OPINION

Reconceptualising ADHD as a neurobiological spectrum: calibration-based perspective

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Summary Although attention-deficit hyperactivity disorder (ADHD) diagnosis requires a categorical decision, phenotypic, twin and molecular genetic evidence indicates that ADHD traits are continuously distributed, with clinical ADHD at the functionally impairing end of this distribution. This paper proposes conceptualising ADHD as a neurobiological spectrum while treating categorical and dimensional approaches as complementary. A calibration perspective is proposed to describe graded differences in the regulation of attention, arousal, motivation and cognitive control, and their context-dependent expression. Genetic findings support dimensional continuity, whereas neurobiological and cognitive findings are consistent with graded regulatory variation. Functional impairment remains central to translating this dimensional framework into diagnosis and treatment decisions.

Keywords ADHD; calibration; dimensionality; neurobiological spectrum.

ADHD: a neurobiological spectrum?

When neurodevelopmental disorders are considered, the limitations of psychiatry's present or absent model of classification become more apparent. This paper argues that attention-deficit hyperactivity disorder (ADHD) traits are continuously distributed in the population and that clinical ADHD represents the end of this distribution associated with functional impairment. Within this framework, it proposes that dimensional biology and categorical clinical diagnosis address different but complementary questions.

From this perspective, the paper proposes a calibration perspective to explain how dimensional regulatory differences associated with ADHD may lead to different functional outcomes depending on context. Here, the term 'calibration' refers to the adjustment of attention, arousal and cognitive control systems according to changing task-based and environmental demands. The term 'neurobiological spectrum' refers to graded and partially overlapping variations at genetic and neurocognitive levels between clinical ADHD and lower levels of ADHD traits in the general population, rather than to a single biological axis. The dimensional approach describes how traits are distributed in the population, whereas categorical diagnosis determines when clinical assessment and intervention are required. This distinction also provides a basis for this perspective to explain why similar ADHD tendencies may result in different functional outcomes across individuals and contexts. This approach is not a new definition of the disorder, but a proposal that integrates existing evidence at the same time; it does not question the need for pharmacological and psychosocial interventions in individuals with impaired functioning.

Category and dimension: complementary perspectives on ADHD

The current literature shows that ADHD symptoms are continuously distributed in the general population and that clinical diagnostic thresholds are pragmatic boundaries based on functioning.[1] For example, taxometric analyses support a dimensional latent structure for inattention, hyperactivity/impulsivity and ADHD.[1] Therefore, rather than forming a qualitatively distinct group, individuals with ADHD may lie at the functionally impairing end of continuously distributed behavioural traits. This can be explained using the hypertension model. Blood pressure is a parameter present in everyone but, when it causes organ damage, it becomes a disease requiring intervention. Similarly, although the dimensional nature of ADHD does not eliminate the need for diagnosis, it changes how the underlying variation is conceptualised. Since there is no single objective biomarker in ADHD corresponding to blood pressure, the clinical threshold cannot be determined by symptom count alone: developmental history, persistence of symptoms, their presence across different settings and functional impairment should be evaluated together.

Lévy et al[2] showed, based on findings from large twin samples, that clinically significant ADHD symptoms are not genetically distinct from lower-level symptoms in the general

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population, but reflect quantitative differences in the same genetic effects. This finding supports the view that ADHD represents the extreme of behavioural traits that vary continuously across the population, rather than a distinct genetic category. Large-scale Genome-Wide Association Studies analyses have further demonstrated the polygenic architecture of ADHD.[3] A population-based, genome-wide meta-analysis showed substantial genetic overlap between continuous ADHD symptom scores and case–control ADHD, reporting an estimated genetic correlation of 0.96.[4] Molecular genetic findings also test this continuity more directly. Polygenic risk scores derived from clinical ADHD samples predict attention problems in population samples, indicating that traits in diagnosed and undiagnosed individuals partly share a common genetic predisposition.[5] A similar continuity is observed in clinical outcomes. In the general population, psychiatric comorbidity and functional impairment have been reported to increase stepwise as symptom count increases.[6]

Categorical analyses apply a clinically useful threshold, whereas dimensional analyses examine variation in trait severity across the entire distribution. Thus, they represent the underlying variation in different ways. These findings raise the question of whether neurocognitive and regulatory processes associated with ADHD also continue in a graded manner across both sides of the categorical diagnostic threshold.

A calibration perspective across the ADHD dimension

Calibration is not proposed as a single biomarker or neurobiological axis that determines an individual's position on the spectrum. Rather, it is an integrative perspective that conceptualises graded differences in the regulation of attention, arousal, motivation and cognitive control systems, and the context-dependent functional expression of these differences. From a spectrum perspective, the importance of this approach lies in considering together the regulatory differences accompanying a continuously distributed liability and the biological and functional expressions of these differences across changing contexts. Catecholaminergic modulation, arousal and state regulation, reward and motivation processes, executive control and coordination across large-scale neurocognitive networks can be considered partially overlapping processes that may contribute to this regulation.

A positron emission tomography study in adults with ADHD reported reduced tonic and increased phasic dopamine release in the right caudate.[7] This finding is consistent with altered dopaminergic regulation, but cannot be interpreted as evidence for a universal dopamine deficiency model. Pharmacological models also emphasise modulation of prefrontal dopaminergic and noradrenergic signalling.[8] From a calibration perspective, these findings can be considered possible neurochemical level examples of regulatory processes.

Regulatory differences can be considered not only at the neurochemical level but also at the level of large-scale neurocognitive network organisation. ADHD has been associated with changes in coordination between the default mode network and task-positive systems. Case–control neuroimaging studies have identified abnormalities across distributed networks rather than in a single ADHD-specific region.[9] Therefore, evidence for the dimensional continuity of neurobiological differences is particularly important. In a large adolescent sample, striatal connectivity was shown to vary with inattention and hyperactivity–impulsivity scores whereas categorical ADHD-control comparisons were not significant.[10] In medication-naïve adults with ADHD, connectivity patterns across different brain networks were shown to vary dimensionally with symptom severity, whereas no clear evidence was found for distinct neurobiological subtypes.[11] Similar associations between ADHD symptom severity and large-scale neurocognitive network organisation have also been demonstrated in a largely non-clinical adult sample.[12] From a calibration perspective, coordination among these networks may be considered one potential neural level through which attention and cognitive resources are directed in a context-appropriate manner between internal processes and external task demands.

The functional counterparts of these dimensional regulatory differences can also be observed in cognitive processes such as working memory, information processing and the efficiency of translating goals into action. Experimental studies in ADHD have shown that increased working memory demands can reduce information-processing efficiency.[13] These findings are consistent with the idea, from a calibration perspective, that regulatory efficiency may vary as task demands increase rather than reflect a fixed capacity deficit. In community-based data from young adults, higher ADHD symptom severity was associated with lower attention and response inhibition performance.[14]

Taken together, these findings suggest that at least some neural and cognitive characteristics associated with clinically diagnosed ADHD continue dimensionally below the diagnostic threshold. Therefore, it may be more appropriate to interpret these neurocognitive findings as potential neurocognitive expressions of variability in regulatory processes rather than as a categorical biomarker.

From dimensional liability to the clinical threshold

The most important clinical question for a dimensional model is: who should receive a diagnosis and treatment when there is a continuous distribution? Dimensionality does not mean clinical practice without thresholds. A person may show symptoms below the diagnostic threshold and still require support. By contrast, an ADHD diagnosis is a necessary clinical decision when symptoms are developmentally inappropriate, persistent, present across multiple contexts and associated with clinically significant functional impairment. Within this framework, functional impairment is the bridge between dimensional biology and categorical clinical decision-making. Two individuals with similar levels of liability may have different functional outcomes. A structured environment with rapid feedback and task demands that are well matched may reduce some difficulties, whereas conditions requiring prolonged monotonous attention, intensive self-organisation or delayed reward may make them more pronounced. Although these differences do not mean that the diagnosis is solely a product of the environment, they show that context can modify the functional expression of an existing neurodevelopmental liability.

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Treatment decisions should also not be based solely on an individual's presumed position on the spectrum. Current clinical guidelines recommend medication for adults when ADHD symptoms continue to cause significant functional impairment in at least one area of life, despite environmental modifications.[15] Therefore, position on the dimensional scale alone does not determine a specific type or intensity of treatment. Clinical decisions should be made by considering functional impairment, context, comorbidities and individual characteristics together. Environmental modifications and psychosocial interventions may reduce functional burden, whereas pharmacological treatments may be used to reduce clinically significant symptoms. Although dimensional measures alone do not determine which treatment should be selected, they may facilitate the monitoring of treatment response and functional impairment. The dimensional approach explains how ADHD-related traits are distributed in the population and why the underlying liability may not show a sharp natural boundary. Categorical diagnosis, in turn, provides the decision tool required for clinical responsibility, intervention and access to services at a given point.

Conceptual and clinical implications

Attention and regulatory traits associated with ADHD show continuity across the population rather than forming a qualitatively discrete distribution. Clinical ADHD represents the end of this distribution associated with functional impairment. The proposed calibration perspective is an integrative conceptual framework that explains how this dimensional liability may interact with changing task-based and environmental demands to produce different functional outcomes. Although findings at the levels of neurochemical regulation, neurocognitive network organisation and cognitive performance are consistent with this perspective, current evidence does not support reducing calibration to a single biological mechanism. The practical implication of the dimensional perspective is that categorical clinical decisions should be complemented by dimensional assessment of severity, context and functioning. Although this approach considers neuro-organisational difference an important component of human cognitive diversity, it does not eliminate the need for evidence-based pharmacological and psychosocial interventions when clinically significant functional impairment is present.

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Data availability

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