Psychol Med. Author manuscript; available in PMC 2020 October 01.

Published in final edited form as:
Psychol Med. 2020 April ; 50(6): 894–919. doi:10.1017/S0033291720000574.

Comparative Meta-analyses of Brain Structural and Functional Abnormalities during Cognitive Control in Attention-Deficit/ Hyperactivity Disorder and Autism Spectrum Disorder

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Abstract

Background—People with attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) have abnormalities in frontal, temporal, parietal and striato-thalamic networks. It is unclear to what extent these abnormalities are distinctive or shared. This comparative meta-analysis aimed to identify the most consistent disorder-differentiating and shared structural and functional abnormalities.

Methods—Systematic literature search was conducted for whole-brain voxel-based morphometry (VBM) and fMRI studies of cognitive control comparing people with ASD or ADHD with typically developing controls. Regional gray matter volume (GMV) and fMRI abnormalities during cognitive control were compared in the overall sample and in age-, sex- and IQ-matched subgroups with seed-based *d* mapping meta-analytic methods.

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Conflicts of interest: KR has received grants from Lilly and Shire pharmaceuticals and speaker's bureau from Shire, Lilly and Medice. Other authors report no biomedical financial interests or potential conflicts of interest.

Ethical standards: The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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Results—Eighty-six independent VBM (1533 ADHD and 1295 controls; 1445 ASD and 1477 controls) and 60 fMRI datasets (1001 ADHD and 1004 controls; 335 ASD and 353 controls) were identified. The VBM meta-analyses revealed ADHD-differentiating *decreased* ventromedial orbitofrontal ($z = 2.22$, $p < .0001$) but ASD-differentiating *increased* bilateral temporal and right dorsolateral prefrontal GMV ($zs \geq 1.64$, $ps \leq .002$).The fMRI meta-analyses of cognitive control revealed ASD-differentiating medial prefrontal underactivation but overactivation in bilateral ventrolateral prefrontal cortices and precuneus ($zs \geq 1.04$, $ps \leq .003$). During motor response inhibition specifically, ADHD relative to ASD showed right inferior fronto-striatal underactivation ($zs \geq 1.14$, $ps \leq .003$) but shared right anterior insula underactivation.

Conclusions—People with ADHD and ASD have mostly distinct structural abnormalities, with enlarged fronto-temporal GMV in ASD and reduced orbitofrontal GMV in ADHD; and mostly distinct functional abnormalities, which were more pronounced in ASD.

1 Introduction

Attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are distinct neurodevelopmental conditions. ADHD is characterized by age-inappropriate symptoms of inattention, hyperactivity and impulsivity, whereas ASD is characterized by difficulties in social interaction/communication and stereotypical repetitive behavior (American Psychiatric Association (APA), 2013). The estimated prevalence of ADHD (5-7%) is higher than ASD (1-2%) worldwide (Elsabbagh et al., 2012; Polanczyk, Willcutt, Salum, Kieling, & Rohde, 2014). In both disorders, brain structural abnormalities have been observed in frontal, temporal, parietal and striato-thalamic networks (Ecker, 2017; Norman et al., 2016), although the extent of their overlap is poorly understood. Furthermore, both disorders are associated with cognitive control deficits (F. Craig et al., 2016), although ASD relative to ADHD seem less severely impaired (see meta-analyses Lipszyc & Schachar, 2010; Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008) and display more heterogeneous performance (Geurts, van den Bergh, & Ruzzano, 2014; Kuiper, Verhoeven, & Geurts, 2016). Exploring abnormalities in brain structure and cognitive control function could help elucidate the disorder-differentiating and shared difficulties in ASD and ADHD.

In ADHD, brain abnormalities are thought to represent delayed maturation in brain structure and function mediating late-developing cognitive functions such as cognitive control (Hoogman et al., 2017; Shaw et al., 2007; Sripada, Kessler, & Angstadt, 2014). Correspondingly, previous voxel-based morphometry (VBM) meta-analyses have revealed reduced gray matter volume (GMV) in ventromedial orbitofrontal cortex (vmOFC) and basal ganglia (BG) (Frodl & Skokauskas, 2012; Nakao, Radua, Rubia, & Mataix-Cols, 2011; Norman et al., 2016). In addition, mega-analytic studies by the ENIGMA consortium of subcortical and cortical structure involving over 1700 and 2200 individuals with ADHD, respectively, found reduced GMV in BG, amygdala and hippocampus (Hoogman et al., 2017), as well as reduced cortical surface areas and thickness in several frontal, temporal and parietal regions (Hoogman et al., 2019).

Converging with these findings are meta-analytic reports of underactivation in lateral and medial frontostriatal networks in ADHD relative to controls during cognitive control such as

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in right inferior frontal gyrus (IFG)/anterior insula (AI), supplementary motor area (SMA)/anterior cingulate cortex (ACC), and the striatum (Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013; McCarthy, Skokauskas, & Frodl, 2014; Norman et al., 2016). The role of right IFG as potential biomarker of ADHD in particular has been discussed in several studies and a meta-analysis showing disorder-specificity of underactivation in right and/or left IFG during cognitive control in ADHD children relative to pediatric bipolar disorder (Passarotti, Sweeney, & Pavuluri, 2010), obsessive-compulsive disorder (Norman et al., 2016) and conduct disorder (Rubia, 2018; Rubia, Halari, et al., 2010).

In ASD, findings from neuroimaging, post-mortem and head circumference development studies have led to the “early brain overgrowth theory” (Courchesne, Campbell, & Solso, 2011; Lange et al., 2015; Redcay & Courchesne, 2005; Schumann et al., 2010), which, however, has been contested in recent studies (Ecker, Schmeisser, Loth, & Murphy, 2017; Raznahan et al., 2013). Meta-analytic and mega-analytic findings have varied but GMV reduction in the striatum and amygdala/hippocampus were among the more frequently reported (Cauda et al., 2011; DeRamus & Kana, 2015; Nickl-Jockschat et al., 2012; van Rooij et al., 2018; Via, Radua, Cardoner, Happe, & Mataix-Cols, 2011). Another recent meta-analysis of VBM studies in over 900 ASD patients, however, primarily found cortical abnormalities such as GMV decrease in medial prefrontal cortex (mPFC) and posterior insula; and increase in left anterior temporal, right inferior temporo-parietal, left dorsolateral prefrontal (dlPFC) and precentral cortices (Carlisi et al., 2017). Furthermore, the ENIGMA mega-analysis has found cortical thickness increase in frontal and decrease in temporal areas (van Rooij et al., 2018).

The whole-brain meta-analysis of fMRI studies of cognitive control in ASD (Carlisi et al., 2017) has found underactivation in salience and executive network areas such as mPFC, left dorsolateral and right ventrolateral prefrontal cortices (vlPFC)/AI, left cerebellum, inferior parietal lobe (IPL) and right premotor area; and overactivation in right temporo-parietal regions including default mode network (DMN) areas.

While these meta-analytic findings suggest partially overlapping brain abnormalities in ASD and ADHD, only direct comparisons can elucidate their differences and commonalities. Among such studies, a VBM study showed reduced GMV in ADHD relative to ASD in right posterior cerebellum and ASD-differentiating reduced left middle/superior temporal gyri (M/STG) (Lim et al., 2015). Furthermore, fMRI studies have shown ASD-differentiating mPFC underactivation during reward reversal (Chantiluke, Barrett, Giampietro, Brammer, Simmons, Murphy, et al., 2015), shared dlPFC underactivation during sustained attention and working memory (Chantiluke, Barrett, Giampietro, Brammer, Simmons, & Rubia, 2015; Christakou et al., 2013); and shared precuneus abnormalities during sustained attention, reward reversal and the resting state (Chantiluke, Barrett, Giampietro, Brammer, Simmons, Murphy, et al., 2015; Christakou et al., 2013; Di Martino et al., 2013). Importantly, during successful motor inhibition, ADHD-differentiating underactivation was observed in left vlPFC and BG and ASD-differentiating overactivation in bilateral IFG (Chantiluke, Barrett, Giampietro, Santosh, et al., 2015). Few imaging studies, however, have directly compared the two disorders and their relatively small sample sizes have limited statistical power.

We therefore conducted meta-analyses of structural and functional abnormalities related to functions that are commonly impaired in ASD and ADHD, i.e., cognitive control. The aim of the study was to establish the most consistently disorder-differentiating and shared structural and functional deficits, which is important for developing disorder-specific or transdiagnostic treatment. Guided by previous comparative studies and meta-analyses in ASD and/or ADHD, we hypothesized that these disorders are characterized by both shared abnormalities in salience, cognitive control, and default mode networks as well as disorder-differentiating impairments. Structurally, we hypothesized ADHD-differentiating reduced GMV in BG/insula relative to ASD (Lim et al., 2015; Norman et al., 2016), and ASD-differentiating increased GMV in frontal and temporo-parietal regions (Carlisi et al., 2017; Lim et al., 2015). During cognitive control, we expected ADHD-differentiating reduced activation in right IFG and BG relative to ASD (Chantiluke, Barrett, Giampietro, Santosh, et al., 2015; Norman et al., 2016; Passarotti et al., 2010; Rubia et al., 2009), ASD-differentiating underactivation relative to ADHD in the medial frontal part of the cognitive control network (Carlisi et al., 2017; Chantiluke, Barrett, Giampietro, Brammer, Simmons, Murphy, et al., 2015), and shared abnormal overactivation in precuneus in both disorders (Chantiluke, Barrett, Giampietro, Brammer, Simmons, Murphy, et al., 2015; Christakou et al., 2013; Di Martino et al., 2013).

2 Methods

2.1 Publication Search and Study Inclusion

Systematic literature search was conducted for peer-reviewed English language publications in PubMed, Scopus, Web of Science and ScienceDirect until 30th November 2018, for whole-brain fMRI or VBM studies in ASD and ADHD. Manual search was conducted in reference lists of previous meta-analyses. Included studies compared the ASD or ADHD groups against typically developing (TD) controls on GMV or on cognitive-control fMRI BOLD signal from stop-signal, go/no-go, switch, Stroop, Simon and flanker, using predefined inhibitory contrasts (Supplement 1). Only whole-brain neuroimaging data were included, to prevent bias towards specific neuroanatomy (Friston, Rotshtein, Geng, Sterzer, & Henson, 2006). Excluded studies used region of interest (ROI) approaches or involved <10 patients, which were deemed lacking in statistical power (Desmond & Glover, 2002; Nakao et al., 2011; Thirion et al., 2007). Studies that potentially report duplicate data from other publications (including mega-analyses), have no TD controls, or have provided no peak coordinates, were excluded. When samples overlapped, the largest sample was included. Mean age, proportion of males, mean IQ, cognitive control tasks, current and lifetime psychostimulant use (i.e., proportion of participants being prescribed the medication) and effect size and coordinate location of peaks for regional GMV and activation differences were extracted from each study and authors were contacted for missing information. Reports of exclusion or inclusion of people with co-occurring ASD or ADHD in the counterpart disorder groups were also extracted from the literature. Data were extracted from all studies by SL, 64% of which were also extracted by LN and CC who achieved 100% agreement. This meta-analysis was not pre-registered in a time-stamped, institutional registry prior to the research being conducted.

2.2 Meta-Analyses

The anisotropic seed-based *d* Mapping (AES-SDM) meta-analytic software (www.sdmproject.com; Supplement 1) employed in previous meta-analyses of ASD and ADHD (Carlisi et al., 2017; Frodl & Skokauskas, 2012; McCarthy et al., 2014; Norman et al., 2016) was used for the present voxel-wise meta-analyses. The software can accommodate statistical parametric maps or peak coordinates and effect sizes (*t*-scores) data. For the latter, AES-SDM computes signed (positive/negative) effect sizes and variance maps of brain regional differences between clinical and control groups by convolving an anisotropic non-normalized Gaussian kernel with Hedges effect size of each peak. Voxels correlated with the peak values are assigned higher effect sizes. Maps are then combined across studies based on random-effect model, accounting for sample size, within-study variability and between-study heterogeneity. Correlated datasets (e.g., when the same group of participants completed several cognitive tasks) were included in the meta-analysis as a single set (Norman et al., 2016), adjusted for shared variance of brain activation or structure across datasets. The Meta-analysis of Observational Studies in Epidemiology (MOOSE) guidelines were followed (Stroup et al., 2000).

Age, sex and IQ were compared across patient and control groups prior to meta-analyses in STATA14 (StataCorp, 2015), using sample-size weighted *F*-statistics. First, VBM meta-analyses were conducted in each disorder relative to TD controls and then between the two disorders. Second, equivalent fMRI meta-analyses were conducted to examine the neural impairments observed during cognitive control within and between disorders using all available data. To explore specific cognitive-control subconstructs (e.g., Bari & Robbins, 2013), we conducted fMRI sub-meta-analyses of prepotent motor response inhibition, including studies employing the go/no-go and stop-signal tasks only. No other cognitive control subconstructs were explored due to insufficient data. To find the most consistent disorder-differentiating neural abnormalities regardless of demographic characteristics, we conducted sensitivity meta-analyses in an age-, sex- and IQ-matched subgroup, instead of covarying group differences in these variables that could result in false positives (Dennis et al., 2009; Miller & Chapman, 2001). An iterative algorithm computed group differences in age, sex and IQ for all possible combinations of studies, selecting the largest subgroups with statistically nonsignificant group differences in the three variables (Carlisi et al., 2017). Findings surviving the sensitivity analyses in the matched subgroups are the only ones reported and discussed.

Furthermore, conjunction analyses were used to asses overlapping fMRI and GMV abnormalities within and across disorders (using threshold $p < .005$). To assess consistency of findings across age groups, supplementary age-stratified sub-meta-analyses were conducted in overall pediatric or adult age groups and in their respective age-, sex- and IQ-matched subgroups, where possible (Supplements 3 and 4). These were complemented with a series of sample-size weighted correlational analyses between the mean sample age and extracted effect sizes of brain abnormalities averaged over 10-mm radius spheres centered at the peaks of disorder-differentiating or shared abnormalities, applying Bonferroni multiple comparison correction (Supplement 5). Non-parametric correlations were chosen due to the bimodal distribution of age among studies. Following previous meta-analyses (Hart et al.,

2013; Norman et al., 2016), psychostimulant effects were explored in separate meta-regressions between brain structure/function and lifetime psychostimulant exposure, i.e., proportions of samples who have ever been exposed to psychostimulants, or current psychostimulant exposure, in brain regions that are found impaired in ADHD (Supplement 6). Supplementary meta-analyses covarying for task types (motor response inhibition, cognitive interference, motor switching, combination of tasks) and behavioral task performance (impaired, unimpaired) were also conducted to assess their impacts on disorder-differentiating findings (Supplement 7).

A statistical threshold $p < .005$ was used for all meta-analyses (Radua & Mataix-Cols, 2009; Radua, Mataix-Cols, et al., 2012), and a reduced threshold $p < .0005$ and a cluster extent 20 voxels was used in the meta-regressions to control for false positives (Radua, Borgwardt, et al., 2012). Egger's tests assessed potential publication bias in the disorder-differentiating and shared findings. Jack-knife sensitivity analyses examined replicability of findings through iterative whole-brain meta-analyses, leaving one dataset out at a time (Supplement 8; Radua & Mataix-Cols, 2009).

3 Results

3.1 Search Results and Sample Characteristics

The systematic literature search identified 140 VBM and fMRI articles of cognitive control in ASD and ADHD. Accounting for datasets of same clinical participants and, where possible, separating data from pediatric and adult participants resulted in 86 independent VBM datasets (38 datasets including 1533 ADHD participants and 1295 controls; 48 datasets involving 1445 ASD participants and 1477 controls) and 60 independent cognitive-control fMRI datasets (42 datasets involving 1001 ADHD participants and 1004 controls; 18 datasets involving 335 ASD participants and 353 controls) (Tables 1-2). Groups did not differ in age in VBM, $F(3, 83) = 1.24$, $p = .30$, and fMRI datasets, $F(3, 56) = .68$, $p = .57$, but did in sex in VBM, $F(3, 83) = 6.03$, $p = .0009$, and fMRI datasets, $F(3, 56) = 5.93$, $p = .0014$; and in IQ in VBM, $F(3, 67) = 16.2$, $p < .0001$) and fMRI datasets $F(3, 45) = 15.5$, $p < .0001$. See Table 3 for demographic characteristics.

3.2 Disorder-Differentiating and Shared Brain Structure Abnormalities

3.2.1 ADHD VBM—Relative to TD controls, ADHD had reduced GMV in vmOFC/vmPFC/rostral ACC (rACC), extending into right caudate, and in right putamen/posterior insula/STG, left precingulate gyrus (pre-CG), right rostrolateral PFC and left vlPFC/STG/temporal pole (Table 4A-i; Figure 1A-i). Age-stratified analyses showed reduced GMV in right STG/putamen/posterior insula, right caudate and rACC/ventromedial PFC (vmPFC) among pediatric participants and vmOFC/subcallosal gyrus in both age groups (Supplements 3,4). Current and lifetime psychostimulant exposures were associated with increased vmOFC GMV (Supplement 6).

3.2.2 ASD VBM—Relative to TD controls, ASD had GMV reduction in dACC/dorsomedial PFC (dmPFC), left cerebellum, right hippocampus/PHG/FFG and dorsomedial thalamus; and enhancement in left anterior inferior/middle/superior temporal gyri (I/M/

STG)/posterior insula, bilateral precuneus/posterior cingulate cortex (PCC) and right middle/superior frontal gyrus (M/SFG) (Table 4A-ii; Figure 1A-ii). Most abnormalities in the overall sample, i.e., in left cerebellum, dorsomedial thalamus, precuneus/PCC and right MFG/SFG/dlPFC GMV were also found among pediatric participants, and enhanced left anterior STG/MTG GMV was found in both age subgroups (Supplements 3,4).

3.2.3 ADHD *vs*. ASD VBM—People with ADHD, relative to ASD, had consistently disorder-differentiating reduced vmOFC/rACC/left caudate GMV; while people with ASD, relative to ADHD, showed increased GMV in left anterior STG/MTG, right MFG/SFG/dlPFC, and right posterior MTG/STG (Table 4A-iii,iv; Figure 1A-iii,iv). Reduced vmOFC GMV was most consistently ADHD-differentiating in adults, while the medial prefrontal GMV reduction in rACC/dmPFC was more apparent among pediatric participants (Supplements 3,4).

3.3 Disorder-Differentiating and Shared Brain Abnormalities during Cognitive Control

3.3.1 ADHD fMRI—During cognitive control, ADHD relative to TD controls, showed underactivation in right thalamus/caudate, left MTG/STG/superior temporal pole, bilateral SMA/dmPFC, left IFG/AI/temporal pole, right AI/putamen, left postcingulate gyrus (post-CG), left MFG/dlPFC and right MTG (Table 4B-i, Figure 1B-i). Age-stratified analyses showed underactivated dmPFC clusters and left post-CG among pediatric participants, in all regions but the dmPFC and left post-CG in adults; and in left M/STG/temporal pole in both groups (Supplements 3,4). Lifetime psychostimulant exposure was positively associated with activation in left IFG (Supplement 6).

During motor response inhibition specifically, underactivation was found in ADHD relative to TD controls in left MFG/dlPFC, left anterior MTG/STG, left post-CG, right vlPFC/OFC/AI,right IFG, and right caudate (Table 4C-i, Figure 1C-i). Age-stratified analyses showed that underactivation in left MTG/STG was apparent among pediatric participants, underactivation in left MFG/dlPFC was apparent in adults, while underactivation in right vlPFC/AI was apparent in both age groups (Supplements 3,4).

3.3.2 ASD fMRI—During cognitive control, ASD relative to TD controls showed underactivation in ACC/midcingulate/dmPFC, left MFG/dlPFC, right MFG/dlPFC, left IPL and left lingual gyrus/cerebellum; and overactivation in left precuneus/midcingulate, right inferior occipital gyrus (IOG), left vlPFC/OFC, left MFG/rostrolateral PFC and right IFG (Table 4B-ii, Figure 1B-ii). Underactivation in rdACC/dmPFC and left IPL; and overactivation in precuneus, left vlPFC/OFC and left MFG/dlPFC were found in adults (Supplements 3,4). No pediatric sub-meta-analyses were conducted due to insufficient fMRI data.

During motor response inhibition, underactivation was found in ASD relative to TD controls in right AI/vlPFC, left cerebellum, right MFG/dlPFC and right PCC/precuneus; while overactivation was found in left vlPFC/OFC and right IOG/fusiform gyrus (FFG)/ITG (Table 4C-ii; Figure 1C-ii). No age-stratified sub-meta-analyses were conducted due to insufficient data.

3.3.3 ADHD *vs*. ASD fMRI—During cognitive control, ASD-differentiating underactivation was found in rACC/midcingulate/dmPFC and left MFG/dlPFC; and overactivation was found in precuneus, right IOG/FFG, right IFG and left vlPFC/OFC (Table 4B-iii,iv; Figure 1B-iii,iv). The age-stratified analysis, which was conducted only in adults due to available data, found ASD-differentiating underactivation relative to ADHD in rdACC/dmPFC and overactivation in precuneus and left vlPFC/OFC (Supplement 4). No ADHD-differentiating abnormalities were found.

During motor response inhibition, ADHD-differentiating underactivation was found in right caudate and IFG. Furthermore, ASD-differentiating underactivation was found in left lingual gyrus/FFG/cerebellum, right precuneus and right MFG/dlPFC; while overactivation was found in right IOG/FFG, left vlPFC/OFC and left MFG/SFG/dlPFC (Table 4C-iii,iv; Figure 1C-iii,iv). Underactivation in right AI (MNI coordinates: 40, 20, -6; 51 voxels) was shared between disorders.

3.5 Multimodal VBM and fMRI Analyses

In ADHD relative to TD controls, overlapping reduced GMV and fMRI underactivation was found during cognitive control in right caudate nucleus (MNI coordinates: 10, 10, 8; 194 voxels). In ASD relative to TD controls, reduced GMV and fMRI underactivation was found in dACC/mPFC (MNI coordinates: 0, 40, 16; 575 voxels).

3.6 Controlling for Task Type and Performance in fMRI Meta-Analysis

The disorder-differentiating impairment during cognitive control was unchanged after covarying for task types and performance. During motor response inhibition, ADHD-differentiating underactivation in right IFG and caudate did not survive covarying for task performance (Supplement 7).

3.7 Publication Bias and Jack-Knife Reliability Findings

Egger tests suggest no significant publication bias for the structural, ts(84) = .32-2.29, ps ≥ .1, and functional abnormalities during cognitive control, ts(58) = .04-1.90, ps ≥ .38, and during motor response inhibition specifically, ts(40) = .41-1.37, ps ≥ .99 (Bonferroni adjusted p-values). Jack-knife reliability analyses suggest robust disorder-differentiating findings (Supplement 8).

4 Discussion

We aimed to elucidate the most consistent disorder-differentiating and shared brain abnormalities in ASD and ADHD. The findings revealed predominantly disorder-differentiating abnormalities, particularly striking in the VBM meta-analysis, where we found ADHD-differentiating *reduced* GMV in vmOFC; and ASD-differentiating *increased* GMV in bilateral temporal and right lateral prefrontal cortices. In fMRI, the findings overall showed predominantly ASD-differentiating abnormalities, including underactivation in medial frontal and overactivation in bilateral ventrolateral regions and precuneus during cognitive control. During motor response inhibition specifically, ADHD-differentiating underactivation was in right IFG and caudate, while ASD had differentiating underactivation

mostly in posterior regions, and overactivation in left dorsal and ventral frontal regions (Figure 2). Both disorders shared underactivation in right AI. The findings overall suggest that people with ADHD and ASD have mostly distinct brain abnormalities.

The ADHD-differentiating decreased GMV in vmOFC relative to ASD may be related to common reports of reward-based decision-making neural network dysfunctions in ADHD (e.g., Chantiluke et al., 2014; Cubillo, Halari, Smith, Taylor, & Rubia, 2012; Rubia, 2018; Tegelbeckers et al., 2018), and supports the hypothesis of distinctive reward processing in ASD and ADHD (Kohls et al., 2014; van Dongen et al., 2015). Reduced vmOFC GMV in ADHD compared to TD controls extends relatively recent meta-analytic findings (McGrath & Stoodley, 2019; Norman et al., 2016), and there have been correlational reports between OFC GMV reduction and increased ADHD symptoms in large-scale general population studies (Albaugh et al., 2017; Fuentes et al., 2012; Korponay et al., 2017). Interestingly, our age-stratified results suggest that the ADHD-differentiating deficit features more consistently in adulthood, when co-occurring addiction behavioral problems increase (e.g., Breyer et al., 2009; Ortal et al., 2015), more so in ADHD than in ASD (e.g., Sizoo et al., 2010; Solberg et al., 2019).

The increased left anterior and right posterior temporal GMV in ASD is a consistent VBM meta-analytic finding in ASD relative to TD controls (Carlisi et al., 2017; Cauda et al., 2011; DeRamus & Kana, 2015; Duerden, Mak-Fan, Taylor, & Roberts, 2012). Enhanced left anterior GMV was the only mega-analytic impairment finding in over 400 people with ASD but it did not survive in a smaller age- and sex-matched subgroups (Riddle, Cascio, & Woodward, 2017). Our meta-analysis may have increased statistical power to detect the differences. The ASD-differentiating increased left temporal GMV extends findings from a small VBM ASD/ADHD comparative study (Lim et al., 2015). The left temporal lobe plays roles in semantic and language processing (Binder et al., 2011), while the right posterior temporoparietal cortices plays a role in social interaction and mentalizing, i.e., the ability to attribute mental states in others (Krall et al., 2015). These structures are important for social cognition, which has been thought to be an ASD-specific impairment (Kana et al., 2015; Lombardo, Chakrabarti, Bullmore, & Baron-Cohen, 2011; White, Frith, Rellecke, Al-Noor, & Gilbert, 2014).

The ASD-differentiating increased right dlPFC GMV has not converged in smaller meta-analyses (Carlisi et al., 2017; DeRamus & Kana, 2015), possibly reflecting heterogeneity in findings. Right dlPFC is important for cognitive control, working memory and cognitive flexibility (Szczepanski & Knight, 2014). Neuronal and grey matter overgrowth have been reported in pediatric ASD in small ROI-based neuroimaging and post-mortem studies (Carper & Courchesne, 2005; Courchesne, Mouton, et al., 2011; Mitchell et al., 2009), but they have not been replicated in wider age range of ASD cases with cognitive flexibility deficits (Griebling et al., 2010).

The disorder-differentiating GMV *increase* in ASD and *decrease* in ADHD may possibly be related to the disorders' contrasting developmental trajectories (Courchesne, Campbell, et al., 2011; Shaw et al., 2007). However, while most of our ADHD-differentiating findings showed reduction in structure and function that may possibly reflect delayed maturation in

ADHD, the ASD-differentiating enhanced clusters were not found consistently in the pediatric sub-meta-analysis. Recent findings suggest that early brain overgrowth is not a defining characteristic of toddlers with high-risk for developing ASD (Hazlett et al., 2017), and may be specific to boys with regressive autism (Nordahl et al., 2011), who are underrepresented across studies. Thus, our findings are unlikely to reflect early brain overgrowth.

In the fMRI meta-analyses, ASD-differentiating impairments were predominant medial and left middle prefrontal underactivation and in bilateral ventrolateral prefrontal overactivation during cognitive control, while ADHD-differentiating underactivation emerged during motor inhibition in right inferior frontal and striatal regions. The ADHD-differentiating underactivation in right IFG and caudate, key regions implicated in inhibitory control (Rae, Hughes, Weaver, Anderson, & Rowe, 2014; Zhang, Geng, & Lee, 2017), are consistent with previous meta-analytic findings in ADHD during cognitive control (Hart et al., 2013; McCarthy et al., 2014; Norman et al., 2016) and extends the role of these regions, in particular right IFG, as disorder-specific neurofunctional biomarker of ADHD, as it has previously been observed relative to bipolar, obsessive compulsive and conduct disorders (Norman et al., 2016; Passarotti et al., 2010; Rubia, 2018; Rubia, Cubillo, et al., 2010; Rubia, Halari, et al., 2010; Rubia et al., 2009).

The mPFC underactivation in ASD is a consistent meta-analytic finding during cognitive control and related functions (Carlisi et al., 2017; Di Martino et al., 2009; Philip et al., 2012). The region is also implicated in functions such as sensory, emotion and social processing domains typically impaired in ASD (Kana, Patriquin, Black, Channell, & Wicker, 2016; Martinez-Sanchis, 2014; South & Rodgers, 2017). Intriguingly, the mPFC underactivation was ASD-differentiating relative to ADHD. While both medial and lateral PFC are activated during motor inhibition (Rae et al., 2014; Zhang et al., 2017), the activation of mPFC are more reliably tapped by cognitive interference tasks thus it may have closer associations to change or conflict detection (Aron, Herz, Brown, Forstmann, & Zaghloul, 2016). Whether our findings represent dissociated frontal brain functional impairment in ASD from ADHD in sub-constructs of cognitive control should thus be tested further meta-analytically when more fMRI findings are available.

While both disorders show functional abnormalities in prefrontal cortex in both hemispheres, the ASD-differentiating atypical activation was more predominantly on the left, in contrast to the right-lateralized ADHD-differentiating fronto-striatal underactivation, although atypical brain lateralization is not uncommon finding in ASD (Dichter, 2012; Floris & Howells, 2018; Kleinhans, Muller, Cohen, & Courchesne, 2008). Most importantly, comparisons between each disorder relative their respective TD controls show that neural impairment in ADHD is overwhelmingly characterized by fronto-striato-temporal underactivation, which is in line with previous findings of a developmental lag of cognitive control networks (Sripada et al., 2014), whereas the impairments in ASD implicate both under- and overactivation clusters in executive, attentional and, even, fronto-parieto-occipital visuo-perceptual brain regions. Of note, abnormally enhanced recruitment of posterior brain regions in ASD has been observed during working memory (Koshino et al., 2005; Vogan et al., 2014), psychomotor vigilance (Christakou et al., 2013; Murphy et al., 2014) and

temporal discounting (Chantiluke et al., 2014; Norman et al., 2017). This thus suggests a complex pattern of neurofunctional abnormality in ASD, with compromised cortical specialization, possibly reflecting both neural impairments inherent to ASD and, possibly, ensuing neuronal compensation (see review Floris & Howells, 2018).

The ASD-differentiating overactivation in PCC/precuneus, which was also unimpaired in ADHD relative to their respective controls, was unexpected, however. The overactivation in precuneus/PCC in ASD, which has been shown in a variety of tasks and resting-state condition (Christakou et al., 2013; Kennedy, Redcay, & Courchesne, 2006; Murdaugh et al., 2012; Spencer et al., 2012), presumably indicates DMN dysregulation. The lack of overactivation in PCC/precuneus in ADHD during cognitive control were apparent in previous meta-analyses (McCarthy et al., 2014; Norman et al., 2016), and could potentially be related to psychostimulant exposure which has shown to normalize DMN functioning (Liddle et al., 2011; Peterson et al., 2009; Rubia et al., 2014).

Finally, shared functional abnormality between disorders was found during motor response inhibition only in right AI, which is part of the salience network (Menon & Uddin, 2010). The limited shared abnormality may seem at odds with typical observation of psychiatric comorbidities in clinical settings, but individuals with ASD and ADHD are typically selectively recruited into imaging studies, excluding specific psychiatric comorbidities, which could have emphasized the differences between disorders, at the cost of their representativeness.

On the other hand, the findings of disorder differences are more likely to be underestimated. First, typically stringent statistical corrections applied by individual studies could have concealed true group differences in coordinate-based meta-analyses. The ADHD-differentiating findings, particularly, are likely underestimated by co-occurring ADHD in the ASD groups due to past preclusion of ADHD diagnosis in ASD (APA, 2000). Studies attempting to include more representative samples of the disorders may have included comorbid ASD and ADHD cases, perhaps at higher rates in ASD than in ADHD, as shown in prevalence studies in community representative samples (Gjevik, Eldevik, Fjæran-Granum, & Sponheim, 2011; Hollingdale, Woodhouse, Young, Fridman, & Mandy, 2019; Salazar et al., 2015; Simonoff et al., 2008). Long-term medication exposure is also likely to have masked the ADHD-differentiating findings.

Other limitations of the meta-analyses include the fewer number of participants with ASD relative to ADHD, which could have reduced power for detecting small ASD-differentiating impairments and increased the probability of false positive findings, particularly in the fMRI meta- and sub-meta-analyses. The representativeness of the meta-analytic findings may also be limited by the fact that many studies, particularly in ADHD, investigated males specifically. In ASD, the majority of studies have focused on adults and predominantly high-functioning individuals only (with few exceptions, i.e., Cai et al., 2018; Contarino et al., 2016; Retico et al., 2016; Riva et al., 2013; Wang et al., 2017; Q. Yang et al., 2018), even though individuals with learning disability made up a significant proportion of the ASD population (e.g., Charman et al., 2011). Furthermore, brain-behavior correlations could not be examined due to variations of disorder trait measures across studies. Finally, the influence

of task discrepancies across studies could not be fully assessed due to limited data for the non-motor cognitive control tasks.

5 Conclusions

These comparative meta-analyses of brain structure and function show predominantly disorder-differentiating abnormalities in the form of *decreased* GMV in vmOFC reward processing regions in ADHD and *increased* GMV in ASD in frontal and temporal cortices, part of the central executive and social cognition networks. Disorder-differentiating fMRI deficits were predominantly observed in ASD in medial frontal executive, ventrolateral prefrontal and DMN regions when including all cognitive control tasks; and in ADHD in inferior fronto-striatal regions during motor response inhibition only. Shared functional abnormality was limited to right AI during motor response inhibition. Therefore, people with ADHD and ASD appear to have mostly distinct brain structure and cognitive-control functional abnormalities. The findings contribute to the elucidation of the differential brain abnormalities in the two disorders, which is important for the understanding of their underlying pathophysiology and could ultimately aid in the development of future, disorder-differentiated behavioral, pharmacological or neurotherapeutic treatments.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgement

We thank all authors of the included studies who provided additional data for this meta-analysis. We also thank Toby Wise for insightful discussions on the statistical methods used in this meta-analysis.

Financial support

SL and LN were supported by Medical Research Council and Institute of Psychiatry, Psychology, and Neuroscience (IoPPN) PhD Excellence awards. CC is supported by a Wellcome Trust Sir Henry Wellcome Postdoctoral Fellowship (206459/Z/17/Z). JR receives support from the Instituto de Salud Carlos III and co-funded by European Union (ERDF/ESF, 'Investing in your future', Miguel Servet Research Contract CPII19/00009 and Research Project Grant PI19/00394). HH was supported by a grant from the Lila Weston Foundation for Medical Research and the Kids Company to KR. ES and KR receive research support from the National Institute of Health Research (NIHR) Biomedical Research Centre at South London and Maudsley Foundation Trust (IS-BRC-1215-20018). ES furthermore is supported by the NIHR through a program grant (RP-PG-1211-20016) and Senior Investigator Award (NF-SI-0514-10073), the European Union Innovative Medicines Initiative (EU-IMI 115300), Autistica (7237) Medical Research Council (MR/R000832/1, MR/P019293/1), the Economic and Social Research Council (ESRC 003041/1) and Guy's and St Thomas' Charitable Foundation (GSTT EF1150502) and the Maudsley Charity. KR and SL are currently supported by an MRC Grant MR/P012647/1 to KR. This study is supported by funding from the UK Department of Health via the National Institute for Health Research (NIHR) Biomedical Research Centre (BRC) for Mental Health at South London and the Maudsley National Health Service (NHS) Foundation Trust and the IoPPN, King's College London, which also support both senior authors, ES and KR. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

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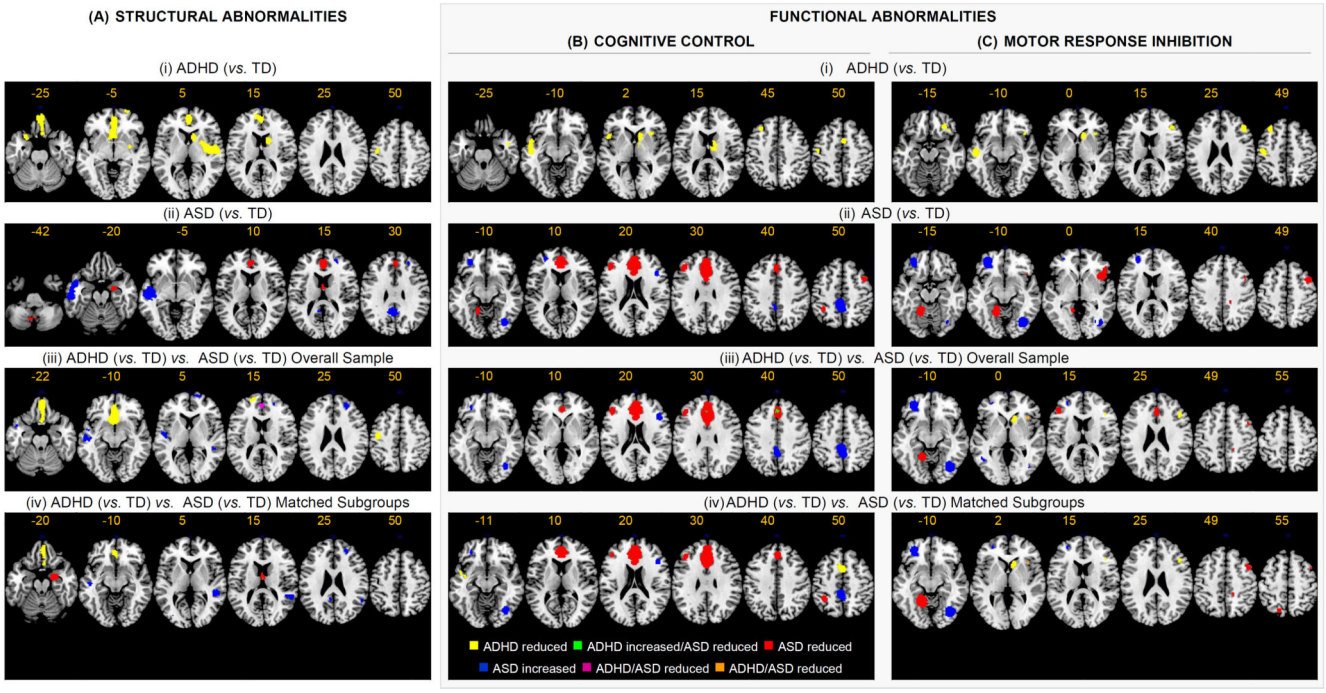

Fig. 1.

Brain abnormalities in the ADHD and ASD groups. Abnormalities in the (A) gray matter volume and brain activations during (B) cognitive control and (C) motor response inhibition. Rows (i) and (ii) show abnormalities in ADHD and ASD relative to typically developing (TD) controls. Abnormalities in ADHD versus ASD, each relative to TD controls are shown in (iii) overall samples and (iv) age-, sex-, and IQ-matched subgroups. In the overall sample, shared reduced rACC/mPFC GMV (MNI coordinates: 0,46,12; 119 voxels), and subthreshold overactivation in ADHD and underactivation in ASD in dmPFC (MNI coordinates: -2, 34, 36; 82 voxels) were observed but did not survive sensitivity analysis. A statistical threshold of $p < .005$ with a cluster extent of 20 voxels were used in all analyses.

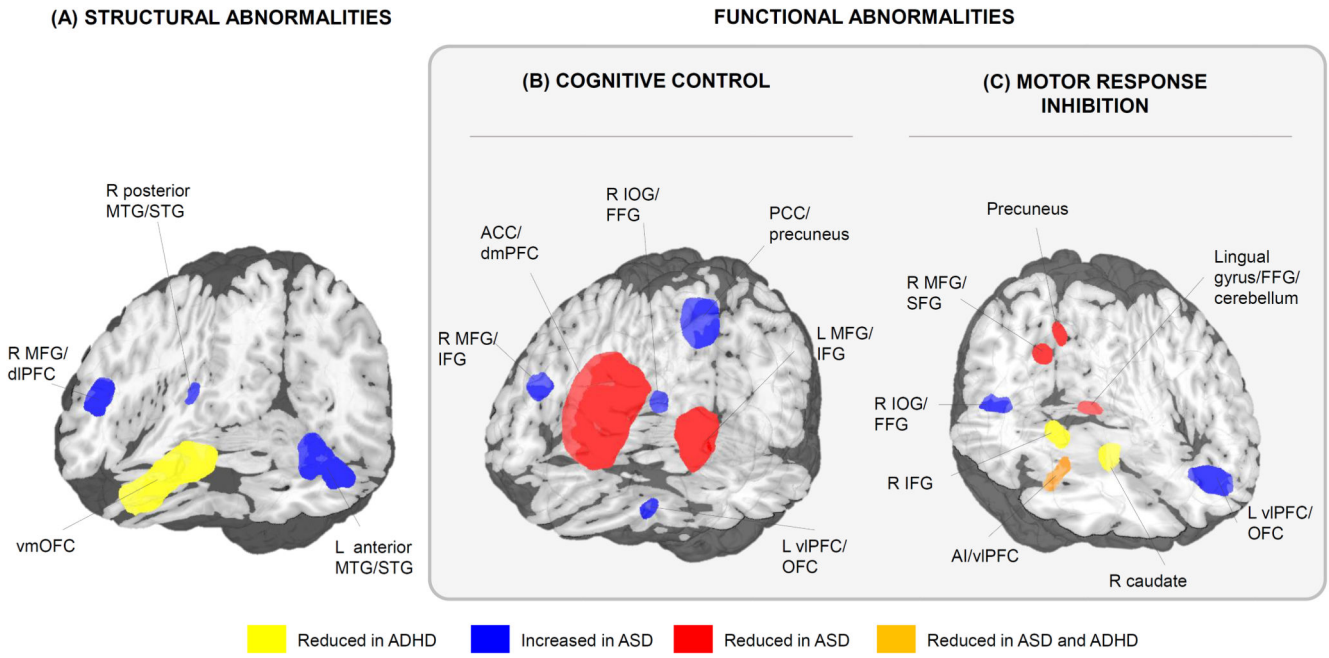

Fig. 2.

Summary of consistent disorder-differentiating and shared brain abnormalities in ADHD and ASD. Findings of (A) gray matter volume abnormalities and brain activation abnormalities during (B) cognitive control and (C) motor response inhibition. A statistical threshold of $p < .005$ with a cluster extent of 20 voxels were used in all analyses.

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Table 1

Sample Characteristics and Summary Findings of VBM and fMRI Cognitive Control Studies in ADHD

Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
(A) VBM studies in ADHD									
Ahrendts et al. (2011)	Adult	--	31 (65)	31.2	n/a	31 (65)	31.5	n/a	ADHD<TD: L/R occipital lobe
Montes et al. (2010)	Adult	--	20 (50)	29.0	102.9	20 (50)	27.6	100.2	ADHD<TD: R caudate
Amico, Stauber, Koutsouleris, and Frodl (2011)	Adult	--	20 (75)	33.6	n/a	20 (75)	34.7	n/a	--
Bonath, Tegelbeckers, Wilke, Flechtner, and Krauel (2016)	Pediat.	--	18 (100)	13.6	106.8	18 (100)	14.1	108.1	ADHD<TD: L/R cerebellum, occipital cortex, L/R hippocampus/ amygdala complex, L ACC
Bralten et al. (2016)	Mixed	--	307 (68)	17.1	97.1	196 (49)	16.7	106.6	ADHD<TD: L pre-CG, L OFC, R frontal pole, L paracingulate/ cingulate cortex/frontal pole, bilateral medial FG/ paracingulate/ cingulate/ subcallosal cortices
Brieber et al. (2007)	Pediat.	--	15 (100)	13.1	104.1	15 (100)	13.3	107.7	ADHD<TD: L/R caudate nucleus, L/R hippocampus, R MFG, L insula, L STG, L MOG; ADHD>TD: L/R SPL, post-CG, L middle cingulate, precuneus, L IPL
Carmona et al. (2005)	Pediat.	--	25 (84)	10.8	n/a	25 (84)	11.2	n/a	ADHD<TD: L pre-/post-CG, paracentral lobule, R rectal gyrus, R inferior and superior orbital frontal, L/R cerebellum
Depue, Burgess, Bidwell, Willcutt, and Banich (2010)	Adult	--	31 (61)	20.0	114.2	21 (65)	19.3	112.6	--
Gehricke et al. (2017)	Adult	--	32 (81)	25.3	n/a	40 (83)	23.9	n/a	ADHD<TD: R M/IFG, R S/MTG, L caudate head, L PHG
He et al. (2015)	Pediat.	--	37 (100)	9.9	n/a	35 (100)	10.7	n/a	ADHD<TD: R OFC, R PMC, L PCC, L posterior midcingulate cortex
Iannaccone, Hauser, Ball, et al. (2015)	Pediat.	--	18 (50)	14.5	114.5	18 (61)	14.8	108.5	ADHD<TD: R medial SFG/ACC/SFG, L SMA/cingulate gyrus, L cerebellum; ADHD>TD: R pre- and post-CG
Jagger-Rickels, Kibby, and Constance (2018)	Pediat.	--	41 (44)	9.6	n/a	32 (56)	9.7	n/a	ADHD<TD: L MTG/STS, R STS L/R caudate/ putamen, L/R SFG, L/R insula, L/R thalamus, R medial OFC, L MFG, L/R occipital lobe, L medial SFG, L pre-CG, R post cingulate/ anterior lingual, R calcarine/cuneus, L supramarginal gyrus
Johnston et al. (2014)	Pediat.	--	34 (100)	12.5	99.8	34 (100)	13.2	103.7	ADHD<TD: Brainstem, putamen
Kappel et al. (2015)	Pediat.	--	14 (71)	9.8	104.6	10 (80)	11.0	111.9	ADHD<TD: R STG, R Heschl's gyrus, R rolandic operculum; ADHD>TD: L paracentral lobule, L/R middle orbital gyrus, R FFG, L rectal gyrus
	Adult	--	16 (94)	23.5	97.8	20 (100)	23.7	108.4	ADHD<TD: R supramarginal gyrus, R precuneus, L OFG, R hippocampus, L rectal gyrus
Kaya et al. (2018)	Pediat.	--	19 (74)	10.3	113.5	18 (67)	10.2	119.7	ADHD>TD: L/R SFG, R MFG, L/R SMA, L/R pre-CG, L post-CG, R MOG, L cuneus

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Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
Kobel et al. (2010)	Pediat.	--	14 (100)	10.4	n/a	12 (100)	10.9	n/a	ADHD<TD: R S/MTG
Kumar, Arya, and Agarwal (2017)	Pediat.	--	18 (100)	9.6	92.1	18 (100)	9.7	109.7	ADHD<TD: L OFC, L MFG/ dlPFC, L MTG, L cerebellum
Li et al. (2015)	Pediat.	--	30 (100)	10.3	121.7	30 (100)	10.3	107.1	ADHD<TD: R insula, R OFC
Lim et al. (2015)	Pediat.	--	44 (100)	13.6	92.2	33 (100)	14.3	110	ADHD<TD: L/R cerebellum, L IPL, pre-/post-CG
Maier et al. (2015)	Adult	--	131 (48)	34.5	113.1	95 (47)	37.7	121	--
McAlonan et al. (2007)	Pediat.	--	28 (100)	9.9	109.9	31 (100)	9.6	116.5	ADHD<TD: R MFG, L/R SFG/IFG, R globus pallidus/putamen/ thalamus, precuneus, L IPL, L SOG, R cerebellum vermis
Moreno-Alcázar et al. (2016)	Adult	--	44 (66)	31.6	105.0	44 (66)	32.6	106.0	ADHD<TD: R SMA/SFG, subgenual ACC, R DLPFC
Onnink et al. (2014)	Adult	--	119 (39)	36.3	107.5	107 (42)	36.9	110.2	--
Overmeyer et al. (2001)	Pediat.	--	18 (83)	10.4	99	16 (94)	10.3	n/a	ADHD<TD: R PCC, R SFG, L/R putamen/globus pallidus
Ramesh and Rai (2013)	Pediat.	--	15 (26)	16.8	n/a	15 (26)	16.7	n/a	ADHD<TD: L cingulate gyrus, R mid-cingulate gyrus, L SFG, L/R medial SFG, L temporal lobe, L MOG, L cuneus, L STG, L supramarginal gyrus
Roman-Urrestarazu et al. (2016)	Adult	--	49 (76)	22.2	96.6	34 (50)	22.9	112.2	ADHD<TD: L/R caudate
Saad et al. (2017)	Pediat.	--	16 (75)	12.8	n/a	28 (68)	13.1	n/a	--
	Pediat.	--	18 (72)	13.7	n/a	28 (68)	13.1	n/a	--
Sasayama et al. (2010)	Pediat.	--	18 (72)	10.6	90	17 (71)	10.0	n/a	ADHD<TD: L/R amygdala/temporal pole/OFC, L/R occipital cortex, R STS, L parietal cortex, L MFG, L temporal pole, R rectal gyrus, L PHG
Seidman et al. (2011)	Adult	--	74 (51)	37.3	116	54 (46)	34.3	115.8	--
Sethi et al. (2017)	Adult	--	30 (63)	33.7	109.0	30 (63)	32.6	110.1	ADHD<TD: R IPC
Shimada et al. (2015)	Pediat.	--	17 (88)	10.3	95.3	15 (73)	12.8	104.1	ADHD<TD: L putamen
Stevens and Haney-Caron (2012)	Pediat.	--	24 (67)	15.7	n/a	24 (70)	16.0	n/a	--
van Wingen et al. (2013)	Adult	--	14 (100)	32.0	104	15 (100)	37.0	99	ADHD<TD: R putamen, R cerebellum; ADHD>TD: L/R midbrain, R pre-CG
Vilgis, Sun, Chen, Silk, and Vance (2016)	Pediat.	--	48 (100)	12.6	92.2	31 (100)	12.8	109.6	ADHD<TD: R SPL, L IPL, L/R S/MFG, medial FG, R MTG
Villemonteix et al. (2015)	Pediat.	--	33 (55)	10.3	105.6	24 (50)	10.0	109.7	ADHD<TD: R insula, R MTG,
	Pediat.	--	20 (80)	10.4	107.4	24 (50)	10.0	109.7	ADHD<TD: R MFG, R pre-CG
Wang, Jiang, Cao, and Wang (2007)	Pediat.	--	12 (100)	13.4	n/a	12 (100)	13.5	n/a	ADHD<TD: L SPL, R MFG, R MTG, R BG; ADHD>TD: R occipital lobe

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Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
P. Yang et al. (2008)	Pediat.	--	57 (61)	11.1	97.9	57 (60)	11.7	n/a	ADHD<TD: L/R caudate, L/R cerebellum, R calcarine sulcus, L cuneus
(B) fMRI cognitive control studies in ADHD									
Banich et al. (2009)	Adult	Stroop	23 (61)	20.0	116	23 (57)	19.0	113	ADHD>TD: R MFG
Bhaijiwala, Chevrier, and Schachar (2014)	Pediat.	Stop	12 (58)	13.8	n/a	12 (50)	15.4	n/a	ADHD<TD: R MFG, R MTG, L cerebellum; ADHD>TD: R MFG/SFG
Booth et al. (2005)	Pediat.	GNG	12 (67)	11.0	n/a	12 (58)	11.7	n/a	ADHD<TD: L/R pre-CG, L/R caudate, R caudate head, R IFG, L/R thalamus.
Carmona et al. (2012)	Adult	GNG	19 (100)	33.6	110.9	19 (100)	29.4	111.7	ADHD<TD: R STG
Chantiluke, Barrett, Giampietro, Santosh, et al. (2015)	Pediat.	Stop	18 (100)	14.3	95	25 (100)	13.4	109	ADHD<TD:L OFC/STL/putamen/GP, L IPL
Chen et al. (2015)	Adult	GNG	29 (100)	24.9	n/a	25 (100)	25.6	n/a	--
Chou, Chia, Shang, and Gau (2015)	Pediat.	Stroop	42 (81)	10.5	108.5	20 (80)	12.0	106.5	ADHD<TD: R MFG/IFG (atomoxetine group pre-treatment), L IFG, L SPL, R medial FG (methylphenidate group pre-treatment); ADHD>TD: L/R SPL, pre-CG, L dlPFC, L dACC (atomoxetine group pre-treatment), post-CG (methylphenidate group pre-treatment)
Congdon et al. (2010)	Adult	Stop	35 (54)	30.9	n/a	62 (45)	30.8	n/a	--
Cubillo et al. (2010) [a]	Adult	Stop, Switch	11 (100)	29.0	92	14 (100)	28.0	106	ADHD<TD: L IFC/AI/PMC, R IFC/AI/caudate/thalamus/ putamen/PMC, L/R SMA/ACC (stop), R IFG/insula/caudate/ putamen L IFG/insula/ putamen/pre-/post-CG/inferior parietal (switch)
Cubillo et al. (2014)	Pediat.	Stop	19 (100)	13.1	92	29 (100)	13.8	110	ADHD<TD: L/R IFG, L M/ITG/parietal gyri, R cerebellum/ FFG; ADHD>TD: L cerebellum, L/R PCC/occipital gyri, R STG/post-CG/posterior insula/ putamen
Cubillo, Halari, Giampietro, Taylor, and Rubia (2011) [a]	Adult	Simon	11 (100)	29.0	92	15 (100)	28.0	112	ADHD<TD: L OFC/IFC/mPFC/ACC/ caudate/PMC
Dibbets, Evers, Hurks, Marchetta, and Jolles (2009) [b]	Adult	GNG	16 (100)	28.9	n/a	13 (100)	28.1	n/a	--
Dibbets, Evers, Hurks, Bakker, and Jolles (2010) [b]	Adult	Switch	15 (100)	28.8	n/a	14 (100)	28.6	n/a	ADHD<TD: R putamen, R dorsal posterior CG, L mPFC, R thalamus, L OFC/ claustrum/post-CG; ADHD>TD: R MTG, R dACC, R precuneus, R lingual gyrus, L pre-CG/SMA, L insula
Durston, Mulder, Casey, Ziermans, and van Engeland (2006)	Pediat.	GNG	11 (100)	14.0	100	11 (100)	15.3	106	--
L. Y. Fan, Chou, and Gau (2017) [c]	Adult.	Stroop	12 (42)	28.9	115.8	12 (42)	30.3	118.3	ADHD>TD: R IFG, RACC
	Adult	Stroop	12 (42)	32.5	119.9	12 (42)	30.3	118.3	ADHD>TD: R IFG

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Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
L. Y. Fan, Shang, Tseng, Gau, and Chou (2018)	Pediat.	Stroop	27 (89)	12.1	105.2	27 (78)	11.8	110.4	ADHD < TD: R MFG, R post-CG, R SPL
Hwang et al. (2015)	Pediat.	Stroop	26 (65)	13.9	106.4	35 (51)	14.5	105.1	ADHD < TD: Medial FG, cerebellar tonsil
Iannaccone, Hauser, Staempfli, et al. (2015)	Pediat.	GNG/ Flanker	18 (50)	14.5	108.5	18 (61)	14.8	114.4	ADHD<TD: R MFG/dlPFC/SFG; ADHD>TD: L/R MTG/STG, L insula/putamen/pre-CG, L ITG/FFG/PHG/MTG, L pons, L amygdala/ hippocampus, L FFG/ lingual gyrus, L post-CG/IPG, R brainstem/ pons/cerebellum
Janssen, Heslenfeld, Mourik, Logan, and Oosterlaan (2015)	Pediat.	Stop	21 (90)	10.6	98.6	17 (76)	10.3	108.7	ADHD<TD: L/R mPFC, R MFG, R IFG, R pre-CG, R ACC, L SFG; ADHD>TD: L/R ACC, L/R MFG, R mPFC, R PCC, R cuneus, L IFG, L post-CG, L pre-CG.
Konrad, Neufang, Hanisch, Fink, and Herpertz-Dahlmann (2006)	Pediat.	Flanker	16 (100)	10.2	103	16 (100)	10.3	105	ADHD<TD: L pre-CG, R putamen; ADHD>TD: L medial SPL
Kooistra et al. (2010)	Adult	GNG	11 (100)	21.5	110	11 (100)	10.1	125	ADHD>TD: L/R ACC, R supramarginal gyrus, L angular gyrus, L/R supramarginal gyrus, R occipital gyrus, L frontal pole, L/R MFG, L/R IFG, L SFG, paracingulate gyrus, medial thalamus, L caudate nucleus, L posterior putamen, L lateral occipital gyrus, L/R pre-CG, R post-CG, R mPFC, L PCC, L STG
J. Ma et al. (2012)	Pediat.	GNG	15 (53)	9.8	100.2	15 (53)	22.3	102.6	ADHD>TD: R ITF, R midbrain, R pre-CG, R calcarine, R IOG, R MOG, L/R cerebellum, L post-CG, L IFG, R hippocampus
I. Ma et al. (2016)	Pediat.	Stroop	25 (76)	15.4	98.3	33 (67)	15.3	108.9	--
Massat et al. (2018)	Pediat.	Stop	18 (44)	10.0	106.8	19 (47)	10.6	114.4	ADHD>TD: L caudate body, L ventral putamen R dorsal putamen, R insula, L caudate tail, R middle CC, R ACC
Passarotti et al. (2010)	Pediat.	Stop	11 (55)	13.1	107.6	15 (48)	9.9	101.2	ADHD<TD: R MFG, R mPFC, L/R I/SFG, L STG; ADHD>TD: R caudate tail, L caudate, L anterior cerebellar vermis
Peterson et al. (2009)	Pediat.	Stroop	16 (81)	14.1	101.2	20 (60)	14.1	118.5	ADHD<TD: L ACC, insula R, precuneus, thalamus, caudate; ADHD>TD: R SFG, hippocampus, L ACC
Rasmussen et al. (2016)	Adult	GNG	50 (82)	24.8	102.1	23 (70)	24.1	109.2	ADHD<TD: R supramarginal gyrus, superior parietal, angular gyrus, L/R MFG. R frontal pole, L/R SPL, L/R IFG, L/R pre-/ post-CG, R caudate/accumbens, L/R precuneus, R OFC, R posterior cingulate gyrus, R insula, R thalamus
Rubia, Smith, Brammer, Toone, and Taylor (2005)	Pediat.	Stop	16 (100)	13.2	100	21 (100)	13.4	95	ADHD<TD: R IFC, OFC, pre-CG, STL
Rubia, Halari, Cubillo, et al. (2011) [d]	Pediat.	Simon	12 (100)	13.0	90	13 (100)	14.0	102	ADHD<TD: R IFC/inferior parietal, L vmPFC/BG/thalamus, R SMA/ACC/PCC/SPL, L STL/MTL/occipital
Rubia, Halari, Mohammad, Taylor, and Brammer (2011) [d]	Pediat.	Stop	12 (100)	13.0	90	13 (100)	13.0	102	ADHD<TD: B IFC, insula, ACC, pre-SMA; thalamus; R MTL, occipital, IPL, precuneus, PCC, cerebellum

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Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
Schulz et al. (2004)	Pediat.	GNG	10 (100)	17.9	88.4	9 (100)	17.5	91.9	ADHD<TD: R pre-CG, R ITG, L hippocampus, R lingual gyrus, L/R cerebellum; ADHD>TD: L/R MFG, L/R IFG, L medial FG, L ACC, L/R IPL, R precuneus
Schulz et al. (2014)	Adult	GNG	14 (100)	23.3	n/a	14 (100)	22.8	n/a	--
Schulz et al. (2017)	Adult	Stroop	27 (89)	24.2	n/a	28 (86)	24.6	n/a	ADHD<TD: L/R cerebellum, L MTG, R lingual gyrus, R IFG, R MTG, R OFG, R SOG, R precuneus, R IPL. R MOG
Sebastian et al. (2012) [e]	Adult	GNG Stroop Stop	20 (55)	33.3	115.3	24 (46)	17.5	115.7	ADHD<TD: R caudate (GNG), R GP (Stop), R post-CG, L paracentral lobule/mid-CC, L STG/MTG, R temporal pole, R GP (Stroop)
Shang, Sheng, Yang, Chou, and Gau (2018) [f]	Adult	Stroop	25 (48)	29.1	112.8	30 (50)	28.2	115.4	ADHD<TD: L IFG, L/R caudate nucleus
	Adult	Stroop	25 (56)	28.5	113.1	30 (50)	28.2	115.4	ADHD<TD: L IFG, L caudate nucleus, L SPL
Siniatchkin et al. (2012)	Pediat.	GNG	12 (83)	9.3	n/a	12 (75)	30.3	n/a	ADHD<TD: ACC, R dlPFC, R caudate nucleus
Smith, Taylor, Brammer, Toone, and Rubia (2006) [g]	Pediat.	GNG	17(100)	12.8	n/a	18 (100)	9.3	n/a	ADHD<TD: L MFG
		Stroop	19 (100)	12.9	n/a	24 (100)	12.8	n/a	--
		Switch	14 (100)	13.3	n/a	27 (100)	12.9	n/a	ADHD<TD: L STG/IFG/pre-CG/insula, R MTG/IFG, IPL/STG
Spinelli et al. (2011)	Pediat.	GNG	13 (69)	10.6	109.2	17 (47)	13.3	108.8	ADHD>TD: R pre-CG, MFG
Szekely et al. (2018)	Adult	Stop	64 (56)	24.0	n/a	84 (57)	24.5	n/a	--
Tamm, Menon, Ringel, and Reiss (2004)	Pediat.	GNG	10 (100)	16.0	109.2	12 (100)	10.6	111.6	ADHD<TD: R ACC/SMA/ MFG; ADHD>TD: L M/I/STG
Thornton, Bray, Langevin, and Dewey (2018)	Pediat.	GNG	20 (90)	12.4	109.4	20 (40)	10.6	112.6	--
van Hulst, de Zeeuw, Rijks, Neggers, and Durston (2017)	Pediat.	GNG	24 (100)	11.2	105.6	26 (100)	10.5	117.3	--
van Rooij et al. (2015)	Pediat.	Stop	108 (64)	15.1	92.7	77 (49)	16.0	109.2	ADHD<TD: L STG, pre-CG; ADHD>TD: L SMA
	Adult	Stop	77 (78)	20.3	99.1	45 (33)	14.6	106.4	ADHD<TD: L SFG, R hippocampal gyrus
Zamorano et al. (2017)	Pediat.	Stroop	17 (100)	11.6	104.2	17 (100)	11.7	109.8	ADHD>TD: R medial/IFG

Abbreviations. N=sample size, y=year, pediat=pediatric (child/adolescent) sample, ADHD=attentional-deficit/hyperactivity disorder, TD=typically developing controls, L/R=Left/Right, GNG=Go/No-Go. Brain regions (in alphabetical order): ACC=anterior cingulate cortex, AI=anterior insula, BG=basal ganglia, CC=cingulate cortex, dlPFC=dorsolateral prefrontal cortex, dACC=dorsal ACC, FFG=fusiform gyrus, GP=globus pallidus, I/M/SFG=inferior/middle/superior frontal gyrus, I/SPL=inferior/ superior parietal lobe, medial FG=medial frontal gyrus, mPFC=medial prefrontal cortex, M/STG=middle/ superior temporal gyrus, OFC=orbitofrontal cortex, OFG=orbitofrontal gyrus, PCC=posterior cingulate cortex, PHG=parahippocampal gyrus, PMC=premotor cortex, pre-/post-CG=pre-/post-central gyrus, SMA=supplementary motor cortex, STL=superior temporal lobe, STS=superior temporal sulcus, vmPFC=ventromedial prefrontal cortex, a-g datasets were combined, adjusting their variance according to the method outlined in Norman et al. (2016). See Supplement Table S2a for further information.

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Table 2

Sample Characteristics and Summary Findings of VBM and fMRI Cognitive Control Studies in ASD

Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
(A) VBM studies in ASD									
Abell et al. (1999)	Adult	--	15 (80)	28.8	n/a	15 (80)	25.3	n/a	ASD<TD: R paracingulate, L IFG, L occipito-temporal junction; ASD>TD: L amygdala, L/R anterior cerebellum, L MTG, R ITG
Boddaert et al. (2004)	Pediat.	--	21 (76)	9.3	n/a	12 (58)	10.8	n/a	ASD<TD: L/R STS
Bonilha et al. (2008)	Pediat.	--	12 (100)	12.4	n/a	16 (100)	13.2	n/a	ASD>TD: L/R IFG, L/R cuneus, L/R ACC, R PCC, claustrum, L/R precuneus, L/R STG, L/R MTG, L ITG, thalamus (pulvinar), L/R SFG, L/R SPL, L insula, L putamen, R caudate, L/R FFG, L/R occipital gyrus, L lingual gyrus, L pre-CG, L post-CG, L/R thalamus, L/R PHG, L/R mPFC, R IPL
Brieber et al. (2007)	Pediat.	--	15 (100)	14.2	106.8	15 (100)	13.3	107.7	ASD<TD: L/R ITG/hippocampus/amygdala, L MOG, L PMC, L hippocampus; ASD>TD: R supramarginal gyrus, L post-CG
Cai et al. (2018)	Pediat.	--	38 (84)	9.6	75.8	27 (96)	8.3	98.6	ASD<TD: R cerebellum anterior lobe, R precuneus, R angular gyrus; ASD>TD: L ITG, L/R MTG
Cheng, Chou, Fan, and Lin (2011)	Pediat.	--	25 (100)	13.7	101.6	25 (100)	13.5	109.0	ASD<TD: R IFG, R pre-CG, L post-CG, cuneus, thalamus, lingual gyrus, STG; ASD>TD: ACC, paracentral lobule, SPL, precuneus, MFG, FFG, subcallosal gyrus
Contarino, Bulgheroni, Annunziata, Erbetta, and Riva (2016)	Pediat.	--	25 (88)	6.1	56	25 (65)	6.1	103	--
M. Craig et al. (2007)	Adult	--	14 (0)	37.9	103.4	19 (0)	35.0	111.2	ASD<TD: Cuneus, L ITG/STG, R MTG, R ACC
D'Mello, Crocetti, Mostofsky, and Stoodley (2015)	Pediat.	--	35 (86)	10.4	n/a	35 (60)	10.4	n/a	ASD<TD: R lingual gyrus, R cerebellum, R angular gyrus; ASD>TD: L PCC/precuneus, R SFG, L MOG
Ecker et al. (2012)	Adult	--	89 (100)	27.0	110	89 (100)	28.0	113.0	ASD<TD: R occipital/ITG/MTG, R cerebellum, R posterior FFG, R lingual gyrus, L/R inferior and R SOG, R cuneus, precuneus, R PCC; ASD>TD: L /R ITG/MTG/STG/ L/R FFG/PHG/insula, L IFG, L putamen/caudate, L thalamus, L/R dlPFC/ MFG/pre-/post-CG, R IPL
Foster et al. (2015)	Pediat.	--	38 (100)	12.4	102.5	46 (100)	12.6	113.1	ASD<TD: R STG, L/R supramarginal gyrus, L cerebellum; ASD>TD: R central sulcus, L medial FG, L/R IFG, L/R pre-CG, L MFG, L pre-SMA, R SFG, L ACC, L OFC, L ITG/STG. L/R MTG, L Heschl's gyrus, R lingual gyrus, L FFG, L post-CG, L PCC, L precuneus, R supramarginal/angular gyrus, L IOG, L/R cuneus, L putamen, L caudate
Freitag et al. (2008)	Mixed	--	15 (87)	17.5	101.2	15 (87)	18.6	112.1	ASD<TD: R intraparietal sulcus
Greimel et al. (2013)	Mixed	--	47 (100)	18.3	107.5	51 (100)	21.4	112.5	ASD<TD: ACC, L/R posterior STS, R MTG

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Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
Groen, Buitelaar, van der Gaag, and Zwiers (2011)	Pediat.	--	17 (82)	14.4	98.0	25 (88)	15.5	105.0	--
Hyde, Samson, Evans, and Mottron (2010)	Mixed	--	15 (100)	22.7	100.4	15 (100)	19.2	106.6	ASD<TD: R post-CG, L/R pre-CG; ASD>TD: Brainstem/ midbrain, reticular, medial FG/OFC, L/R MFG
Itahashi et al. (2015)	Adult	--	46 (100)	30.2	106.0	46 (100)	30.5	109.2	--
Katz et al. (2016)	Adult	--	23 (100)	26.6	n/a	32 (100)	29.8	n/a	ASD>TD: L/R OFC, ACC
Kaufmann et al. (2013)	Pediat.	--	10 (80)	14.7	102.3	10 (80)	13.8	109.5	ASD<TD: Lateral portion of R precuneus; ASD>TD: L medial FG, R precuneus
Ke et al. (2008)	Pediat.	--	17 (82)	8.9	108.8	15 (80)	9.7	109.8	ASD<TD: R PHG; ASD>TD: L/R supramarginal gyrus, post-CG, R MFG, R cerebellum
Kosaka et al. (2010)	Adult	--	32 (100)	23.8	101.6	40 (100)	22.5	109.7	ASD<TD: R insula, R IFG, R IPL
Kurth et al. (2011)	Pediat.	--	52 (73)	11.2	102.2	52 (73)	11.1	106.0	ASD<TD: Hypothalamus
Kwon, Ow, Pedatella, Lotspeich, and Reiss (2004)	Pediat.	--	20 (100)	13.5	n/a	13 (100)	13.6	n/a	ASD<TD: R ITG, R entorhinal cortex, R FFG
Langen et al. (2009)	Pediat.	--	99 (92)	12.9	107.6	89 (92)	12.4	110.0	--
Lim et al. (2015)	Pediat.	--	19 (100)	14.9	113.0	33 (100)	14.9	110.0	ASD>TD: L MTG/STG, L medial FG
Lin, Ni, Lai, Tseng, and Gau (2015)	Pediat.	--	28 (100)	10.7	106.9	43 (100)	10.6	115.2	ASD<TD: R post-CG, precuneus, L MOG; ASD>TD: L subcallosal gyrus, L/R sublobar areas
	Pediat.	--	40 (100)	14.7	101.5	18 (100)	15.5	108.7	--
	Adult	--	18 (100)	22.2	99.6	29 (100)	23.4	116.8	ASD>TD: L/R SFG, L MFG
Lin, Tseng, Lai, Chang, and Gau (2017)	Pediat.	--	20 (100)	13.5	103.8	54 (100)	12.8	112.5	ASD>TD: L cerebellum crus I
McAlonan et al. (2002)	Adult	--	17 (90)	32.0	96.0	24 (92)	33.0	114.0	ASD<TD: R cerebellum, L/R lenticular nucleus, R CG, L/R MFG, R SFG, precuneus
McAlonan et al. (2008)	Pediat.	--	33 (82)	11.6	113.2	55 (86)	10.7	117.1	ASD<TD: L dlPFC, L/R BG, L/R inferior cerebellar vermis, L STS, posterior parietal cortices
Mengotti et al. (2011)	Pediat.	--	20 (90)	7.0	n/a	22 (91)	7.7	n/a	ASD<TD: L SMA, R IFG; ASD>TD: R IPL, L/R ITG, L SPL, R SOG, precuneus
Mueller et al. (2013)	Adult	--	12 (75)	35.5	111.3	12 (67)	33.3	110.8	ASD<TD: L/R TPJ, L/R temporal lobe, L/R IFG, L MFG, L frontal pole, L/R mPFC, paracingulate gyrus
Ni et al. (2018)	Pediat.	--	81 (100)	12.6	107.4	61 (100)	12.4	112.0	ASD<TD: L lateral occipital/superior parietal cortex, L frontal pole
Pereira et al. (2018)	Mixed		19 (82)	17.4	99.8	25 (66)	18.5	105.8	ASD<TD: L/R cerebellum, posterior lobe, R cerebellum anterior lobe, L FFG, L/R CG, L paracentral lobule, R MFG, L claustrum,

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Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
									R medial FG, L PHG L amygdala, L post-CG, R SFG; ASD>TD: L/R brainstem, R cerebellum, posterior lobe
Poulin-Lord et al. (2014)	Mixed	--	23 (87)	19.8	100.3	22 (86)	22.6	107.3	--
Poustka et al. (2012)	Pediat.	--	18 (89)	9.7	111.0	18 (89)	9.7	112.8	--
Radeloff et al. (2014)	Mixed	--	34 (91)	19.1	105.7	26 (85)	19.5	107.8	--
Retico et al. (2016)	Pediat.	--	76 (50)	4.6	71	76 (50)	4.6	73	ASD<TD: L posterior MTG, L cerebellum, L/R temporal pole; ASD>TD: L precuneus, L PCC, R posterior STG, R MTG/ITG, R mPFC, L SPL, mid-CC
Riedel et al. (2014)	Adult	--	30 (63)	35.4	124.5	30 (63)	35.5	123.6	--
Riva et al. (2013)	Pediat.	--	26 (89)	5.8	51.6	21 (62)	6.8	n/a	ASD<TD: L/R cerebellum crus II, vermis 8/9, L/R hippocampus, L IFG, L MOG/SOG, L ITG, L SFG. R IOG, R post-CG
Rojas et al. (2006)	Mixed	--	24 (100)	20.8	94.8	23 (100)	21.4	118.7	--
Sato et al. (2017)	Adult	--	36 (69)	27.0	110.4	36 (69)	24.9	n/a	ASD<TD: R IOG/ITG/MTG, L FFG, L/R amygdala, R PHG, R L/ MFG, R SMA, L/R CG, ACC, L/R medial SFG, R OFG
Schmitz et al. (2006)	Adult	--	10 (100)	38.0	105.0	12 (100)	39.0	106	ASD>TD: L IFG, ACC, R SFG, L/R MFG
Toal et al. (2010)	Adult	--	65 (88)	31.0	98.0	33 (91)	32.0	105	ASD<TD: R cerebellum/PHG/ FFG, R ITG/STG L PHG/STG/TG/FFG/ cerebellum
Waiter et al. (2004)	Pediat.	--	16 (100)	15.4	100.4	16 (100)	15.5	99.7	ASD<TD: R thalamus; ASD>TD: L SFG, R FFG, R mPFC, L MTG, R PCC, L/R STG, L lingual gyrus, L IFG, L MFG, L IOG, L PHG
Wang et al. (2017)	Pediat.	--	31 (100)	4.8	62.5	31 (100)	4.8	97.1	ASD>TD: L STG, L post-CG
Wilson, Tregellas, Hagerman, Rogers, and Rojas (2009)	Adult	--	10 (80)	30.1	91.5	10 (70)	29.4	127.2	--
Q. Yang et al. (2018)	Pediat.	--	16 (63)	10.4	45.3	16 (63)	10.5	97.5	ASD<TD: L cerebellum, L caudate
(B) fMRI studies in ASD									
Ambrosino et al. (2014)	Pediat.	GNG	19 (100)	11.5	112	19 (100)	11.1	120	--
Chantiluke, Barrett, Giampietro, Santosh, et al. (2015)	Pediat.	Stop	19 (100)	14.7	112	25 (100)	13.4	109	ASD<TD: L IPL; ASD>TD: R IFC, L IFG/MFG
Daly et al. (2014)	Adult	GNG	14 (100)	31	115	14 (100)	31	123	ASD<TD: R IFG, L thalamus; ASD>TD: R caudate, R cerebellum
Denisova et al. (2017)	Mixed	Simon	20 (95)	22.7	n/a	20 (95)	23.2	n/a	ASD>TD: R lingual gyrus, R cerebellum, R ITG
Duerden et al. (2013)	Adult	GNG	16 (69)	27.2	112	17 (71)	30.7	114	ASD<TD: R MFG; ASD>TD: R IFG, R FFG
J. Fan et al. (2012)	Adult	Flanker	12 (75)	30	115	12 (83)	28	120	ASD<TD: ACC
Gooskens et al. (2018)	Pediat.	Stop	26 (35)	11.3	108.9	53 (45)	10.8	111.9	--

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Source	Age group	Task	Patients			Controls			Summary findings
			N (% male)	Age, y	IQ	N (% male)	Age, y	IQ	
Kana, Keller, Minshew, and Just (2007)	Adult	GNG	12 (92)	26.8	110	12 (92)	22.5	117	ASD<TD: L ITG, R PHG, R calcarine sulcus, R PMC, R mid-CC, L/R post-CG, R insula/IFG, L lingual gyrus
Kennedy et al. (2006)	Adult	Stroop	15 (100)	25.5	96	14 (100)	26.1	n/a	ASD>TD: mPFC, precuneus
Prat, Stocco, Neuhaus, and Kleinhans (2016)	Adult	GNG	16 (63)	25.3	107.4	17 (65)	25.6	111.0	--
Schmitz et al. (2006)	Adult	GNG, Switch, Stroop	10 (100)	38	105	12 (100)	39	106	ASD>TD: L M/IFG, OFC (GNG), insula (Stroop), and IPL, mesial parietal lobe (Switch)
Shafritz, Dichter, Baranek, and Belger (2008)	Adult	Switch	18 (89)	22.3	103	15 (87)	24.3	111	ASD<TD: L dlPFC, ACC, L intraparietal sulcus, L BG, L insula
Shafritz, Bregman, Ikuta, and Szeszko (2015)	Mixed	GNG	15 (80)	18.1	102	15 (80)	18.4	115	ASD<TD: R IFG/insula
Solomon et al. (2014)	Pediat.	Switch	27 (19)	15.4	108	27 (19)	16.1	113	ASD<TD: L PCC, L lingual gyrus, L MOG
Vaidya et al. (2011)	Pediat.	Stroop	11 (100)	10.8	114	14 (100)	11.0	119	ASD<TD: ACC, L MFG, R caudate
van Hulst et al. (2017)	Pediat.	GNG	26 (100)	10.8	109.6	26 (100)	10.5	117.3	--
Velasquez et al. (2017)	Adult	GNG	19 (68)	25.8	111	22 (73)	29.0	112	ASD<TD: R ACC, post-CG, R lateral occipital cortex
Yerys et al. (2015)	Pediat.	Switch	20 (80)	11.3	115	19 (68)	11.4	120	ASD>TD: L MFG/ pre-CG, L SFG/ dACC, R IFG

Abbreviations. N=sample size, y=year, pediat.=pediatric (child/adolescent) sample, ASD=autism spectrum disorder, TD=typically developing controls, L/R=Left/Right, GNG = Go/No-Go. Brain region (in alphabetical order): ACC=anterior cingulate cortex, BG=basal ganglia, dACC=dorsal ACC, dlPFC=dorsolateral prefrontal cortex, FFG=fusiform gyrus, I/M/medial/SFG=inferior/middle/medial/superior frontal gyrus, I/M/STG=inferior/middle/superior temporal gyrus, I/SPL=inferior/superior parietal lobe, mPFC=medial prefrontal cortex, OFC=orbital frontal cortex, OFG=orbital frontal gyrus, PCC=posterior cingulate cortex, pre-/post-CG=pre-/post-central gyrus, PHG=parahippocampal gyrus, PMC=premotor cortex, SMA=supplementary motor area, STS=superior temporal sulcus. See Supplement Table S2b for further information.

Table 3
Characteristics of Overall Sample and Subgroups Matched on Age, Sex and IQ

	ADHD	ASD	TD_{ADHD}	TDASD
(A) Overall Sample				
VBM				
N	1533	1445	1295	1477
% males	69	88	67	87
Mean age (range), y	20.9 (6-65)	17.2 (2-59)	21.1 (6-65)	16.4 (2-58)
Mean FSIQ (SD)	103 (7.2)	99 (17.3)	110 (10.8)	108 (5.1)
fMRI during Cognitive Control				
N	1001	335	1004	353
% males	77	83	69	80
Mean age (range), y	18.3 (7-50)	20.7 (7-52)	18.6 (7-50)	19.2 (7-52)
Mean FSIQ (SD)	102 (7.1)	109 (5.2)	109 (13.2)	114 (4.4)
fMRI during Motor Response Inhibition				
N	745	212	743	232
% males	77	82	69	78
Mean age (range), y	18.9 (7-50)	21.6 (8-52)	19.0 (7-50)	19.1 (8-52)
Mean FSIQ (SD)	101 (7.2)	109 (3.7)	109 (14.8)	114 (4.4)
(B) Matched Subgroups				
VBM				
N	825	758	778	781
% males	78	80	78	80
Mean age (range), y	16.7 (6-65)	17.4 (3-59)	17.4 (6-65)	16.1 (2-58)
Mean FSIQ (SD)	102 (8.3)	97 (18.0)	109 (4.7)	107 (12.6)
fMRI during Cognitive Control				
N	586	335	628	353
% males	83	83	79	80
Mean age (range), y	17.6 (7-50)	20.7 (7-52)	17.6 (7-50)	19.2 (7-52)
Mean FSIQ (SD)	105 (7.7)	109 (4.8)	109 (16.0)	114 (4.1)
fMRI during Motor Response Inhibition				
N	354	212	421	232
% males	78	82	73	78
Mean age (range), y	21.3 (7-50)	21.6 (8-52)	21.6 (7-50)	19.1 (8-52)
Mean FSIQ (SD)	106 (9.6)	109 (3.6)	114 (7.8)	114 (4.3)

Abbreviations: N = overall number of subjects, TDADHD = typically developing controls in the ADHD studies, TDASD = typically developing controls in the ASD studies, VBM = voxel-based morphometry, fMRI = functional Magnetic Resonance Imaging, % males = proportion of males among the samples, y = year, FSIQ = full scale IQ, and SD = standard deviation. The demographic characteristics presented here were calculated from the independent datasets with non-overlapping participants.

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Table 4
Brain Structural and Functional Abnormalities in ADHD relative to ASD

Contrasts	MNI coord. x, y, z	SDM Z p-value		Voxels No	BA
(A) Structural Abnormalities					
(i) ADHD (*vs.* TD)					
ADHD < TD					
L/R vmOFC/vmPFC/rACC and R caudate	2,48,-18	2.48	.00009	2902	11/10
R putamen/posterior insula/STG	30,-4,4	1.98	.0009	902	48/22
L pre-CG	-40,-6,56	1.88	.001	110	6
R rostrolateral PFC	28,66,-4	1.98	.0009	79	11
L vlPFC/STG/temporal pole	-26,16,-24	1.93	.001	57	38
ADHD > TD					
Nil	--	--	--	--	--
(ii) ASD (*vs.* TD)					
ASD < TD					
L/R dACC/dmPFC	4,42,18	1.61	.0004	575	24/32
L cerebellum	-10,-66,-48	1.35	.002	113	
R hippocampus/PHG/FFG	24,-6,-22	1.38	.002	104	34/20/36
Dorsomedial thalamus	0,0,20	1.39	.001	46	
ASD > TD					
L anterior I/M/STG/posterior insula	-60,-20,-14	2.55	.000002	1828	20/21/22/48
L/R PCC/precuneus,	-8,-50,26	1.99	.0003	437	23/26/7
R MFG/SFG/dlPFC	24,48,24	2.13	.0001	218	46
ADHD (*vs.* TD) *vs.* ASD (*vs.* TD)					
(iii) Overall Sample					
ADHD (*vs.* TD) reduced *vs.* ASD (*vs.*TD)					
vmOFC/rACC/L caudate	4,22,-10	2.22	.00009	1543	25/11
L post-CG	-42,-14,48	1.86	.0007	276	6
L MFG/SFG/dlPFC	-14,58,14	1.90	.0006	114	10
ASD (*vs.* TD) increased *vs.* ADHD (*vs.*TD)					
L anterior STG/MTG	-46,-16,-2	1.91	.0005	601	48/22/20/21
R MFG/SFG/dlPFC	24,46,24	1.98	.0004	150	46
R MFG/SFG	18,66,6	1.73	.001	91	10
L MTG/temporal pole	-46,6,-26	1.68	.002	56	20
R posterior MTG/STG	46,-36,4	1.64	.002	52	21
(iv) Matched Subgroups					
ADHD (*vs.* TD) reduced *vs.* ASD (*vs.*TD)					
vmOFC/rACC	2,48,-18	2.03	.001	442	11/10
L MOG	-22,-88,10	2.02	.001	85	18

Contrasts	MNI coord. x, y, z	SDM Z	p-value	Voxels No	BA
ASD (*vs.* TD) reduced *vs.* ADHD (*vs.* TD)					
R PHG/amygdala/FFG/hippocampus	28,-6,-24	1.37	.0002	386	28/34/36/35
Dorsomedial thalamus	2,-6,16	1.18	.0006	92	
ASD (*vs.* TD) increased *vs.* ADHD (*vs.* TD)					
R MTG/STG/angular gyrus	50, -40 ,8	2.28	.0004	463	42/40/22/21/39
R MFG/SFG/dlPFC	24, 44, 24	2.00	.001	65	46
L anterior MTG/STG	-50, -20, -8	1.84	.003	36	48/22/21/20
PCC	-6, -50, 28	1.84	.003	33	23/30
(B) Functional Abnormalities during Cognitive Control					
(i) ADHD (*vs.* TD)					
ADHD < TD					
R thalamus/caudate	10,-4,12	1.74	.0001	449	
L M/STG/superior temporal pole	-52,-16,-12	1.31	.001	285	21/22/38
L/R SMA/dmPFC	4,6,52	1.33	.001	130	6/24
L IFG/AI/temporal pole	-50,14,4	1.25	.002	82	45/48
R AI/putamen	32,20,0	1.27	.002	65	48
L post-CG	-48,-12,54	1.26	.002	53	6
L MFG/dlPFC	-30,30,44	1.24	.002	35	9
R MTG	46,-2,-24	1.18	.003	33	21
ADHD > TD					
Nil	--	--	--	--	--
(ii) ASD (*vs.* TD)					
ASD < TD					
L/R ACC/midcingulate/dmPFC	0,34,20	2.12	.000001	2708	24/10/32
L MFG/dlPFC	-42,34,24	1.42	.0003	370	45/46/48
R MFG/dlPFC	42,10,48	1.28	.0007	113	9
L IPL	-34,-46,52	1.11	.002	91	40
L lingual gyrus/cerebellum, lobule IV/V	-14,-48,-10	1.08	.003	89	30/19
ASD > TD					
Precuneus/midcingulate	-2,-44,56	1.48	.00007	1115	7/5/4
R IOG	36,-70,-6	1.37	.0002	226	19
L vlPFC/OFC	-30,40,-8	1.14	.0009	90	47/11
L MFG/rostrolateral PFC	-30,48,10	1.14	.0008	81	10
R IFG	42,22,18	1.08	.002	64	45
ADHD (*vs.* TD) *vs.* ASD (*vs.* TD)					
(iii) Overall Sample					
ASD (*vs.* TD) reduced *vs.* ADHD (*vs.* TD)					
rACC/midcingulate/dmPFC	0,30,20	2.42	~0	2903	24/10/32
L MFG/dlPFC	-44,34,24	1.19	.0004	319	45/46/48

Contrasts	MNI coord. x, y, z	SDM Z p-value		Voxels No	BA
ASD (*vs.* TD) increased *vs.* ADHD (*vs.* TD)					
Precuneus	0,-42,50	1.50	.00009	1001	7/4
R IOG/FFG	36,-70,-8	1.35	.0003	147	19
R IFG	44,24,18	1.27	.0005	138	48/45
L vlPFC/OFC	-30,44,-8	1.04	.002	32	47/11
L MFG	-30,50,8	1.02	.003	25	10
(iv) Matched Subgroups					
ADHD (*vs.* TD) reduced *vs.* ASD (*vs.* TD)					
SMA	-4, 12, 54	-1.33	.0003	353	6
L STG/MTG/temporal pole	-40, -6, -12	-1.01	.003	40	48/21/38
ASD (*vs.* TD) reduced *vs.* ADHD (*vs.* TD)					
rACC/dmPFC	2,28,20	2.30	.0000002	2815	24/32/9/10
L MFG/dlPFC	-40,34,28	1.03	.001	211	45/45/48
L IPL/SPL	-32,-46,54	1.06	.0009	192	40/7/2
ASD (*vs.* TD) increased *vs.* ADHD (*vs.* TD)					
Precuneus/PCC	-2, -40, 50	1.25	.0006	433	5/4
R FFG/IOG/ITG	36, -70 ,-8	1.49	.0001	247	19/37
R IFG	44, 24, 18	1.24	.0007	88	48/45
L vlPFC/OFC	-30, 40, -8	1.10	.002	57	11/47
(C) Functional Abnormalities during Motor Response Inhibition					
(i) ADHD (*vs.* TD)					
ADHD < TD					
L MFG/dlPFC	-30,30,44	1.70	.0002	235	9/46/8
L anterior MTG/STG	-52,-18,-10	1.57	.0005	210	22/21/20/48
L post-CG	-48,-10,54	1.52	.0006	198	6
R IFG	46,32,22	1.54	.0005	188	45/48/46
R vlPFC/OFC/AI	30,36,-16	1.30	.002	172	47/38
R caudate	10,20,0	1.38	.001	91	--
ADHD > TD					
Nil	--	--	--	--	--
(ii) ASD (*vs.* TD)					
ASD < TD					
R AI/vlPFC	44,20,0	1.42	.0004	491	47/45/48
L cerebellum, hemispheric lobule IV & V/ lingual gyrus/FFG	-14,-46,-10	1.33	.001	492	30/19/18/37
R MFG/dlPFC	40,12,50	1.43	.0004	251	9
R PCC/precuneus	16,-36,44	1.35	.0008	58	7
ASD > TD					
L vlPFC/OFC	-28,48,8	1.19	.0003	703	11/10/46/47

Contrasts	MNI coord. x, y, z	SDM Z	p-value	Voxels No	BA
R IOG/FFG/ITG	36,-72,-8	1.50	.00001	489	19/37
ADHD (*vs.* TD) *vs.* ASD (*vs.* TD)					
(iii) Overall Sample					
ADHD (*vs.* TD) reduced *vs.* ASD (*vs.* TD)					
R caudate	10,22,0	1.20	.0008	98	--
R IFG	42,26,20	1.14	.001	97	48/45
ASD (*vs.* TD) reduced *vs.* ADHD (*vs.* TD)					
L lingual gyrus/FFG/cerebellum lobule IV	-18,-54,-12	1.17	.001	243	19/37/30
rACC/dmPFC	0,32,22	1.09	.002	203	24/32
L MFG/IFG	-44,34,16	1.11	.002	109	45/48
R precuneus	16,-36,44	1.27	.0008	37	7
R MFG/dlPFC	42,10,48	1.04	.003	20	9/6
ASD (*vs.* TD) increased *vs.* ADHD (*vs.* TD)					
R IOG/FFG	36,-70,-8	1.84	.00001	417	19/37/18
L vlPFC/OFC	-28,42,-14	1.21	.0007	372	11/47
L MFG/SFG/dlPFC	-30,46,6	1.12	.001	183	10/46/11
L posterior MTG	-48,-54,4	1.16	.001	63	37/21
(iv) Matched Subgroups					
ADHD (*vs.* TD) reduced *vs.* ASD (*vs.* TD)					
R caudate	8,20,2	1.17	.001	117	--
R IFG	42,26,20	1.18	.001	74	48
ASD (*vs.* TD) reduced *vs.* ADHD (*vs.* TD)					
L cerebellum lobule IV/FFG/lingual gyrus	-16,-48,-12	1.39	.0003	1082	19/37
R MFG/dlPFC	42,14,48	1.22	.0007	104	9/6
L SPL/precuneus	-18,-70,58	1.00	.002	50	7
R precuneus	16,-36,44	1.21	.0008	41	7
ASD (*vs.* TD) increased *vs.* ADHD (*vs.* TD)					
R IOG/FFG	36,-70,-10	-1.82	.00001	504	19/37
L vlPFC/OFC	-28,36,-8	-1.17	.001	265	11/47
L MFG/SFG/dlPFC	-28,54,8	-1.10	.002	171	10/46/11

Abbreviations: MNI = Montreal Neurological Institute, SDM = Seed-based d mapping, BA = Brodmann Area, TD = typically developing controls, brain regions (in alphabetical order): AI = anterior insula, dACC = dorsal anterior cingulate cortex, dlPFC = dorsolateral prefrontal cortex, dmPFC = dorsomedial prefrontal cortex, FFG = fusiform gyrus, IFG = inferior frontal gyrus, I/MOG = inferior/middle occipital gyrus, I/M/STG = inferior/middle/superior temporal gyrus, I/SPL = inferior/superior parietal lobe, M/SFG = middle/superior frontal gyrus, PCC = posterior cingulate cortex, PFC=prefrontal cortex, PHG=parahippocampal gyrus, pre-CG=precentral gyrus, rACC = rostral anterior cingulate cortex, SMA = supplementary motor area, vlPFC = ventrolateral prefrontal cortex, vmOFC = ventromedial orbitofrontal cortex, vmPFC = ventromedial prefrontal cortex. Bold prints indicate regions which survived the sensitivity analyses in subgroups matched on age, sex and IQ.