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Cognitive Enhancement, Prescription Stimulant Use and Health-Related Decision-Making: The Role of Methylphenidate in Healthy Individuals and Subthreshold ADHD

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Abstract

The use of methylphenidate beyond clinically diagnosed attention-deficit/hyperactivity disorder (ADHD) has become an important issue in health, educational, and occupational contexts, particularly in relation to cognitive enhancement, performance-related expectations, and the potential misuse of prescription stimulants. ADHD is a neurodevelopmental condition associated with impairments in attention, executive function, and working memory, while subthreshold ADHD symptoms may also be accompanied by measurable cognitive and functional difficulties. The aim of this narrative review was to compare the cognitive effects of methylphenidate in healthy individuals and in individuals with subthreshold ADHD symptoms, with particular attention to whether its action should be interpreted as cognitive enhancement or compensatory improvement. The review was based on a targeted selection of literature identified through PubMed, including randomized controlled trial evidence, meta-analyses, and review studies. Evidence from healthy individuals indicates that methylphenidate produces limited, domain-specific, and inconsistent cognitive effects, with no reliable improvement in attention, working memory, or processing speed. In contrast, individuals with ADHD demonstrate clinically meaningful improvements, particularly in attention and executive function. Subthreshold ADHD may represent an intermediate phenotype, characterized by measurable but less severe impairments, and may therefore show greater responsiveness to methylphenidate than healthy individuals, although direct evidence remains limited. Overall, the findings suggest that the effects of methylphenidate depend on

baseline cognitive functioning. These results have implications for health-related decision-making, responsible prescribing, and the evaluation of stimulant use outside strictly diagnosed ADHD populations, where the distinction between treatment, compensation, and enhancement is clinically and socially relevant.

Keywords: *methylphenidate; ADHD; subthreshold ADHD; cognitive enhancement; working memory; attention; processing speed*

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental condition characterized by persistent patterns of inattention, hyperactivity, and impulsivity [1]. Contemporary evidence suggests that ADHD may be conceptualized as part of a broader clinical spectrum, encompassing a range of symptom presentations and severity levels [1,2]. Within this framework, subthreshold or subclinical ADHD is defined in the literature as the presence of clinically relevant symptoms without meeting full diagnostic criteria. This condition has been associated with impairments in attention, executive function, and working memory [2].

In parallel, the use of pharmacological agents such as methylphenidate has expanded beyond strictly diagnosed ADHD populations. Methylphenidate, a central nervous system stimulant, is widely used in the treatment of ADHD and has been shown to improve attention and executive functioning in affected individuals [3]. The cognitive effects of methylphenidate are related to its action in the prefrontal cortex, a brain region critically involved in attention and executive function [4]. However, its use among individuals without a formal diagnosis has increased, particularly in academic and professional contexts, where it is used with the intention of enhancing cognitive performance [5,6].

The cognitive effects of methylphenidate in healthy individuals remain inconsistent. Evidence from meta-analyses and review studies suggests modest improvements in selected domains such as working memory, with limited or no effects on other cognitive functions, particularly in individuals with higher baseline cognitive performance [5,6]. Systematic review data indicate that the cognitive effects of methylphenidate in healthy individuals are generally limited and not consistently observed across different cognitive domains [7]. Experimental findings further indicate that the effects of methylphenidate may depend on baseline cognitive ability, with greater benefits observed in individuals with lower initial performance [8,9]. In contrast, individuals with ADHD demonstrate more robust and clinically meaningful improvements following methylphenidate administration, particularly in

attention, response inhibition, and executive function, as supported by large-scale meta-analytic evidence [3,10].

These differences raise important questions about whether the effects observed in individuals without a formal diagnosis reflect true cognitive enhancement, or rather an improvement toward typical levels of cognitive performance. Despite increasing interest in stimulant use beyond clinical populations, subthreshold ADHD appears to have received relatively limited attention in the literature as a distinct group [2]. This population may represent an intermediate phenotype between clinically diagnosed ADHD and healthy individuals, potentially exhibiting differential responsiveness to methylphenidate. Understanding the effects of methylphenidate in this group may help clarify whether its action is better conceptualized as cognitive enhancement, compensatory improvement, or a combination of these effects.

The distinction between enhancement and treatment carries important clinical and societal implications. The increasing off-label use of stimulant medications raises concerns regarding misuse and unrealistic expectations of performance enhancement [5,6]. At the same time, a better understanding of how methylphenidate affects cognition across different levels of baseline functioning may contribute to more precise diagnostic and therapeutic approaches. Therefore, the aim of this narrative review is to compare the cognitive effects of methylphenidate in healthy individuals and those presenting with subthreshold ADHD symptoms.

Material and methods

This study was conducted as a narrative review and did not involve original data collection. It was based on a targeted selection of scientific literature addressing the cognitive effects of methylphenidate in both healthy individuals and ADHD-related populations. PubMed was used as the primary database for literature search. The literature search focused on the following keywords: methylphenidate, ADHD, cognitive function, working memory, attention, and subthreshold ADHD. No strict time restriction was applied; however, priority was given to more recent studies where available.

Studies were selected based on their relevance to the research question and their contribution to understanding the cognitive effects of methylphenidate across populations of interest, including healthy individuals, individuals with diagnosed ADHD, and those presenting with subthreshold symptoms. Priority was given to randomized controlled trial evidence, meta-analyses, review articles, and large observational studies [11–13]. The

included literature comprised both experimental studies, primarily randomized, double-blind, placebo-controlled trials, and observational or review-based evidence [11–13]. This enabled comparison of findings across different study designs and populations.

Given the limited availability of direct experimental studies specifically addressing subthreshold ADHD, conclusions regarding this group were based on comparisons between findings from healthy individuals and those with diagnosed ADHD. This approach involved integrating findings from studies conducted in healthy individuals, clinically diagnosed ADHD populations, and broader literature on the dimensional nature of ADHD and its associated cognitive and behavioral characteristics [14–16]. The synthesis was conducted narratively, with a focus on identifying consistent patterns across studies and comparing the magnitude and nature of cognitive effects observed in different populations.

Effects in Healthy Individuals

Attention

Evidence regarding the effects of methylphenidate on attention in healthy individuals remains inconsistent and generally does not support a robust enhancement effect. In a randomized, double-blind, placebo-controlled trial conducted by Repantis et al., 48 healthy male participants aged 21–36 were assessed across multiple cognitive domains following administration of methylphenidate, modafinil, and caffeine. Cognitive testing was performed at time points corresponding to peak pharmacological activity, allowing for reliable assessment of short-term effects [11]. In this study, methylphenidate did not produce a significant improvement in sustained attention, as measured by performance in the psychomotor vigilance task (PVT). By contrast, caffeine was associated with a reduction in reaction time, indicating improved vigilance. Modafinil also did not demonstrate a statistically significant advantage over placebo in this domain. Notably, participants reported reduced subjective fatigue following methylphenidate administration despite the absence of measurable improvement in sustained attention [11].

Complementary findings have been reported in neuroimaging-based studies. Becker et al. examined both behavioral outcomes and resting-state functional connectivity following administration of the same stimulants in healthy individuals. The results showed modulation of connectivity between the frontoparietal network and the default mode network, which are involved in attention control and regulation of internally directed processes. These changes may indicate that stimulant-related effects can be detected at the level of neural network organization even when behavioral measures remain unchanged [12].

Evidence from review articles further supports a cautious interpretation of stimulant effects on attention in healthy individuals. Burguete et al. discussed ethical concerns suggesting that expectations of cognitive enhancement in healthy individuals are often not supported by consistent experimental findings [17]. Taken together, the available evidence suggests that methylphenidate does not produce a consistent or generalized improvement in attention in healthy individuals. While some studies indicate measurable neurophysiological changes or task-specific effects, these findings do not translate into stable or clinically meaningful improvement in attention [11,12,17].

Working memory

Evidence regarding the effects of methylphenidate on working memory in healthy individuals is inconsistent and does not support a robust enhancement effect. In a randomized, double-blind, placebo-controlled trial conducted by Repantis et al., working memory was assessed using the Backward Digit Span (BackSpan) task, which measures the ability to maintain and manipulate information in real time. In this study, methylphenidate did not significantly improve working memory performance compared to placebo. Similarly, neither modafinil nor caffeine produced a significant effect in this domain [11].

Importantly, the absence of significant differences was observed under well-controlled experimental conditions in a homogeneous group of healthy young men without psychiatric or neurological disorders and without the use of other psychoactive substances [11]. This strengthens the interpretation that methylphenidate does not produce reliable working memory enhancement in cognitively intact individuals. The same trial also demonstrated that methylphenidate improved certain aspects of declarative memory, particularly delayed recall after 24 hours [11]. This suggests that potential cognitive effects of methylphenidate are domain-specific and not uniformly distributed across different memory systems.

Similar findings have been reported in neuroimaging-based studies. Becker et al. examined both behavioral outcomes and resting-state functional connectivity following administration of stimulants in healthy individuals. While behavioral effects on working memory tasks were not consistently observed, stimulant administration was associated with modulation of connectivity in prefrontal, parietal, and temporal regions involved in memory and cognitive control [12]. These findings suggest that pharmacological effects may be detectable at the level of neural network organization even in the absence of clear behavioral improvement. Taken together, the available evidence suggests that methylphenidate does not

produce consistent or clinically meaningful improvements in working memory in healthy individuals [11,12].

Processing speed

Evidence regarding the effects of methylphenidate on processing speed in healthy individuals is limited and inconsistent [11,12]. In a randomized, double-blind, placebo-controlled trial conducted by Repantis et al., no significant improvement in processing speed was observed following administration of methylphenidate, modafinil, or caffeine, as measured by the Zahlen-Verbindungs-Test (ZVT). The authors noted that the study had a pilot design, which may have limited its ability to detect small differences between substances [11].

Importantly, earlier findings cited in the same study suggested that improvements in perceptual processing speed may occur primarily in individuals with lower baseline performance. However, such an effect was not observed in the study sample, which consisted of cognitively intact healthy participants [11]. This suggests that baseline performance may influence the detectability of pharmacological effects in this domain. Consistent with these findings, Becker et al. reported that evidence for the effects of methylphenidate on processing speed in healthy individuals is less robust than that for memory or selected aspects of attention. The authors also noted that improvements in processing speed are more frequently observed with caffeine, although such effects may reflect increased arousal rather than specific cognitive changes [12]. Taken together, the available evidence suggests that methylphenidate does not produce consistent or clearly interpretable improvements in processing speed in healthy individuals [11,12].

Effects in Subthreshold ADHD

Subthreshold ADHD is a clinically relevant condition characterized by the presence of ADHD symptoms that do not meet full diagnostic criteria but are nevertheless associated with measurable functional impairment. Available evidence indicates that individuals with subthreshold symptoms may experience difficulties in attention, executive functioning, and academic or social performance, as well as an increased risk of psychiatric comorbidities [14–16]. These findings support the view that subthreshold ADHD should not be considered equivalent to typical development.

A key conceptual framework for understanding subthreshold ADHD is the dimensional model of psychopathology. Ogundele and Morton emphasize that ADHD-related traits are distributed along a continuum rather than confined to a categorical distinction between

disorder and health. Within this framework, subthreshold ADHD can be understood as a level of symptom severity that is lower than in diagnosed ADHD but still associated with clinically meaningful impairment [14].

Longitudinal evidence further supports this interpretation. In a cohort study by Schiavone et al., individuals with subthreshold ADHD symptoms in childhood demonstrated intermediate outcomes in adulthood when compared with individuals with diagnosed ADHD and those without symptoms. Specifically, they showed impairments in selected cognitive domains, including executive function and processing speed, although these impairments were less pronounced than in individuals with full ADHD [15]. These findings indicate that subthreshold ADHD is associated with persistent, measurable differences in cognitive functioning over time.

This pattern can be interpreted in the context of the pharmacological mechanism of methylphenidate, which increases dopaminergic and noradrenergic signaling in brain regions involved in attention and executive function. In individuals with ADHD, this modulation is associated with measurable improvement in core symptoms, including inattention and executive dysfunction [18–20]. In contrast, in cognitively intact individuals, where baseline functioning is relatively preserved, the potential for measurable improvement may be more limited. Within this framework, individuals with subthreshold ADHD may represent an intermediate case, in which baseline deficits are present but less pronounced than in diagnosed ADHD.

Additional evidence highlights the broader clinical relevance of subthreshold ADHD. Ogundele and Morton report that children and adolescents with subthreshold symptoms are more likely to experience family dysfunction, academic difficulties, and psychiatric comorbidities compared with individuals without symptoms [14]. Similarly, Powell et al. found that greater ADHD symptom severity in adults was associated with earlier onset of depression, higher recurrence rates, and more persistent symptoms, suggesting that subclinical ADHD traits may contribute to more complex clinical presentations [16].

When these findings are considered alongside evidence from studies in healthy individuals and those with diagnosed ADHD, a consistent pattern emerges. In healthy individuals, the cognitive effects of methylphenidate are limited, domain-specific, and often inconsistent [11,12]. In contrast, in individuals with diagnosed ADHD, stimulant treatment is associated with clinically meaningful improvements in core symptoms such as inattention and executive dysfunction [13,18–20]. This difference suggests that the magnitude and clinical relevance of

treatment effects may depend on baseline levels of cognitive and behavioral impairment. Based on this comparison, it may be hypothesized that individuals with subthreshold ADHD could show greater responsiveness to methylphenidate than healthy individuals, as their baseline level of functioning is likely to be less optimal. At the same time, the expected magnitude of improvement would likely be smaller than that observed in individuals with fully diagnosed ADHD. However, direct experimental evidence in subthreshold ADHD populations remains limited, and conclusions regarding treatment effects in this group must therefore be interpreted with caution [14].

Comparison

The key difference between healthy individuals and those with subthreshold ADHD lies in the interpretation of the cognitive effects of methylphenidate. In healthy populations, its effects are typically discussed in the context of cognitive enhancement, that is, changes in performance in individuals whose baseline functioning is already within the normal range. In contrast, in subthreshold ADHD, where measurable cognitive and functional difficulties are present, the effects of methylphenidate may be more appropriately interpreted in terms of partial compensation for existing deficits [14,18–20].

In studies of healthy individuals, the effects of methylphenidate are generally limited, selective, and inconsistent across cognitive domains. In a randomized trial by Repantis et al., methylphenidate did not significantly improve sustained attention or working memory, while its measurable effects were restricted to delayed recall of declarative memory and reductions in subjective fatigue [11]. Similarly, Becker et al. reported changes in functional connectivity, reflected as altered interactions between brain networks involved in attention and cognitive control, but did not observe consistent improvements across behavioral measures of cognitive performance [12]. These findings suggest that, in healthy individuals, the effects of methylphenidate are task-specific rather than indicative of broad cognitive enhancement [11,12].

This interpretation is further supported by evidence indicating that the magnitude of observed effects may depend on baseline performance. In healthy volunteers, greater improvements have been reported more frequently in individuals with lower baseline cognitive performance, whereas effects are less pronounced in those functioning at a higher level prior to intervention [8,11]. This pattern suggests that the measurable impact of methylphenidate may be influenced by the initial level of cognitive functioning.

In subthreshold ADHD, where baseline cognitive performance may be less optimal than in healthy individuals, this relationship may have different implications for the interpretation of drug effects. The presence of subclinical symptoms suggests that some degree of functional impairment is already present, although less pronounced than in diagnosed ADHD [14–16]. Within this context, the effects of methylphenidate may be more consistent with partial improvement of existing deficits rather than enhancement of already intact functions.

At the same time, these interpretations must be treated with methodological caution. Direct experimental studies evaluating the effects of methylphenidate specifically in individuals with subthreshold ADHD are limited. Therefore, the present comparison is based on indirect inference from studies in healthy individuals, clinically diagnosed ADHD populations, and research on the dimensional nature of ADHD symptoms [11,14–16,18–20]. While this approach provides a consistent interpretative framework, it does not allow for definitive conclusions regarding treatment effects in subthreshold ADHD.

Discussion

The present findings suggest that the differences observed between healthy individuals and those with subthreshold ADHD are not solely related to the magnitude of the pharmacological effect of methylphenidate, but also reflect differences in baseline cognitive functioning and clinical relevance of symptoms [1,3,11,12]. In healthy individuals, the effects of methylphenidate are generally limited, domain-specific, and inconsistent, with improvements typically restricted to selected cognitive measures rather than broad enhancement of overall cognitive performance [5,6,9]. This is consistent with previous systematic reviews showing that stimulant-related cognitive enhancement in healthy individuals is typically modest and variable [7].

In contrast, individuals with subthreshold ADHD present with measurable, although less severe, cognitive and functional difficulties, which may influence the interpretation of drug effects [2,14–16]. This distinction is consistent with the dimensional model of ADHD, in which subthreshold symptoms represent a less severe but clinically relevant manifestation within the same spectrum of attentional and executive dysfunction [1,2].

Longitudinal data further support this interpretation. Schiavone et al. demonstrated that individuals with subclinical ADHD symptoms show intermediate cognitive outcomes between diagnosed ADHD and healthy controls in adulthood [15]. Similarly, Ogundele and Morton highlighted that subthreshold cases are associated with increased functional and psychiatric risk, despite not meeting full diagnostic criteria [14]. Powell et al. further showed that

elevated ADHD symptoms in adults with recurrent depression are associated with greater clinical severity and chronicity, suggesting that subclinical ADHD traits may influence the course of other psychiatric conditions [16].

Taken together, these findings suggest that the effects of methylphenidate may depend on the level of baseline impairment. In individuals with more pronounced deficits, including those with subthreshold ADHD, the drug may act through partial normalization of cognitive function, whereas in healthy individuals, its effects are more limited and less predictable [8,11,12,15,16]. However, this interpretation should be considered cautiously, as direct experimental evidence in subthreshold ADHD populations remains limited.

From a clinical perspective, these observations highlight the importance of assessing functional impairment rather than relying solely on diagnostic thresholds when considering potential interventions [1,14]. Treatment decisions should take into account symptom severity, impact on daily functioning, comorbidities, and the overall balance between potential benefits and risks [1,3,13–16]. At the same time, potential adverse effects of methylphenidate, including cardiovascular and psychiatric risks, should be carefully evaluated prior to initiating treatment, particularly in populations without a clear diagnostic indication [19,20].

Several limitations should be acknowledged. First, the lack of randomized controlled trials specifically targeting subthreshold ADHD limits the ability to draw definitive conclusions regarding treatment effects in this population [2,14–16]. Second, the heterogeneity of definitions and diagnostic criteria for subthreshold ADHD across studies complicates direct comparisons and generalization of findings [2,14]. Third, studies conducted in healthy individuals often involve small, homogeneous samples and short-term administration, which may limit external validity [5,6,9]. Overall, the available evidence supports a cautious interpretation of methylphenidate effects across different populations, emphasizing the role of baseline functioning in determining both the magnitude and clinical relevance of observed outcomes.

Conclusion

The available evidence suggests that methylphenidate appears to have different effects in healthy individuals compared to those with subthreshold ADHD symptoms. In healthy populations, its effects are generally limited and domain-specific, whereas in individuals with subthreshold ADHD, they may be more consistent with partial compensation for existing cognitive deficits [11,12,14–16]. Subthreshold ADHD is not a clinically neutral condition, as it is associated with persistent functional impairment, cognitive burden, and an increased risk

of developing other mental disorders [2,14–16]. This suggests that individuals with subthreshold ADHD may be more appropriately viewed as representing a milder form within the ADHD spectrum rather than as part of the cognitively typical population [1,2].

However, the precise effects of methylphenidate in subthreshold ADHD remain unclear, including which symptoms respond most consistently to treatment and how to balance potential benefits and risks over the long term [14–16,19,20]. Future research should focus on individuals with subthreshold ADHD using consistent diagnostic criteria and should evaluate not only cognitive test outcomes but also functional outcomes, including academic, occupational, emotional, and social domains, as well as the long-term safety of pharmacological interventions [14–16,19,20].

Disclosure Section

Author's contribution:

Conceptualization: Mateusz Chmiela

Methodology: Mateusz Chmiela, Anna Korzeniowska

Literature search: Mateusz Chmiela, Anna Czesyk, Maciej Słysz

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AI Statement

AI was utilized for two specific purposes in this research: text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies, and assistance in refining the academic English language of the manuscript, ensuring clarity,

consistency, and adherence to scientific writing standards. AI tools were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

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