 47.4%, p = 0.065). Only two of the studies focused on higher-level EFs, and group differences in terms of reasoning were not significant (k = 2, g = –0.255, 95% CI [–0.882, 0.371]) and showed low heterogeneity (Q = 1.210, I^2 = 17.3%, p = 0.271).

ALE analysis results across the 32 studies

Regions of greater activation in the HC individuals than in those with ADHD (HC > ADHD) were observed under EF conditions. These regions were the bilateral

Table 2 Characteristics of brain imaging among the included studies

Study Name (Year, Country/ Region)	Withdrawal from ADHD medication before scan	Task	Contrast	Foci			Correction of Multiple Comparisons (Tool used for analyses)
				Number of Foci	MNI	Talairach	
Inhibitory Control							
Bhaijiwala et al (2014, Canada)	24 hours	Stop Signal Task	TD>ADHD: Prospective and reactive inhibition	11		*	Voxel size = 540 mm^3, with a minimum Z = 2.32, overall α of 0.046; Corrected (AFNI)
			ADHD>TD: Prospective and reactive inhibition	3			
Braet et al (2011, Ireland)	At least 24 hours	The Sustained Attention to Response Task	TD>ADHD: Successful inhibition	2	*		Voxel size = 540 mm^3; Cluster-size threshold was determined using Monte-Carlo simulations (1000 iterations), p = 0.05; Corrected (AFNI)
Chevrier & Schachar (2020, Canada)	6 (6/14) for 24 hours	Stop Signal Task	ADHD>TD Error detection and Post-error slowing	3		*	Voxel size = 920 mm^3, with a minimum Z = 1.96, for an overall α < 0.05; Corrected (AFNI)
Fan et al (2014, Taiwan)	6 (6/25) for at least 1 week	Counting Stroop Task	ADHD>TD INCON vs CON	3	*		Voxel level with a cluster size threshold of 10 voxels; FWE Corrected, $p < 0.05$ (SPM5)
Fan et al (2018, Taiwan)	NA	Counting Stroop Task	ADHD>TD INCON vs CON	3	*		At $p < 0.05$ FWE corrected at the voxel level (SPM12)
Hart et al (2014, United Kingdom)	1 (1/30) subject for over 1 year, 9 (9/30) for 48 hours, 20 (20/30) NA	Stop Task	TD>ADHD Motor response inhibition	8		*	A voxel-level threshold of p < 0.05 and p < 0.01 for clusters (XBAM)
			ADHD>TD Motor response inhibition	1			
Iannaccone et al (2015, Switzerland)	At least 48 hours	Modified Speeded Flanker Task	TD>ADHD Inhibition	1	*		Cluster extent threshold of p < 0.05; Cluster size = 340.86 mm^3; voxel-threshold p<0.005; Corrected (SPM8)
			ADHD>TD Inhibition	10			
Karakaş et al (2015, Turkey)	NA	Modified Stroop Test	TD>ADHD INCON vs CON	2		*	Activation areas were defined at a threshold value of ≥50 jointly activated voxels in a specific region-of-interest; FDR q= 0.05; Corrected (SPM8)
Konrad et al (2005, Germany)	NA	Modified Attention Network Test	TD>ADHD Conflict condition	2	*		Cluster threshold of 10 voxels, $p < 0.001$; Uncorrected (SPM2)
			ADHD>TD Conflict condition	1			
Lemiere et al (2012, Belgium)	At least 48 hours	The Escape Delay Incentive Task	ADHD>TD No escape delay >Escape delay	8	*		FWE corrected p<0.05 (SPM8)
Ma et al (2016, Netherlands)	15 (15/25) for 24 hours	Rewarded Stroop Color-word Task	ADHD>TD Reward	4	*		Cluster-level threshold of $p < 0.05$; FWE corrected (SPM8 and MATLAB 2013)
Openneer et al (2021, Netherlands)	48 hours for stimulant medication	Stop Signal Task	ADHD>TD Stop-failed-Stop success	4	*		Cluster-level threshold of $p < 0.05$; FWE corrected (SPM12)

Table 2 (continued)

Study Name (Year, Country/ Region)	Withdrawal from ADHD medication before scan	Task	Contrast	Foci			Correction of Multiple Comparisons (Tool used for analyses)
				Number of Foci	MNI	Talairach	
Pliszka et al (2006, United States of America)	6 months	Stop Signal Task	TD>ADHD Unsuccessful inhibition	5		*	Cluster-size threshold of Z ≥ 2.3, $p < 0.01$ (9 voxels); Corrected (FSL)
			ADHD>TD Unsuccessful inhibition	2			
Poissant et al (2016, Canada)	At least 24 hours	Auditory Consonant Trigrams Test	TD>ADHD Incoherent and Coherent conditions	4	*		Cluster-level threshold of $p = 0.001$; Cluster-size threshold of 10 voxels; Uncorrected (SPM8)
Posner et al (2011, United States of America)	7 (7/15) NA, 8 (8/15) for at least 48 hours	The Cognitive Stroop Task and The Emotional Stroop Task	ADHD>TD Positively valenced distraction, Positive words >Neutral words	1		*	Voxel-wise α=0.01, cluster-wise $p < 0.0001$; Corrected (SPM5)
Schulz et al (2004, United States of America)	6 months	The Go/No-Go Task	TD>ADHD No-go vs Go trials	6		*	Cluster-size threshold of kappa=120 voxels; Uncorrected $p < 0.01$ (SPM99)
			ADHD>TD No-go vs Go trials	10			
Shang et al (2022, Taiwan)	Require to take the medication in the morning 2 to 4 hours before the second fMRI	The Counting Stroop Task	TD>ADHD INCON vs CON	7	*		Cluster size ≥ 10 voxels, uncorrected $p < 0.05$ (SPM8)
			ADHD>TD INCON vs CON	4			
Spinelli et al (2011, United States of America)	2 (2/13) for on the day prior and day of testing	Go/No-Go task	ADHC>TD Post-error vs Post-correct inhibition trials	9	*		Uncorrected $p = 0.005$; a cluster size threshold equivalent to a corrected threshold $p<0.05$ (SPM5)
Tamm et al (2004, United States of America)	18 hours	Modified Go/No-Go task	TD>ADHD A vs B	1		*	Joint expected probability distribution of Z>1.67, $p<0.05$ and extent $p<0.05$ threshold, corrected (SPM99)
			ADHD>TD A vs B	1			
Tamm et al (2006, United States of America)	5 (5/14) for 18 hours, 9 (9/14) for NA	Oddball Task	TD>ADHD Oddball vs Standard stimuli	4		*	Joint expected probability distribution of Z>1.67, $p<0.05$ and extent $p<0.05$ threshold, corrected (SPM99)
Vaidya et al (2005, United States of America)	1 (1/10) for 4 weeks, 2 (2/10) for 36 hours, 7 (7/10) NA	Modified Eriksen Flanker Task	TD>ADHD INCON vs Neutral trials	2		*	$p < 0.001$; extent threshold of 10 voxels, Uncorrected (SPM99)
Wang et al (2013, China)	NA	Numeral version of Cued Continuous Performance Task	TD>ADHD No Go Condition	6		*	Cluster-size threshold of 54 voxels, $p < 0.05$; Corrected (SPM8)
			ADHD>TD No Go Condition	7			
Reasoning							

Table 2 (continued)

Study Name (Year, Country/ Region)	Withdrawal from ADHD medication before scan	Task	Contrast	Foci			Correction of Multiple Comparisons (Tool used for analyses)
				Number of Foci	MNI	Talairach	
Silk et al (2008, Australia)	NA	Adapted version of the Raven's Standard Progressive Matrices Trail	TD>ADHD Target stimulus vs Baseline	34	*		Cluster of voxels threshold of z = 2.33, $p < 0.05$, Corrected (SPM2)
Tamm & Juranek (2012, United States of America)	NA	Fluid Reasoning Task	TD>ADHD: Fluid reasoning vs Control condition	2	*		Z>2.3, a corrected cluster significance of $p < 0.05$, Corrected (FEAT from FSL)
Working Memory							
Bechtel et al (2012, Switzerland)	Approximately 1 and 1.5 hours	N-back Task	TD>ADHD 2- and 3-back task	17	*		A threshold of p < 0.0001 uncorrected at the voxel level and a cluster size of 20 (SPM5)
Fassbender et al (2011, United States of America)	9 (9/13) for 48 hours	Visual Serial Addition Task	TD>ADHD WM-related contrast	1		*	Significant required a voxel-wise threshold of p ≤0.005, combined with a minimum cluster-size of 282 μl; $p < 0.05$; Corrected (AFNI)
			ADHD>TD WM-related contrast	4			
Malisza et al (2012, Canada)	At least 36 hours for stimulant medication	A Spatial Working Memory N-back Task	ADHD>TD 1-back	2		*	FWE=0.001, Cluster size= 10 corrected (SPM5)
			TD >ADHD 1-back	11			
Massat et al (2012, France)	NA	A Verbal Working Memory N-back Task	TD>ADHD WM-related 2-back condition>Vigilant/ Control condition	14	*		Cluster-size threshold of 20 voxels, $p < 0.05$; Corrected (SPM8)
Sheridan et al (2007, United States of America)	2 (2/10) for 24 hours, 5 (5/10) for at least 1 year, 3 (3/10) NA	Delayed Match-to-Sample Task	TD>ADHD Encoding 6 letters >2 letters	2	*		Gaussian random correction cluster-level threshold of $p = 0.05$, voxel level threshold of t=2.82 (SPM2)
Silk et al (2005, Australia)	NA	Mental Rotation Task Paradigm	TD>ADHD Stimuli >Baseline	10		*	Cluster-level threshold of $p < 0.05$; voxel level $p < 0.01$; Uncorrected (SPM2)
			ADHD>TD Stimuli >Baseline	9			
Steven et al (2007, United States of America)	At least 24 hours	Three-Stimulus Auditory Oddball Task	TD>ADHD Novel Stimuli vs Standard Baseline	4	*		Surpasses the critical threshold at the Bonferroni-corrected $p = 0.05$, and meets or surpasses the critical threshold at the uncorrected $p = 0.001$
Vance et al (2007, Australia)	NA	Mental Rotation Task	TD>ADHD Rotation vs Baseline stimuli	4	*		Cluster-level threshold of $p < 0.05$; Corrected (FEAT from FSL)

Note. ADHD: Attention Deficit/Hyperactivity Disorder; AFNI: Analysis of Functional Neuroimages; CON: Congruent condition

FEAT: FMRIB's Expert Analysis Tool; FDR: False Discovery Rate; FSL: FMRIB Software Library; FWE: Family-Wise Error; INCON: Incongruent condition; MNI: Montreal Neurological Institute coordinate system; NA: Not available; SPM: Statistical Parametric Mapping; TD: Typically developing control

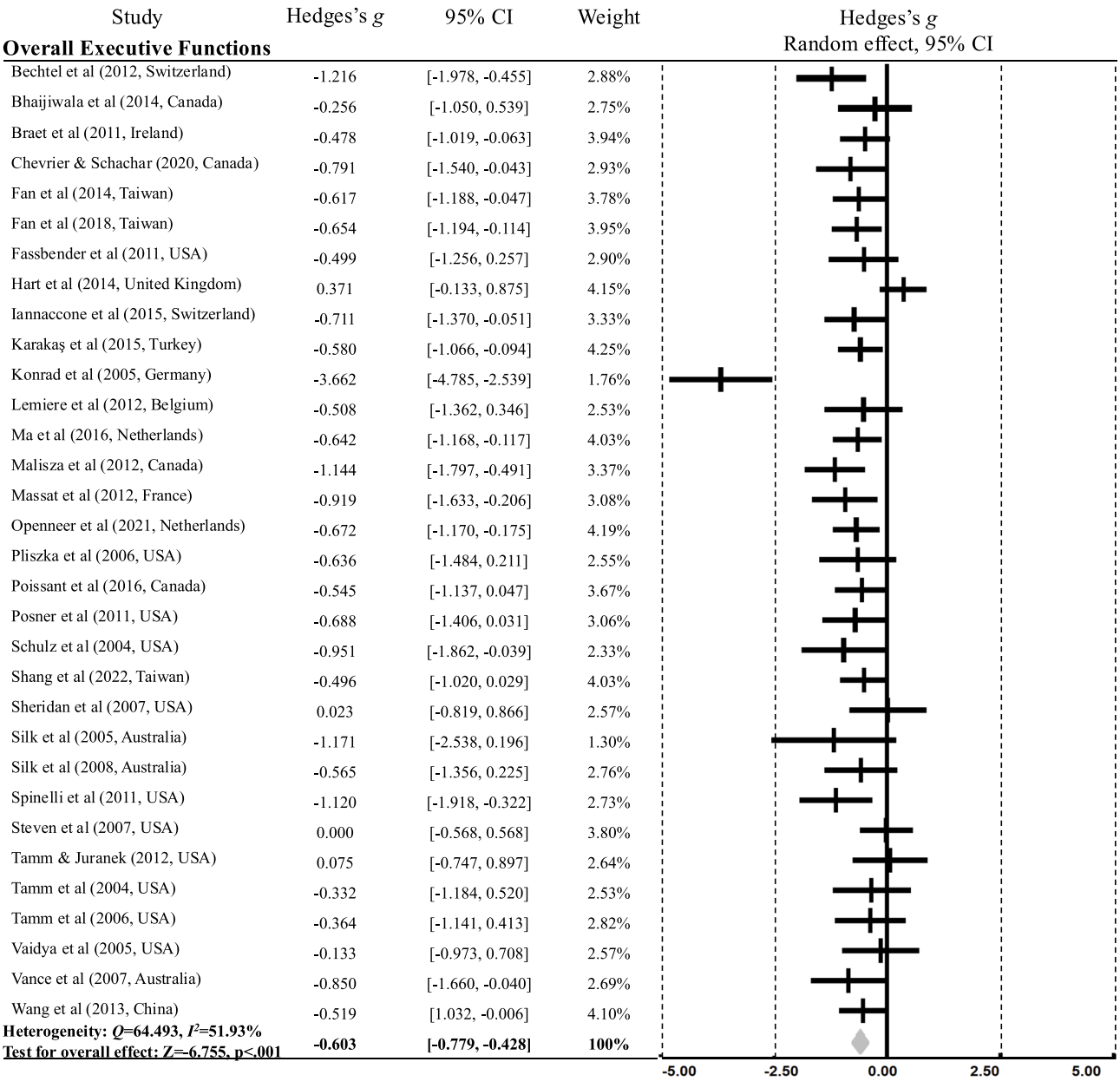

Fig. 2 Meta-analysis of overall executive function

IFG (orbitofrontal), bilateral supramarginal gyrus, bilateral middle occipital gyrus, left inferior parietal gyrus, bilateral angular gyrus, left caudate, and left cerebellar regions Crus1 and Crus2. Conversely, regions of reduced activation in HC relative to ADHD (ADHD > HC) during EF conditions relative to control conditions comprised the left middle frontal gyrus, right supplementary motor area, left insula, left precuneus, left putamen, and right middle temporal gyrus (Table 3 and Fig. 4).

ALE analysis results of 22 inhibition control studies

Out of the 32 studies, 22 investigated IC. Given the limited number of studies on WM and reasoning, we utilized the ALE method only for the studies focusing on IC. Regions of greater activation in HC compared to ADHD (HC > ADHD) were observed under the IC condition, notably in the right IFG (orbitofrontal). In contrast, regions of lower activation in HC compared to ADHD (ADHD > HC) during the IC condition, relative to the control condition, were the right supplementary motor

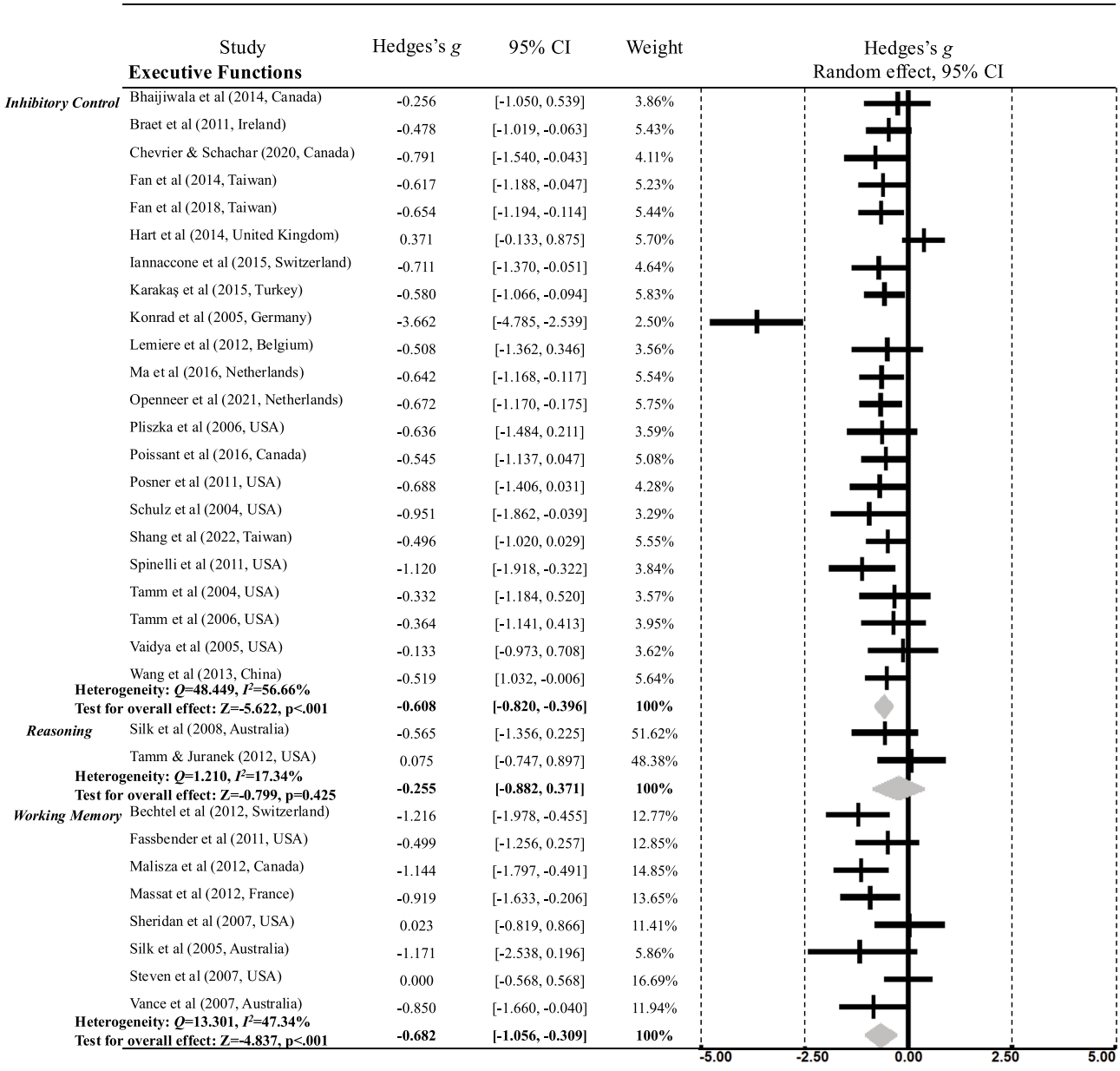

Fig. 3 Meta-analysis of core and higher-level executive function

area, left putamen, and right middle temporal gyrus (Table 4 and Fig. 5).

Correlations between brain activation and EFs

Eight of the studies reported Pearson's correlations between brain activation and neuropsychological performance (e.g., inhibition controls [42, 43, 45, 50, 57, 62] and WM [44, 59]. Specifically, three of the studies reported positive associations between IC and brain activation in the IFG [42, 43, 45] in the ADHD group, and two of the studies reported positive correlations between IC and brain activation in anterior cingulate activation (ACC) in the ADHD group. [42, 57]. In the HC group, two of the studies reported positive associations between IC and brain activation in the left superior parietal lobule (SPL). [42, 43]

We found that in the ADHD group, greater activation in the right IFG (k = 3, $r = 0.336$, 95%CI [0.024, 0.588]) and ACC (k = 2, $r = 0.530$, 95% CI [0.223, 0.742]) were positively correlated with better IC. In the HC group, greater activation in the left superior parietal lobule (SPL) was positively correlated with better IC (k = 2, $r = 0.472$, 95% CI [0.214, 0.669]) (Fig. 6).

Table 3 Significant clusters for the comparison between attention-deficit/hyperactivity disorder (ADHD) and healthy controls (HC) using ale analysis across the entire 32 studies

Region	Hemisphere	AAL	Peak			Z value	Peak ALE *p* value	Adjusted *p* value
			x	y	z			
HC > ADHD								
Inferior frontal gyrus, orbital part	L	15	−38	28	−16	3.73	0.014	0.020*
Inferior frontal gyrus, orbital part	R	16	38	30	−12	4.41	0.018	0.020*
Supramarginal gyrus	L	19	−54	−44	32	3.78	0.014	0.020*
Supramarginal gyrus	R	20	50	−34	34	4.26	0.017	0.020*
Middle occipital gyrus	L	51	−46	−76	10	4.40	0.018	0.020*
Middle occipital gyrus	R	52	44	−74	6	4.11	0.016	0.020*
Inferior parietal gyrus	L	61	−52	−46	44	3.94	0.015	0.020*
Angular gyrus	L	65	−56	−58	28	4.68	0.020	0.020*
Angular gyrus	R	66	46	−60	32	4.36	0.018	0.020*
Caudate	R	72	16	14	10	3.74	0.014	0.020*
Cerebellum_Crus1	L	91	−52	−62	−28	3.87	0.015	0.020*
Cerebellum_Crus2	L	93	−8	−76	−30	3.75	0.014	0.020*
ADHD > HC								
Middle frontal gyrus	L	7	−38	34	36	4.54	0.017	0.017*
Supplementary motor area	R	20	6	−10	66	3.94	0.013	0.017*
Insula	L	29	−40	6	−2	4.21	0.015	0.017*
Precuneus	L	67	−10	−48	14	3.95	0.013	0.017*
Putamen	L	73	−22	10	−6	4.42	0.016	0.017*
Middle temporal gyrus	R	86	52	−6	−18	3.98	0.013	0.017*

* $p < 0.05$, and the P-values were adjusted with false discovery rate correction method

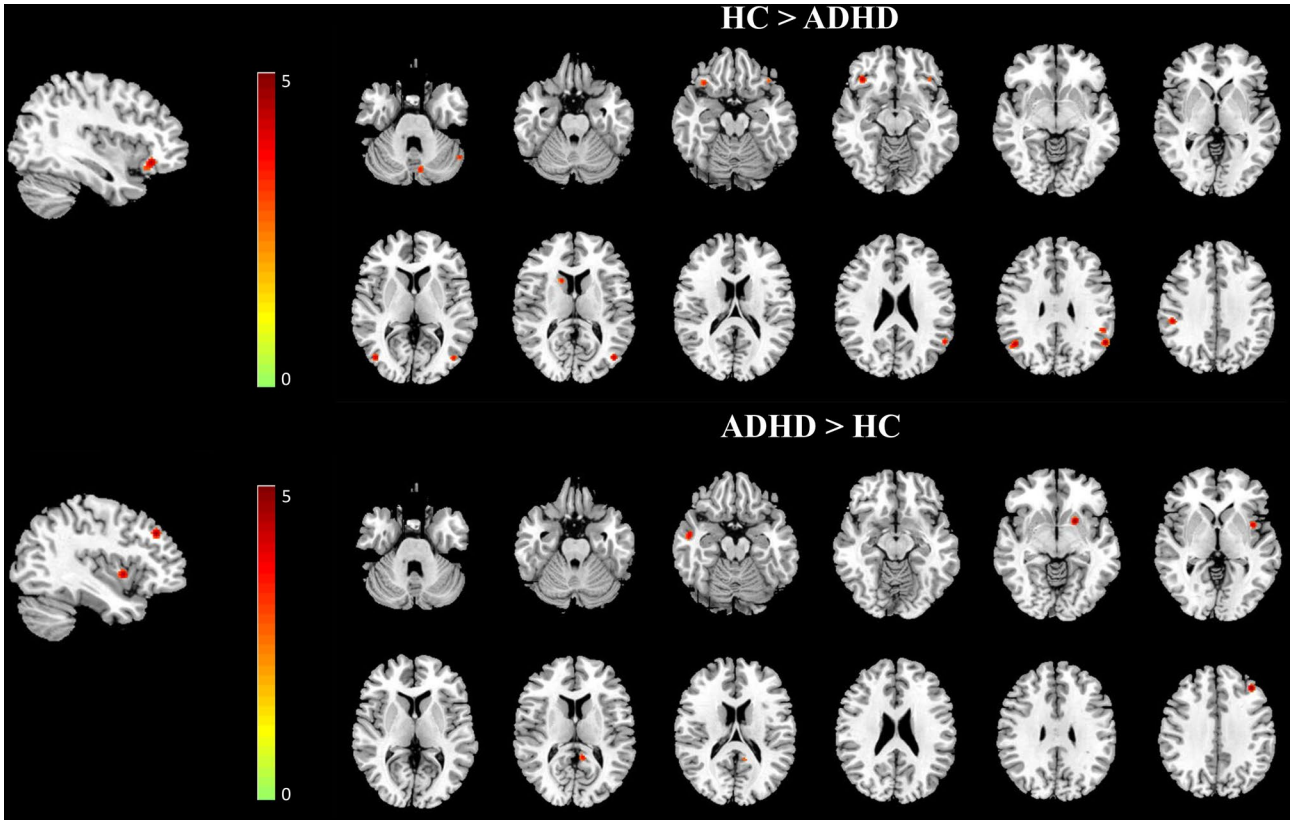

Fig. 4 Ale significant results for the hc > ADHD contrast and ADHD > hc contrast across the entire 32 studies (FDR correction, $p < 0.01$)

Table 4 Significant clusters for the comparison between attention-deficit/hyperactivity disorder (ADHD) and healthy controls (HC) using ale analysis in studies focusing on 22 studies inhibition control

Region	Hemisphere	AAL	Peak			Z value	Peak ALE *p* value	Adjusted *p* value
			x	y	z			
HC > ADHD								
Inferior frontal gyrus, orbital part	R	16	38	30	−12	3.96	0.012	0.012*
ADHD > HC								
Supplementary motor area	R	20	6	−10	66	4.02	0.013	0.016*
Putamen	L	73	−22	10	−6	4.48	0.016	0.016*
Middle temporal gyrus	R	86	52	−6	−18	4.05	0.013	0.016*

* $p < 0.05$, and the P-values were adjusted with false discovery rate correction method

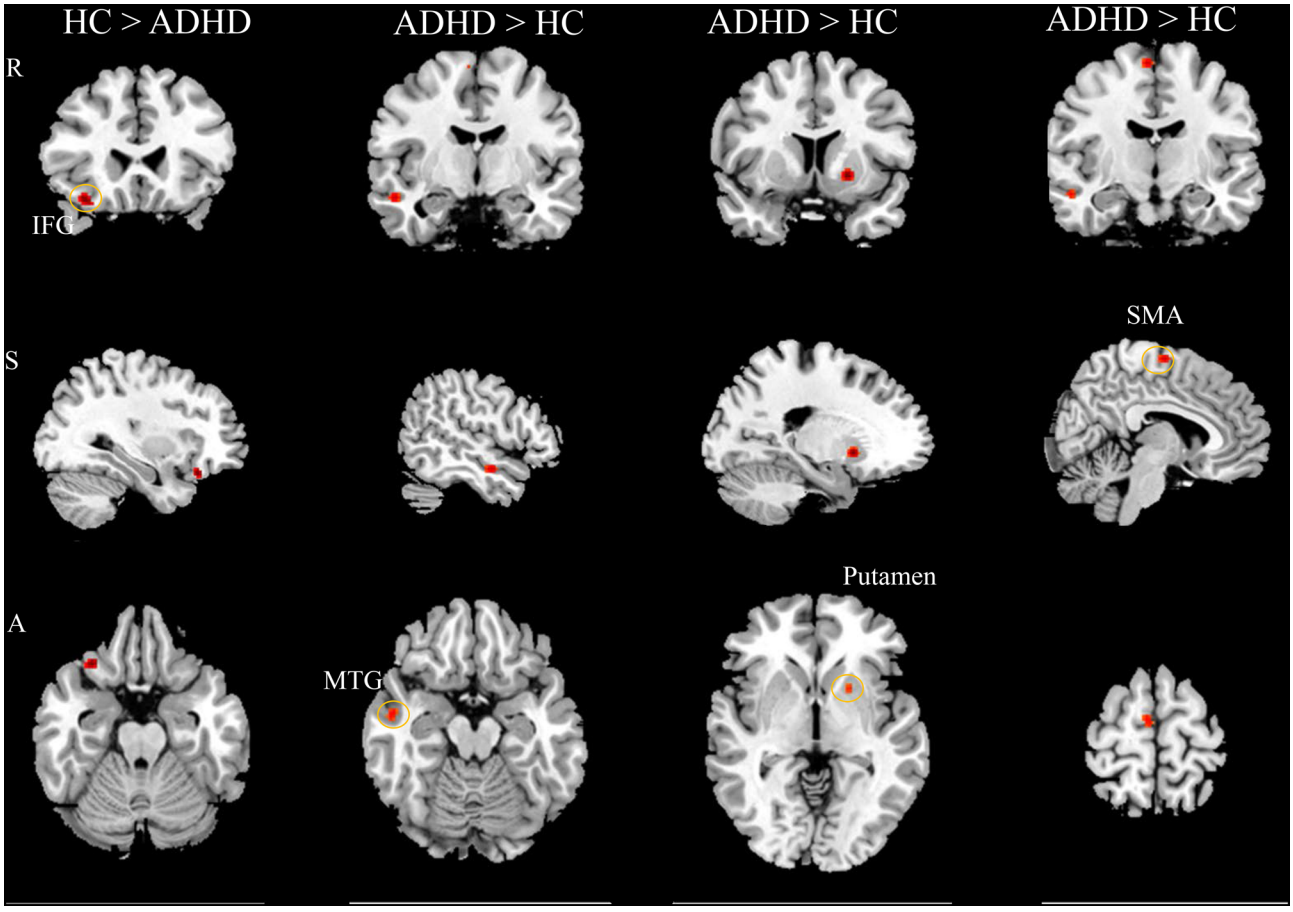

Fig. 5 Ale significant results for the hc > ADHD contrast and ADHD > hc contrast in studies focusing on inhibition control (FDR correction, $p < 0.01$)

Discussion

In this systematic review and ALE meta-analysis, we synthesized data from 32 fMRI studies of children and adolescents with and without ADHD, that used EF tasks and tb-fMRI.

Our results showed that children and adolescents with ADHD had moderate-to-large deficits in overall EFs relative to their peers without ADHD, which is consistent with previous findings [70]. In line with previous studies, our ALE analysis revealed that activation of the IFG in the HC group was significantly greater than in the ADHD group during IC, reasoning, and WM processes. The IFG plays key roles in numerous cognitive domains, including motor control, empathy, language processing, and EFs [71]. Various studies have reported that individuals with ADHD exhibited reduced brain activation in the IFG compared with HC groups.This reduction has been linked to impaired response inhibition [47, 67], difficulties in rule processing or maintenance [61], a lack of readiness to respond to changing conditions [39], reduced WM load effects [59], and deficiencies in attentional orienting and WM processes [63]. Interestingly, four studies, by Fan et al. [42, 43], Schulz et al. [57], and

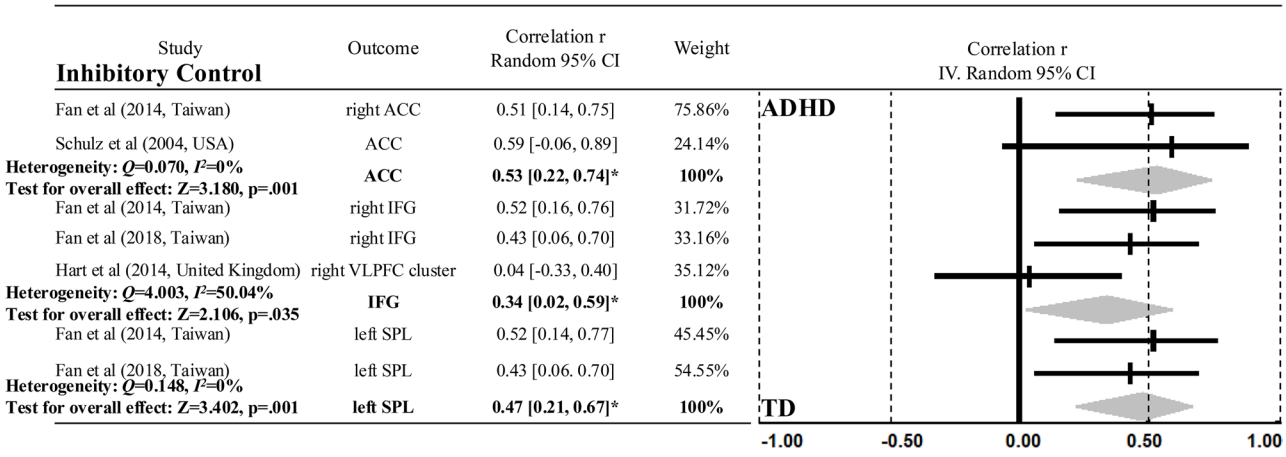

Fig. 6 Meta-analysis of associations between executive function and brain activation

Fassbender et al. [44], found significantly greater activation in the IFG during the Stroop task, go/no-go task, and WM task among adolescents with ADHD than in the HC groups. Schulz et al. attributed the discrepancies to the different tasks used in the studies or variations among the participants in each study [57]. One of the studies suggested that the disparity in IFG findings may also have stemmed from variations in the monoamine oxidase A (MAOA) genotype. This genetic perspective potentially explains the variability in brain activation patterns linked to ADHD [72].

Within developmental neuroscience, these findings emphasise that typical maturation of the prefrontal cortex, including the IFG, supports improved executive EF during adolescence [73]. In neurotypical individuals, increased IFG activation reflects ongoing development of inhibitory control, reasoning, and working memory. In ADHD, reduced IFG activation indicates delayed or atypical maturation of these EF networks. As adolescents grow, neurotypical brains tend to show increased and more targeted IFG activation, leading to better EF. However, in ADHD, these neural differences may persist or worsen, contributing to ongoing deficits [74]. Overall, the developmental trajectory likely involves a lag in IFG maturation in ADHD. Longitudinal studies are needed to determine whether these neural activation patterns catch up over time or remain impaired.

The inferior parietal gyrus is another critical region associated with EFs [75]. The frontal-parietal pathway is often disrupted in individuals with ADHD [76]. Notably, findings related to the parietal gyrus are more consistent across studies [40, 44, 45, 52, 55, 60, 61, 63–65]. These studies have consistently shown reduced activation in the parietal gyrus in ADHD patients compared with HC participants. Braet et al. (2011) revealed that during successful inhibition, two parietal regions, namely the cuneus and inferior parietal gyrus, showed reduced activation in individuals with ADHD. fMRI studies of WM have also shown hypoactivity in parietal regions in ADHD patients [44]. Two other studies related to reasoning found poor performance and hypoactivation in the parietal region [61, 64]. Thus, dysfunction of the parietal gyrus plays a significant role in IC, WM, and reasoning in children and adolescents with ADHD.

In addition to the disrupted activations in cortical regions, recent research has suggested that abnormalities in subcortical regions are also critical in the pathophysiology of ADHD [77]. Consistent with our results, between-group comparisons revealed reduced activation patterns in ADHD children in a widespread cortico-subcortical network, including the bilateral occipital and inferior parietal lobes, the caudate nucleus, and the cerebellum. Importantly, these regions are part of a system innervated by dopaminergic pathways [52]. Heyder's review highlighted that information processing and integration related to executive control are mediated by the frontostriatal and fronto-cerebellar circuits. [78] In the frontostriatal circuitry, there are five distinct loops: the motor loop, the oculomotor loop, the dorsolateral prefrontal loop, the lateral orbitofrontal loop, and the anterior cingulate loop. The motor and oculomotor loops are primarily related to motor functions, while the remaining three—dorsolateral prefrontal, lateral orbitofrontal, and anterior cingulate—are non-motor loops, associated with affect and motivation dysfunctions [79]. According to our findings, the motor-related loops appeared to be more activated in individuals with ADHD than in the HC participants, whereas the non-motor loops tended to be more activated in the HC groups. This finding suggests that the complexity of ADHD symptoms requires treatments that consider both aspects of circuitry function. Five of the 32 screened studies reported hypoactivation in the cerebellum [38, 52, 55, 57, 64]. This included one reasoning task [64], two WM tasks [38, 52], and two inhibition tasks [55, 57]. This result suggests that the

deficits of the cerebellum loop are involved in dysfunctions across all types of EFs in ADHD.

Our results showed that a significant, moderate-to-large difference in IC was observed between those with ADHD and those without. Our results showing a significant deficit in inhibition control in youth with ADHD support Barkley's theory [80] that impaired inhibition is the primary neuropsychological deficit in ADHD. This inhibition impairment likely contributes to broader executive dysfunctions, such as working memory and self-regulation difficulties, which are key features of the disorder. Under the IC condition, individuals with ADHD exhibited lower activation in the right IFG than the HC participants; whereas the ADHD groups showed greater activation in the right supplementary motor area, left putamen, and right middle temporal gyrus. As mentioned above, the IFG is a critical region associated with EFs, playing a vital role in IC and attentional processing [81–83]. The right IFG plays an important role in the suppression of non-essential information as well as in the allocation of sustained attention and task-specific information [7, 21]. Shaw's research indicated that as part of the developmental process, increased cortical thickness in the right orbitofrontal and right inferior frontal cortex would counterbalance the growth in the left occipital regions. However, in ADHD, while the posterior component remained, the prefrontal component was absent [84]. In line with the findings on lateralization, the studies we included demonstrated that the right IFG is less active in ADHD children than in children without ADHD [39, 47, 58].

Similar to what is described above, the cortico-subcortical connections, especially fronto-striatal circuitry, are activated in individuals with ADHD. The putamen and motor cortex are integral components of this circuit [48]. Furthermore, Wang et al. identified abnormal activation within the fronto-temporo-limbic network during inhibition tasks [69]. Our results may reflect a compensatory recruitment of accessory brain regions to accomplish a cognitive task in ADHD patients. In other words, given the atypical activation in sustained-attention regions, an alternative network may be activated in children with ADHD.

Notably, our ALE results revealed no differences in activation in ACC between the ADHD group and the HC group, which contradicted prior findings [42, 43, 54, 57, 66, 69]. The ACC plays a crucial role in processing both top–down and bottom–up information, as well as in monitoring conflicting neural processes by facilitating connectivity between the prefrontal and parietal cortices [85]. Compared with the HC group, the adolescents with ADHD had lower ACC activation during the "no go trials" in the go/no go task [66] and greater ACC activation in the Stroop task (incongruent versus congruent) [42, 43]. These discrepant findings in ACC activation may be a key factor contributing to the absence of significant ACC clusters in our ALE analysis. Moreover, distinct neural processes are reflected by different contrasts, even when the same tasks are employed. For instance, in go/no-go tasks, go trials typically engage neural activity associated with response inhibition, whereas no-go trails generally assess neural activity linked to error monitoring [83]. Furthermore, certain studies have indicated that adolescents with childhood ADHD exhibit greater activation in the ACC and IFG under the incongruent condition than under the congruent condition [43], whereas others have demonstrated reduced activation in the IFG under the same condition [58], which reflects the effects of varying subject populations across study contexts on activation results [57].

More importantly, we found a significant positive correlation between inhibition control and brain activation in the IFG ($r = 0.336$, $p = 0.035$). Greater IFG activation was positively correlated with rapid visual information processing, indicating that the ADHD group may have required increased activation to compensate for deficits in IC [42, 43]. Taken together, our findings lend support to interventions targeting neural mechanisms of IC in children and adolescents with ADHD [86].

At the behavioral level, a statistically significant group difference in WM was observed ($g = -0.682$). Significantly poorer WM was found in youth with ADHD than in their peers without ADHD. Eight neuroimaging studies have compared brain activation patterns between children and adolescents with ADHD and those without during WM-related tasks. Two of these studies utilized the N-back paradigm but used different contrasts. Specifically, Bechtel et al. [38] used 2-back and 3-back WM tasks to compare neural activation between ADHD and HC groups. They found that the HC group exhibited greater activation than the individuals with ADHD in the frontal cortex, insula, parietal cortex, cerebellum, and cingulate gyrus. Similarly, Massat et al. [52] used an N-back task to compare activation between the 2-back and 0-back conditions. Their findings indicated that the HC group showed greater activation than the individuals with ADHD in multiple areas, namely the bilateral inferior parietal lobule, bilateral angular gyrus, left inferior parietal lobule, bilateral posterior cingulate cortex, left middle cingulate cortex, left calcarine fissure, right lingual gyrus, right caudate nucleus, and bilateral cerebellum. These results suggested a consistent pattern, whereby the HC group exhibited greater activation than the ADHD group during N-back tasks. The mental rotation task was investigated in two other studies. Silk et al. [60] conducted a mental rotation task and found greater activation in the HC group than in the ADHD group. Enhanced activity in HC participants was observed in the

superior and inferior frontal gyri, the right IFG extending into the right caudate head, and the right superior and inferior parietal lobules. In contrast, the ADHD group showed greater activation in the left middle and superior temporal gyri, posterior cingulate cortex, and medial superior prefrontal cortex. Vance et al. [68] also used a mental rotation task and observed that the HC had greater activation than the individuals with ADHD in the right precuneus, right cuneus, right inferior parietal lobule, and right caudate nucleus. The discrepancies between the findings of these two studies may be attributed to differences in sample sizes and the statistical correction methods used.

Other fMRI studies show inconsistent WM findings, likely due to differences in task design. Examples include oddball [63], visual serial addition [44], and delayed match-to-sample tasks [59], and spatial WM [51]. For instance, using the delayed match-to-sample task, Sheridan et al. [59] found that the HC group generally exhibited higher activation than the ADHD group in areas including the right IFG and right superior parietal lobe. In contrast, Fassbender et al. [44] observed increased activity in the ADHD group in four brain regions during the visual serial addition task, namely the right insula/claustrum, IFG, putamen, and left medial frontal gyrus. It is difficult to synthesize the fMRI results, due to the high levels of heterogeneity among the included studies. Generally, the HC groups showed greater activation in regions associated with WM, including the frontal cortex, parietal lobes, and cerebellum. In some cases, the individuals with ADHD demonstrated increased activation in other brain areas, which may suggest a compensatory mechanism or differences in neural processing strategies.

There are several limitations in this study. First, only a small number of studies examined WM and reasoning in relation to brain activations in children and adolescents with ADHD. This scarcity limited our ability to analyze these particular domains of EFs with ALE meta-analysis. When fewer studies contribute, convergence across experiments is less stable. As a result, the ALE findings may not capture the full range of neural substrates. Accordingly, these ALE results should be considered preliminary until validated by larger datasets in future research. Second, fMRI data do not establish a definitive or quantitative baseline state of activation [87], as they rely on variations in signal between two conditions. Therefore, interpreting these data requires careful consideration of the relative changes of brain activity, and longitudinal approaches may strengthen inference. Third, male participants dominated the children and adolescents with ADHD study samples (82.0% boys), limiting our ability to detect differences in EFs and brain activations between sexes; future studies should include more balanced samples. Fourth, genetic makeup that can influence brain activity was not consistently reported, which constrains interpretation; genotype-informed designs are recommended. Fifth, we found that in our included studies, measuring task-based fMRI for inhibitory control and working memory are the dominant focuses in children and adolescents with ADHD. Therefore, task-based fMRI studies that target cognitive flexibility and higher-order functions (e.g., planning, problem-solving skills) are recommended for future research. Lastly, considerable heterogeneity existed across the included studies in both the tools and protocols used in assessing EFs and fMRI. Such variability may have reduced the comparability of findings and lowered the specificity of convergent activations. It may also have obscured domain-specific effects. Accordingly, our ALE results should be regarded as identifying core brain regions that generalize across these studies, rather than networks unique to particular tasks or participant characteristics. Future adoption of more harmonized task protocols could improve the consistency and interpretability of findings.

Conclusion

This study examined ADHD research through ALE analysis in an attempt to clarify the relationship between brain regions that regulate EFs and behavior. Our results highlight a consistent disruption in the frontoparietal pathway in ADHD, characterized by hypoactivation, along with altered connections between cortical and subcortical regions. These findings advance understanding of the pathophysiology of ADHD and suggest potential targets for intervention. However, several limitations should be noted. Substantial heterogeneity in task paradigms and the limited number of studies in some EF domains reduce the specificity and generalizability of our conclusions. Future studies should adopt more harmonized task protocols, enlarge sample sizes across domains, and integrate complementary analytic approaches. Such efforts will provide a more stable picture of EF dysfunction in ADHD and help identify precise neural targets for interventions and treatments aimed at strengthening executive and cortico-subcortical networks.

Supplementary information

The online version contains supplementary material available at https://doi.org/10.1186/s12888-025-07593-7.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Acknowledgements

Everyone who contributed significantly to the work has been listed.

Author contributions
HZ & XL contributed to the Conceptualization, Methodology, Validation, Data Curation, Writing – original draft, Writing – reviewing & editing, Visualization. RC, YSF & YKY contributed to the Methodology, Validation, Data Curation. CLH, DL, PW contributed to the Methodology, Writing – reviewing & editing. DS contributed to the Conceptualization, Methodology, Writing – reviewing & editing.

Funding
Not applicable.

Data availability
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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Received: 29 July 2025 / Accepted: 31 October 2025
Published online: 18 November 2025

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