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Neuroimaging Biomarkers for ADHD and ASD: Recent Advances, Transdiagnostic Overlaps, and Implications for Precision Psychiatry

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Abstract. Neurodevelopmental disorders such as Attention-Deficit/Hyperactivity Disorder (ADHD) and autism spectrum disorder (ASD) are highly prevalent, often comorbid conditions that significantly impact cognitive, behavioural, and social functioning. Neuroimaging has emerged as an important tool in understanding the underlying neural mechanisms of these disorders, contributing to efforts toward biologically grounded diagnosis and personalised intervention. This review synthesises findings from 28 recent neuroimaging studies (2022–2025) using MRI, fMRI, EEG, and advanced computational modelling to examine structural, functional, and network-level abnormalities in ADHD and ASD, as well as transdiagnostic overlaps. ADHD-specific studies highlight fronto-striatal dysconnectivity, delayed cortical maturation, and reduced basal ganglia iron levels, with recent advances in machine learning enhancing precision phenotyping. ASD-focused research underscores heterogeneity in cerebellar morphology, atypical predictive coding, and genetically driven cortical thinning. Transdiagnostic investigations reveal converging alterations in executive control networks and shared dimensional traits across diagnostic boundaries, supporting dimensional models of neurodevelopment. Despite these advances, challenges remain, including small and heterogeneous samples, overreliance on cross-sectional data, limited reproducibility, and interpretability issues in multimodal integration. The review concludes by advocating for longitudinal, harmonised, and multisite neuroimaging designs aligned with frameworks like the Research Domain Criteria (RDoC), as well as the incorporation of machine learning models that integrate cognitive, clinical, and biological data. By identifying shared and distinct neural markers, this review aims to support the development of robust, developmentally informed, and transdiagnostically valid neuroimaging biomarkers to inform early diagnosis and tailored interventions in neurodevelopmental disorders.

Keywords: Neurodevelopmental disorders, ADHD, ASD, Transdiagnostic

1. Introduction

A wide range of disabilities that interfere with brain development have been grouped together under the general category of "neurodevelopmental disorders" [1]. Rare genetic syndromes, cerebral palsy,

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congenital neural anomalies, autism and attention deficit hyperactivity disorder (ADHD are just a few of the neurological and psychiatric conditions that fall under this broad category despite their clinical and etiological differences [1]. Among these, autism spectrum disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) are two of the most frequently diagnosed, both showing high prevalence rate and exerting considerable impacts on individual functioning. According to a random-effects meta-analysis of relevant studies, 8.0% of children and adolescents worldwide suffer from ADHD [2]. ADHD symptoms typically appear before the age of twelve [3], affecting an individual's capacity to focus (inattention), their level of activity (hyperactivity), and their ability to control impulses (impulsivity) [3]. An inattentive child or adolescent may be easily distracted, having trouble following directions or listening to others, or forgetting to do simple things like put on socks or clean their teeth [3]. Although ASD is less prevalent than ADHD, it remains an important neurodevelopmental condition to consider. According to statistics from the CDC's ADDM Network, 3.2% of 8-year-old children have been diagnosed with ASD [4]. The diagnostic criteria for AD include deficits in verbal and nonverbal communication, restricted and repetitive patterns of behaviour, and impairments in social interaction [5]. Regardless of socioeconomic class, colour, nationality, or culture, these fundamental characteristics are present [5]. However, due to substantial individual differences among ASD patients, the prevalence of these diagnostic features can vary significantly across individuals [5]. Although diagnostic frameworks such as DSM-5 permit dual diagnoses, distinguishing between them remains challenging due to symptom overlap and heterogeneous clinical presentations [6]. For example, assessments are made more difficult by the overlap between characteristics like social inattentiveness and sensory sensitivity in ASD and fundamental attentional deficiencies in ADHD. This can result in delayed or incorrect diagnoses, which has long-term effects on treatment strategies and developmental outcomes.

To further understand the mechanisms underlying neurodevelopmental disorders, neuroimaging has become one of the most critical tools in assessing their neural bases. In addition to identifying brain variations in various neurodevelopmental diseases, neuroimaging techniques are being increasingly used for early diagnosis, treatment monitoring, and exploration of developmental trajectory. Magnetic resonance imaging (MRI) is a neuroimaging technique that uses strong magnets to generate a powerful magnetic field, causing protons in the body to align with it and enabling the acquisition of detailed three-dimensional antomical image [7]. MRI is commonly utilized to measure cortical thickness and volumetric abnormalities, facilitating the detection of atypical brain development such as cortical overgrowth in ASD or delayed cortical thinning in ADHD [8,9]. Functional MRI (fMRI), as another frequently used neuroimaging technique, reveals brain function by identifying regions activated during sensory stimulation or cognitive tasks [10]. However, since fMRI has relatively low temporal resolution, electroencephalography (EEG) is frequently used to further record brain activities by measuring brain's electrical signals with high temporal precision [11]. By enabling more nuanced characterisation of neurodevelopmental disorders, these technologies have advanced research towards individualised treatments and objective, biologically based diagnostics.

Despite the significant advances enabled by neuroimaging techniques, several critical gaps remain. First, the identification of reproducible biomarkers is hindered by the heterogeneity of clinical presentations and differences among subtypes [12]. The developmental interpretation of ASD and ADHD is limited by methodological inconsistencies and the predominance of cross-sectional study designs, despite meta-analyses highlighting both shared and disorder-specific brain abnormalities [12]. Second, the co-occurrence of ASD and ADHD complicates the interpretation of

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neural signatures. It remains unclear whether deficits such as attentional disruption arise from two different diagnostic categories or reflect manifestations of a shared underlying neurobiology [13]. Additionally, current diagnosis and treatment focus primarily on behavioural symptoms rather than brain abnormalities. Given that many individuals with ASD and ADHD may share certain biomarkers, it is necessary to re-examine the validity of the existing diagnostic classifications for these disorders.

To further address these challenges, this review aims to synthesise different neuroimaging findings in both ADHD and ASD, emphasizing developmental trajectories, comorbidity, and shared neural mechanisms. This review begins with a short methodological section which describes the literature search strategy and including criteria. Then we outlined the disorder-specific neuroimaging findings and transdiagnostic studies. Lastly, we concluded the current limitations and proposed future research directions. The purpose of this review is to provide guidance for the development of reliable neuroimaging biomarkers that aid with dimensional diagnosis, early detection, and tailored treatments for neurodevelopmental disorders.

2. Method

This review searched PubMed database for articles reporting neuroimaging findings in individuals with ASD or ADHD published between January 2022 and April 2025. The search terms used were (Comorbidity [title/abstract] OR transdiagnostic [title/abstract]) AND (neuroimaging [title/abstract] OR MRI [title/abstract]) AND ("Developmental Disorder"[title/abstract] OR "Autism"[title/abstract] OR "ADHD"[title/abstract]). The studies were included if they met the following criteria: 1. Studies described neuroimaging (EEG, fMRI etc.) findings related to ASD or ADHD patients, 2. Studies focused on ADHD or ASD transdiagnostic diagnosis, symptomatology, treatment response, or developmental trajectory, 3. Studies used original data and reported reliable results, and 4. Studies published in English and were fully accessible from Pubmed. The studies were excluded if they met the following criteria: 1. Studies were review papers, case studies, conference abstracts or meta-analyses, and 2. Studies were not related to this topic. After carefully assessment, at total of 26 papers were included and reviewed in this study.

3. Abnormal brain structure and function in ADHD patients

Neuroimaging studies have repeatedly observed the changes in both brain structure and function, particularly in areas related to executive functions, reward processing and attention regulation in Attention-deficit/Hyperactivity Disorder (ADHD) patients (Table. 1).

Table 1. ADHD related papers summary

Ref No.	Authors	Main Topic	MRI Modality	Participants	Participants Age	Neuropsychological Test	Key Findings
1	Long et al. [14]	Meta-analysis of gray matter abnormalities in pediatric BD and ADHD	Voxel-based morphometry (VBM)	268 PBD and 1,333 ADHD youth	Children and adolescents (mean 12–16 years)	Not specified; review/meta-analysis	Shared GMV reductions in right insula and ACC; distinct alterations in IFG, hippocampus, and precentral gyrus across disorders.

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2	Schulze et al. [15]	Brain iron levels and their association with cognition and comorbidities in ADHD	Quantitative Susceptibility Mapping (QSM)	111 children (ADHD = 58; controls = 53)	13.2 years (mean)	Sustained attention, inhibition (SSRT), working memory, IQ	No group differences in iron susceptibility; higher iron linked to better sustained attention; internalizing disorders showed higher iron (uncorrected).
3	Dupont et al. [16]	Sex differences in GPe functional connectivity in adults with ADHD, with/without comorbidities	Resting-state fMRI (seed-based analysis of external globus pallidus)	137 adults with ADHD (75F/62M), 45 healthy controls	18–50 years	DSM-IV ADHD diagnosis; CAARS, WURS-k, SCID; comorbidity history	ADHD males showed decreased GPe connectivity to frontal and occipital regions vs. females; FC patterns differed by sex and comorbid depression; no sex × overweight/SUD interaction.
4	Kim et al. [17]	Resting-state fMRI and graph theory in adolescents with social phobia ± ADHD	Resting-state fMRI (To achieve whole brain coverage with acceptable image repetition times, a voxel resolution of 3 × 3 × 3 mm with 46 slices was chosen.)	158 adolescents (36 SP-only, 60 SP+ADHD, 62 healthy controls)	Mean = 14.16 years	GOASSESS (modified Kiddie-SADS)	Altered default mode and cerebellar connectivity in SP; ADHD impacts topology (e.g., superior occipital gyrus); graph metrics used to classify groups via machine learning.
5	Li et al. [18]	Personalized fMRI analysis to identify generalizable biomarkers for symptom domains across SCZ, BP, and ADHD	Resting-state and task-based fMRI	142 adults with SCZ, BP, or ADHD (discovery = 78; validation = 55)	Adults (mean 34 years)	67 clinical symptom items across YMRS, HAMD, BPRS, ASRS	Symptom-domain biomarkers (e.g., attention, appetite, psychosis) are more reproducible with individualized fMRI than group-based; replicated in external sample.
6	Segura et al. [19]	Brain-symptom-genomic mapping in children with autism and ADHD	Resting-state fMRI	166 children (6–12 years) with ASD or ADHD	6–12 years	ADOS-2, KSADS, SRS-2, SWAN, SCQ, CBCL	Autism symptom severity correlated with iFC in FPN and DMN; no significant ADHD-symptom associations; implicated gene expression linked to neuron projection.
7	Wu et al. [20]	Comparison of clinical, cognitive, and neuroimaging differences between ADHD-I and ADHD-C	Structural MRI, Diffusion Tensor Imaging (DTI)	277 children with ADHD (145 ADHD-I, 132 ADHD-C), 98 controls	Children	CBCL, Conners' Rating Scale, TMT, Stroop, CANTAB	ADHD-C had more behavioral/emotional problems; ADHD-I had greater anxiety; neuroimaging showed distinct structural patterns.

In terms of brain structure, ADHD has been associated with mild yet widespread reductions in grey matter volume, particularly in the prefrontal and subcortical regions [14]. By examining voxel-

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based morphometry across 29 studies [14], reported consistent reductions in the bilateral dorsolateral prefrontal cortex, anterior cingulate cortex, and putamen. These regions play a critical role in attentional control and response inhibition, suggesting that structural immaturity or underdevelopment in these regions may underlie core symptoms of inattention and impulsivity [14] Furthermore, the cognitive and affective characteristics of ADHD patients were explained by the more pronounced decreases in volumes in the left precentral gyrus, left inferior frontal gyrus, and right superior frontal gyrus [14]. Using quantitative susceptibility mapping (QSM) to examine brain iron levels [15], added a new dimension to ADHD neuroimaging, reporting that children with ADHD have lower levels of iron in basal ganglia, a biomarker potentially linked to dopaminergic dysfunction, a well-established neurochemical characteristic of the disorder. Additionally [16], found that adult males with ADHD exhibited decreased connectivity between the globus pallidus externus (GPe) and the anterior cingulate cortex, a pattern not observed in females.

fMRI studies have revealed global alterations in functional networks. Using graph theory on resting-state fMRI data from teenagers [17], found changed global efficiency and modularity in important networks, such as the default mode and salience networks. This result is consistent with other research indicating that internally directed cognition and salience attribution are dysregulated in ADHD [15-17].

Moreover, individual-level analyses have highlighted the potential of inter-individual variability advancing precision psychiatry [18], who employed individualised functional connectomes to distinguish ADHD from healthy controls more accurately than group-level analyses, serve as an example of the shift towards personalised neuroimaging in ADHD [19]. employed machine learning and connectome-based symptom mapping in a related field to pinpoint particular functional circuits associated with the severity of clinical symptoms. They suggested customised targets for potential interventions such as transcranial magnetic stimulation (TMS) by simulating the effects of neuromodulation, suggesting that neuroimaging-guided treatment personalisation may be a promising direction. Additionally, [20] categorize ADHD into inattentive and mixed subtypes and found that combined-type patients exhibited worse cognitive performance and more widespread functional abnormalities.

Consequently, these research agree on a paradigm that conceptualises ADHD as a disorder with altered neurodevelopmental trajectories, including changes in structural maturation, iron deposition and functional networks. There is potential to move beyond categorical diagnosis and towards more physiologically based, individualised care by combining multimodal neuroimaging with individualised and symptom-focused approaches. To implement these strategies in clinical settings, translational validation, harmonisation of analytical workflows, and replication in larger samples are still necessary.

4. Abnormal brain structure and function in ASD patients

Neuroimaging studies dedicated to ASD have discovered a complex and heterogeneous neural profile, spanning structural, functional, and network-level alterations. The six ASD-specific studies (Table. 2) reviewed here offer insights into the neurobiological basis of symptom variability, cerebellar morphology, predictive coding disruptions, and the roles of genetic and clinical heterogeneity, but also show some limitations which need to be addressed in future research.

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Table 2. ASD related papers summary

Ref No.	Authors	Main Topic	MRI Modality	Participants	Participants Age	Neuropsychological Test	Key Findings
1	Elandaloussi et al. [21]	Cerebellar morphology and its association with social cognition in autism	Structural MRI (cerebellar volumetry using CERES)	n = 850 children and adolescents from Healthy Brain Network	Mean = 10.8 years (range 5–18 years)	Social Responsiveness Scale (SRS)	Cerebellar volume in cognitive lobules is associated with social communication and IQ; suggests cerebellum's role in social functioning across diagnoses.
2	Duan et al. [22]	Predicting anxiety (MASC-2 score) in children with ASD using fMRI and spectral graph neural networks	Resting-state functional MRI (rs-fMRI)	70 children with ASD and 26 typically developing controls	8–15 years	Multidimensional Anxiety Scale for Children (MASC-2)	Spectral features (FFT, PSD) improved anxiety prediction accuracy over standard correlation-based models; best model MAE ≈ 13.77.
3	Nenadić et al. [23]	Predictive coding abnormalities across neuropsychiatric disorders	fMRI, EEG, behavioral paradigms	72 studies across SCZ, ASD, mood, cognitive, PTSD, SUD	All ages	Oddball, illusion, and decision tasks for predictive coding	SCZ shows impaired non-social predictive coding; ASD shows deficits in social cue prediction; predictive coding linked to symptom severity.
4	De met et al. [24]	Interaction of autistic and schizotypal traits on hippocampal structure and function in healthy adults	Structural MRI and arterial spin labeling (ASL)	318 for MRI, 346 for ASL (nonclinical adults)	18–40 years	Autism Quotient (AQ), Multidimensional Schizotypy Scales, SPQ-B, O-LIFE	Synergistic effects of schizotypy and autistic traits on hippocampal subfield volumes and rCBF; some traits modulate structure/function interactions.
5	De met et al. [25]	Genetic heterogeneity and clinical profiles in developmental and epileptic encephalopathies (DEEs)	Structural MRI and EEG (EEGs were performed during the sleep-wake cycleusing 21 electrodes (including Fp1, Fp2, F3, F4, F7,F8, T3, T4, T5, T6, C3, C4, P3, P4, O1, O2, Fz, Cz, Pz,a ground electrode and a reference electrode))	20 children with DEE	0–16 years	IQ/DQ assessments (Gesell, WPPSI, WISC)	High genetic heterogeneity; DEE often linked to developmental delay, autism, intellectual disability; 80% had normal MRIs, but EEG abnormalities common.

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6	van Oort et al. [26]	Linked ICA of multimodal neuroimaging and biobehavioral dimensions across disorders	Multimodal MRI (VBM, DTI, rs-fMRI, stress fMRI)	295 adults (225 with psychiatric diagnoses, 70 controls)	18–74 years	CAARS, AQ-50, IDS-SR, BRIEF-A, PID-5, ASI, PTQ	ECN-FPN connectivity under stress linked to negative affect and cognitive symptoms; multimodal DMN component linked to ASD diagnosis.

From a structural perspective [21], used structural MRI and the CERES segmentation pipeline to investigate cerebellar morphology in a large sample of 850 children and adolescents. They reported that greater volume in cerebellar cognitive lobules, such as Crus I and II, was associated with enhanced social responsiveness, reinforcing the cerebellum's expanding role in social cognition. However, the reliance on anatomically rather than functionally defined regions may have weakened region-function specificity. Additionally, the study's cross-sectional design limited its ability to track developmental changes over time.

Turning to functional neuroimaging and predictive modeling [22], applied a novel Spectral Brain Graph Neural Network (SpectBGNN) to resting-state fMRI data from 70 children with ASD to predict anxiety symptoms. Their use of frequency-domain features (e.g., FFT, PSD) outperformed conventional connectivity measures, illustrating the potential of AI in psychiatric phenotype prediction. However, the model's generalizability was limited by a modest sample size, narrow focus on a single comorbidity, and absence of external validation. Additionally [23], synthesized evidence across fMRI, EEG, and behavioral paradigms to test predictive coding models, finding that individuals with ASD exhibited reduced sensory precision in auditory and sensorimotor circuits. While supporting theories of atypical perceptual inference, the heterogeneity of tasks and demographics in the synthesis limits interpretability, and the underrepresentation of children restricts developmental conclusions.

These studies approached ASD from transdiagnostic or multimodality perspectives [24]. used structural MRI and arterial spin labeling in a large non-clinical sample to explore autistic and schizotypal traits. They found shared alterations in temporal regions and distinct cortical surface features, supporting models of dimensional interaction across psychiatric phenotypes. However, the reliance on self-report measures, the absence of clinical diagnoses, and the cross-sectional design weaken causal inferences about neurodevelopmental mechanisms [25]. also adopted a structural lens, examining children with developmental and epileptic encephalopathy (DEE) who exhibited ASD-like traits. Their findings highlighted cortical thinning linked to diverse genetic variants, emphasizing the complexity of genotype–phenotype relationships. Nevertheless, the small and specific DEE sample reduces generalizability to idiopathic ASD, and the lack of formal statistical integration across EEG, MRI, and genetic data limits the interpretive power of their multimodal approach. Similarly, focusing on functional dynamics [26], applied linked independent component analysis (ICA) to integrate structural, diffusion, and functional MRI data in youth with ASD. They identified multimodal brain components associated with executive function and internalizing symptoms, exemplifying the strength of integrated dimensional imaging. Nonetheless, linked ICA's statistical complexity hinders mechanistic interpretation and practical clinical translation, and replication in independent cohorts is still required.

Taken together, these findings enhance our understanding of ASD as a complex and biologically heterogeneous disorder. They demonstrate how genetic variation, network-level dynamics, cerebellar morphology, and computational models contribute to explaining the variability in ASD. Nonetheless, common challenges — such as small or highly specific samples, lack of longitudinal data, overreliance on cross-sectional designs, and limited external validation — underscore the need

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for reliable, developmentally sensitive, and reproducible neuroimaging research. Future studies would benefit from larger, stratified cohorts; interpretable multimodal integration; and designs that link brain mechanisms to meaningful clinical outcomes.

5. Transdiagnostic alterations in brain structure and function

Neurodevelopmental disorders including ASD and ADHD show similarities in brain anatomy, behaviour, and cognition, as demonstrated by an expanding body of research (Table. 3). This has led to a transdiagnostic shift in neuroimaging, aiming to uncover shared brain mechanisms, developmental pathways, and symptom dimensions rather than focusing solely on categorical diagnoses The reviewed studies — including meta-analyses, multimodal clustering, genetic correlations, and functional network mapping — provide an integrated perspective on brain-based similarities among ASD, ADHD, and related psychiatric disorders.

Table 3. Transdiagnostic papers summary

Ref No.	Authors	Main Topic	MRI Modality	Participants	Participants Age	Neuropsychological Test	Key Findings
1	Park et al. [27]	Transdiagnostic cortical thickness alterations across six psychiatric disorders	Structural MRI (cortical thickness)	28,546 total (12,876 patients, 15,670 controls)	All ages (pooled ENIGMA samples)	Not applicable (meta-analysis of imaging data)	A shared morphological alteration pattern found across disorders; paralimbic regions most affected; correlated with myeloarchitecture, neurotransmitter systems (dopamine, serotonin), and functional gradients.
2	Rootes-Murdy et al. [28]	Transdiagnostic cortical gray matter patterns across eight psychiatric disorders using federated neuroimaging	Voxel-based morphometry (VBM)	4,102 individuals across eight sites	Mixed ages across psychiatric and control groups	Not applicable	Gray matter reductions found in bilateral insula, medial PFC, and parahippocampal regions across multiple disorders; federated analysis ensures data privacy.
3	Norbom et al. [29]	T1w/T2w-ratio and multimodal clustering to identify brain-based subtypes in ASD and ADHD	T1w/T2w MRI, cortical thickness, and surface area	310 participants (ASD = 136, ADHD = 100, TD = 74)	2.6–23.6 years	SCQ, RBS-R, SWAN, IQ measures	No case-control differences in T1w/T2w; multimodal clustering revealed 3 subgroups with distinct cortical profiles but similar clinical traits.
4	Watanabe & Watanabe [30]	Neural dynamics of comorbid ASD+ADHD vs. pure ASD/ADHD	Resting-state fMRI	Pure ADHD (N=30), pure ASD (N=30), ASD+ADHD (N=33), TD (N=67)	5–13 years	ADI-R, CPRS	ASD+ADHD children show unique frontoparietal dynamics not seen in pure ASD/ADHD; ADHD-like traits in comorbid group are distinct.

DOI: 10.54254/2753-7048/2025.ND26282

5	Vandewouw et al. [31]	Transdiagnostic subgroups based on resting-state connectivity across ASD, ADHD, and OCD	Resting-state fMRI	POND (N=551) and HBN (N=551); ages 5–19	5–19 years	SWAN, IQ tests, diagnostic measures	Biologically homogeneous subgroups identified, associated with behavior (e.g., impulsivity), not diagnosis; replicated across two datasets.
6	Meda et al. [32]	Meta-analysis of set-shifting task fMRI studies across multiple disorders	Task-based fMRI (set-shifting)	466 patients and 457 healthy controls	Adults (18–43 years)	Set-shifting tasks (e.g., WCST, TMT, Stroop)	Shared hyperactivation in medial frontal, ACC, superior parietal, and temporal regions; frontoparietal network involved across disorders.
7	Nakua et al. [33]	Link between cortico-amygdalar connectivity and externalizing/internalizing behaviors in ASD, ADHD, and OCD	T1-weighted MRI, rs-fMRI, and DWI	346 children aged 6–18 (ASD, ADHD, OCD, TDC)	6–18 years	Child Behavior Checklist (CBCL)	No significant associations between externalizing/internalizing behaviors and cortico-amygdalar connectivity; null findings validated with bootstrapping.
8	Xie et al. [34]	Identification of a neuropsychopathological (NP) factor linking multiple psychiatric disorders	Task-based fMRI, Resting-state fMRI	IMAGEN (N=1,750), with validation in ABCD, HCP, ADHD-200, STRATIFY/ESTRA (total N=4,942)	Adolescents to young adults	Multiple behavioral symptom assessments (externalizing, internalizing), DAWBA	NP factor reflects shared crossdisorder brain connectivity signatures; generalizable across samples and timepoints; linked to delayed PFC development and executive function deficits.
9	van Eijndhoven et al. [35]	Protocol using RDoC to study shared and distinct mechanisms of psychiatric comorbidity	Structural and functional MRI	Target N = 650 patients, 150 controls (adults)	≥18 years	CAARS, AQ-50, IDS-SR, ASI, SCID, MATE-Cr, BRIEF-A, etc.	Study integrates multi-level data (genes to behavior) across NDD and stress-related disorders; promotes dimensional, RDoC-based models.
10	Wen et al. [36]	Review of machine learning approaches for dimensional neuroimaging endophenotypes	Multimodal MRI (review: structural, functional, DTI)	Review (AD, SCZ, MDD, ASD, MS; >20 studies)	All ages (Review)	Not applicable (review)	Machine learning enables discovery of brain-based subtypes and transdiagnostic endophenotypes; DNE framework proposed.
11	Zhang et al. [37]	Symptom subtyping in ASD and ADHD using clustering, neurocognition, and DTI	Structural MRI and Diffusion Tensor Imaging	164 (ASD=65, ADHD=47, TD=52)	Children	AQ, SNAP-IV, CANTAB, C-WISC-III, PPT, VF	Three transdiagnostic subtypes identified with distinct symptom-neurocognition-connectivity profiles; corpus callosum and fine motor function key mediators.
12	Hoy et al. [38]	Genetic and neural correlates of transdiagnostic symptom dimensions across the lifespan	Structural and functional MRI (multi-study review)	46 genomic/neuroimaging studies in general population samples	All ages (children to older adults)	Latent dimensional models (HiTOP, bifactor, PCA, etc.)	Transdiagnostic factors (e.g., p-factor) associated with polygenic scores (ADHD, neuroticism) and global cortical structure; brain alterations and genetic risk shared across disorders.

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13	More au et al. [39]	Comparison of CNVs, PRSs, and idiopathic conditions in terms of their effect on functional brain connectivity	Resting-state fMRI	33,452 individuals across 9 datasets	Mixed (children to older adults)	Not applicable (genetic-connectome analyses)	CNVs had larger effects on connectivity than PRSs; effects aligned with CNV gene count and severity score; PRSs had limited connectivity impact.

The structural convergence across illnesses, especially in cortical morphology, is a major theme in transdiagnostic neuroimaging [27]. reported that large-scale cortical thickness gradients capture shared alterations between ASD and ADHD, especially within association cortices involved in attention and cognitive control. Similarly [28], implicated reductions in the prefrontal and temporal lobes in their analysis of transdiagnostic grey matter similarities between ASD and ADHD. By grouping young people with ASD and ADHD based on T1w/T2w ratios and cortical thickness [29], built on this and identified neurobiologically defined subtypes that transcend diagnostic categories. Despite the fact that these studies show a strong anatomical basis for dimensional models, conclusions on developmental trajectories are limited due to their reliance on cross-sectional samples and the lack of replication in independent cohorts.

Complementing these structural findings, functional connectivity studies have identified shared disruptions in executive and salience networks [30]. reported that children with comorbid ASD and ADHD exhibit less flexible frontoparietal dynamics compared to those with either disorder alone, suggesting that neural rigidity may underlie overlapping cognitive deficits [31]. similarly used resting-state fMRI to identify replicable subgroups across neurodevelopmental conditions, revealing distinct patterns of network integration and segregation that do not align with diagnostic labels. These studies underscore the importance of individualised and dimensional approaches. However, the absence of task-based validation and overreliance on resting-state data limits interpretation of functional specificity.

Task-based and meta-analytic analysis reveal another layer of convergence. In a meta-analysis of fMRI research on set-shifting tasks [32], discovered that the dorsolateral prefrontal cortex and anterior cingulate cortex—areas linked to cognitive flexibility—were consistently hypoactivated in ASD and ADHD. These findings support a dimensional view that executive dysfunction is a common cognitive. However, this study did not account for comorbidities within samples and instead relied on activation maps drawn from the literature, which could complicate findings. Dimensional models have also been used to study the neurobiological relationships between traits. For example [33], found that lower prefrontal–limbic coupling predicted behavioural dysregulation across diagnostic boundaries in their examination of cortico-amygdalar connectivity in relation to externalising symptoms. Smilarly [34], discovered a "neuropsychopathological factor" that is common to several mental illnesses and is connected to anatomical changes in the medial prefrontal cortex and anterior insula. Although these studies frequently lack developmental granularity and rely on trait-based measures without diagnostic confirmation, they provide strong support for cross-disorder endophenotypes.

The Research Domain Criteria (RDoC) framework and multimodal integration have also influenced transdiagnostic perspectives. For example [35], presented the MINDS study protocol, which utilises cognitive tasks, diffusion imaging, and fMRI to evaluate constructs such as working memory and threat processing across both ADHD and ASD. This design is a prime example of the potential of dimensional, cross-domain imaging, even though it is still in the recruitment stage. In the same way [36], examined machine learning applications for dimensional neuroimaging,

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highlighting the potential of multi-domain integration while also warning against task heterogeneity and overfitting, which compromise the generalisability of the model.

Through the clustering of neurobiological traits and symptom dimensions, machine learning and data-driven approaches have significantly advanced the area. By using unsupervised clustering on behavioural and neuroimaging data [37], discovered transdiagnostic symptom categories that provided a more compelling explanation for variation than diagnostic labels. After reviewing multi-study neuroimaging-genetic data [38], came to the conclusion that latent dimensional factors—like the p-factor—offer more reliable connections with brain structure than models specific to a given illness. Likewise [39], shown that genetic heterogeneity, such as polygenic risk scores and CNVs, influences brain connectivity in ASD and ADHD, especially in frontal-temporal networks. Despite their potential, these models are frequently hampered by differences in sample size, neuroimaging acquisition methods, and the lack of external validation.

Taken together, these findings show that there is significant neurobiological overlap between ASD and ADHD, especially in networks that promote attention, cognitive flexibility, and emotion regulation as well as in prefrontal, cingulate, and temporal areas. Transdiagnostic approaches provide a robust framework for understanding shared brain mechanisms and tailoring interventions to symptom dimensions rather than categorical diagnoses. However, to realise the promise of physiologically grounded precision psychiatry, harmonised, longitudinal, and multi-center research is needed to address typical obstacles, such as cross-sectional designs, limited sample numbers, and inconsistent imaging modalities.

6. Conclusions and future directions

This review synthesised current neuroimaging research on ADHD, ASD, and their transdiagnostic overlap. Converging evidence from structural, functional, and multimodal imaging modalities shows that extensive changes in the brain networks and regions underlying executive function, social cognition, attention regulation, and sensory integration constitute the foundation of neurodevelopmental disorders. ASD-specific research found unique cerebellar contributions, predictive coding anomalies, and genotype-related structural variability, whereas ADHD studies focused on disrupted fronto-striatal circuitry maturation, altered functional connectivity, and emerging applications of individualised models. Crucially, transdiagnostic research transcended categorisation frameworks and revealed shared-dimensional links between the brain and behaviour that transcended diagnostic boundaries, especially in the temporal, cingulate, and prefrontal cortices.

The discipline still faces significant methodological and translational obstacles in spite of these advancements. The use of cross-sectional data in several studies limited our knowledge of developmental trajectories. Replication across independent cohorts is still uncommon, and generalisability is diminished by small or demographically limited samples. Furthermore, although promising, the integration of multimodal data is frequently statistically difficult and uninterpretable, which hinders practical use. Reproducibility and meta-analytical synthesis are further complicated by the variation in analytic pipelines, imaging acquisition parameters, and neuropsychological evaluations.

Therefore, future research should prioritise longitudinal, multisite designs employing standardised protocols. Dimensional frameworks, such as those aligned with the RDoC, may provide more comprehensive descriptions of both shared and disorder-specific mechanisms. Furthermore, to improve diagnostic and prognostic value in the real world, machine learning models ought to include clinical, cognitive, and biological aspects. To ensure that neuroimaging

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technologies benefit diverse populations and capture the entire range of neurodevelopmental diversity, the field must strive for transparency, interpretability, and inclusivity.

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