UNDERGRADUATE PAPER

NEUROBIOLOGICAL AND NEUROPSYCHOLOGICAL DIFFERENCES IN AUTISM AND ADHD

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Abstract

Autism spectrum disorder and attention-deficit/hyperactivity disorder share cognitive and behavioral features, yet their overlap does not imply identical neurobiology. This narrative review integrates a large structural neuroimaging investigation, a meta-analysis of task-based functional magnetic resonance imaging, and a review of cerebellar and neurocognitive functioning. The evidence indicates partly distinct cortical and functional patterns alongside shared difficulties involving executive functions and distributed brain networks. Cerebellar contributions extend beyond movement to cognitive and emotional processes, although causal pathways remain uncertain. Psychosocial stress is relevant to daily functioning and the interpretation of symptoms, but the reviewed evidence does not establish stress as the cause of observed brain differences. Diagnostic interpretation is further complicated by co-occurring conditions, developmental variation, and the underrepresentation of female participants and people with greater support needs. These findings support multidimensional assessment that connects developmental history, neuropsychological functioning, and environmental demands. Current group-level imaging results provide explanatory context rather than a validated diagnostic test for an individual.

Keywords: autism, ADHD, neuropsychology, cerebellum, diagnosis

Neurobiological and Neuropsychological Differences in Autism and ADHD

Autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) can involve overlapping difficulties with attention, cognitive flexibility, and emotional regulation. Similar behavior, however, can emerge from different combinations of neurodevelopmental characteristics and environmental demands. Interpreting that behavior requires attention to both biological variation and the circumstances in which difficulties become visible. This narrative review integrates structural neuroimaging, task-based functional imaging, and neuropsychological evidence to examine their implications for symptom presentation and diagnosis (Bedford et al., 2025; Cundari et al., 2023; Tamon et al., 2024). The central argument is that neurobiology can inform a more precise understanding of autism and ADHD while individual assessment must remain sensitive to developmental history, co-occurrence, and functional needs.

Structural and Functional Brain Differences

In a large study published in *Biological Psychiatry*, Bedford et al. (2025) examined structural magnetic resonance imaging (MRI) data from 4,255 participants, including 1,399 autistic individuals, 987 individuals with ADHD, and 1,869 typically developing comparison participants. Their population-modeling approach compared anatomical measurements with age- and sex-referenced brain charts derived from 75,241 individuals. These centile scores locate a measurement within a reference distribution; an unusual centile is not itself evidence of a diagnosis. The approach nevertheless improves comparisons by placing anatomical variation within a developmental framework. Because the study was cross-sectional, these age-referenced differences describe variation between participants rather than observed change within the same individual. Small average differences also imply substantial overlap between diagnostic groups.

Autism was associated with greater cortical thickness and volume in the superior temporal gyrus, with small effects of approximately Cohen's $d = 0.13$ to 0.15. ADHD showed more widespread increases in cortical thickness alongside lower cortical volume and surface area. Adjustment for global brain measures substantially changed the ADHD results, indicating that many regional differences reflected broader anatomical variation. An exploratory subgroup of 203 participants with documented autism and ADHD showed widespread increases in cortical thickness and some reductions in surface area. Incomplete secondary-diagnosis information limits conclusions about whether this pattern represents a distinct co-occurring phenotype (Bedford et al., 2025).

Functional evidence complements these anatomical findings. Writing in the *American Journal of Psychiatry*, Tamon et al. (2024) synthesized 243 task-based functional MRI studies, encompassing reported samples of 3,084 participants with ADHD, 2,654 autistic participants, and 6,795 comparison participants. To reduce bias from studying different tasks in each diagnosis, they balanced cognitive task categories using the Research Domain Criteria framework. Shared activation differences included greater activation in the lingual and rectal gyri and lower activation in frontal and temporal regions. Diagnosis-specific patterns were more prominent than shared patterns. However, task-related activation is not a direct measure of a person's everyday competence, and anatomical and functional results cannot be treated as interchangeable evidence of one underlying mechanism.

The functional findings require care because functional MRI measures changes in blood oxygenation as an indirect correlate of neural activity. Greater activation can reflect different strategies or task demands rather than greater ability. Balancing task categories reduces one source of bias without equating stimuli, medication exposure, or participant characteristics

(Tamon et al., 2024). Consequently, a region's involvement in both diagnoses does not establish identical cognitive processing, and its apparent specificity does not validate a diagnostic biomarker.

Neuropsychology and Cerebellar Networks

Neuropsychology helps connect biological findings to the cognitive processes involved in daily functioning. Cundari et al. (2023) reviewed attention, processing speed, memory, executive functions, and social cognition across ADHD, autism, and spinocerebellar ataxia type 3. In ADHD, the reviewed literature frequently identified difficulties with inhibition, sustained attention, and planning, although some studies found preserved performance. Autistic samples also showed variable difficulties with cognitive flexibility, working memory, and social cognition. This variability argues against assuming that every person with a diagnosis has the same cognitive profile. A low score on an inhibition or memory task cannot, by itself, distinguish autism from ADHD.

The cerebellum provides one possible link across these domains. Beyond coordinating movement, it interacts with cerebral networks involved in cognition and emotional processing. Cundari et al. (2023) describe evidence connecting cerebellar differences with timing, learning, and executive functioning. Altered communication between the cerebellum and other regions may contribute to difficulties adapting behavior as demands change. Much of this account draws on heterogeneous human studies and animal models, however, so it does not establish a single cerebellar cause for either condition. Comparisons with a degenerative neurological disease also identify potentially shared functional systems without demonstrating equivalent developmental mechanisms.

Stress and Symptom Presentation

Stress adds a clinically relevant dimension to this interpretation. Cundari et al. (2023) discuss how cognitive difficulties can contribute to psychosocial stress at school and work. They also describe organizational demands associated with frustration and anxiety in ADHD, and sensory overload or disrupted routines associated with distress in autism. These observations suggest that the impact of cognitive differences depends partly on environmental demands. They do not establish that stress causes autism, ADHD, or the cortical differences reported by Bedford et al. (2025). Nor do the imaging studies directly test a causal pathway from stress to diagnostic presentation.

A reasonable implication is to examine the contexts in which symptoms intensify and the supports available in those settings. Anxiety, sleep difficulties, and fatigue may complicate the interpretation of attention or emotional regulation problems (Cundari et al., 2023). Assessment should therefore consider whether distress accompanies longstanding neurodevelopmental difficulties, reflects an additional condition, or varies with situational demands. This contextual interpretation is a clinical implication of the synthesis, rather than a tested diagnostic protocol. Future research could test this interpretation by repeatedly measuring stress and symptom severity while accounting for sleep, medication, and co-occurring anxiety. Such a design would help determine whether changes in stress accompany changes in presentation within individuals.

Diagnostic Interpretation and Representation

Biological variation also raises questions about whose presentation is represented in research. Bedford et al. (2025) found sex-related differences in autism-associated cortical patterns; the superior temporal volume finding appeared to apply primarily to male participants. Comparable sex-by-diagnosis interactions were not detected for ADHD. The study measured sex

assigned at birth and could not examine gender identity effects. Its results therefore cannot explain all diagnostic experiences of women or gender-diverse people. They do show why findings dominated by male samples require caution when generalized to other populations.

For families and adults seeking clarification, the practical goal of diagnosis is an account of difficulties that can guide useful support. The reviewed evidence favors considering co-occurring autism and ADHD alongside each condition's specific features (Tamon et al., 2024). Developmental history and functioning across settings can contextualize neuropsychological results, while cognitive profiles may help identify needs that a diagnostic label alone leaves unexplained (Cundari et al., 2023). Connecting assessment to documented support needs is a reasonable service implication. None of these studies directly measures whether a particular assessment pathway improves access to services.

Limitations and Conclusion

This review draws on three publications with different designs and constitutes a focused narrative synthesis rather than a systematic search. Bedford et al. (2025) report small effects, cross-sectional data, and limited representation of people with greater support needs. Tamon et al. (2024) note residual task differences, possible participant overlap, and unresolved medication effects. Cundari et al. (2023) integrate diverse findings without a pooled estimate. These limitations preclude individual diagnostic rules or causal conclusions. Together, the publications support a model of autism and ADHD involving partially shared cognitive systems and distinguishable biological patterns. Their strongest diagnostic implication is the need to integrate that evidence with individual development and context, including stress, while preserving a clear distinction between explaining group differences and identifying one person's needs.

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