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DSM-5 Conceptualization of Attention-Deficit/Hyperactivity Disorder and the Prescription of Non-Medical Psychostimulants: An Exploration of a Bioethical Dilemma

Conceptualización del Trastorno de Déficit Atencional e Hiperactividad del DSM-5 y la prescripción de psicoestimulantes de uso no médico: una exploración a un dilema bioético

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Abstract

This article analyzes the historical evolution of ADHD and its close relationship with the use of psychostimulants, showing how diagnostic changes, clinical evidence, and social pressures have shaped their contemporary understanding. Although stimulants reduce symptoms and risks associated with ADHD, their neurobiological basis remains uncertain, and diagnostic expansion has led to overdiagnosis and medicalization. Non-medical, especially in educational and work context, highlights a central bioethical dilemma: the growing blurring between treatment and performance optimization. Epistemic critiques of clinical trials, the influence of the pharmaceutical industry, the identity effects of diagnosis, and the tensions between autonomy, justice, and care are examined. The article proposes strengthening institutional ethics that limit commercial interests, ensure transparency in diagnostic construction, and promote a broader understanding of attention disorders, integrating biological, social, and cultural dimensions.

Keywords: medicalization, psychostimulants, ADHD, bioethical dilemma.

1. Introduction

From its earliest descriptions in the 20th century to its current formulation as a neurodevelopmental disorder in the DSM-5, Attention Deficit Hyperactivity Disorder (ADHD) has undergone multiple transformations in its conceptualization that have reshaped the medical, social, and ethical understanding of human behavior. At the same time, pharmacotherapy with psychostimulants, especially methylphenidate and amphetamine derivatives, has taken on a central role in the management of ADHD, establishing itself as the first line of treatment due to its effectiveness in reducing symptoms and functional risks. However, the use of these drugs has expanded beyond the clinical setting, particularly among adolescents and adults without a medical diagnosis who seek to improve their academic or work performance. This trend has reignited discussions about

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medicalization, overdiagnosis, the influence of the pharmaceutical industry, and the boundaries between treatment and optimization.

In this context, a relevant bioethical dilemma arises: how to assess the legitimacy of the therapeutic and non-therapeutic use of psychostimulants when the scientific basis of ADHD remains controversial, diagnostic processes become more inclusive, and social pressures toward productivity shape the demand for these substances?

This article reviews the historical evolution of the concept of ADHD, analyzes the available clinical evidence, and examines the epistemic and ethical critiques that question contemporary forms of prescription and non-medical use. Based on this, a bioethical reflection is proposed that allows us to distinguish between therapeutic intervention and performance optimization in a scenario marked by the medicalization of everyday life and commercial interests that influence clinical practice.

2. State of the art

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder defined by problematic levels of inattention, disorganization, and/or hyperactivity-impulsivity, which often persists into adulthood, with consequences for social, academic, and occupational functioning (1).

The prevalence of ADHD is estimated to be close to 5.3% (2). Treatment options include pharmacological and non-pharmacological measures. Among pharmacological measures, the use of psychostimulants (mainly methylphenidate and amphetamine derivatives) is considered a first-line treatment. Psychostimulants have a protective effect in the treatment of ADHD, preventing the development of substance use disorders (addictions) (2).

In population-based registry studies (intraindividual designs such as *self-controlled* case series), the use of medication for ADHD was

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consistently associated with lower rates of self-harm (IRR≈0.77–0.85), unintentional injuries (IRR≈0.87–0.93), traffic accidents (IRR≈0.71–0.87), and criminal behavior (IRR≈0.73–0.84) throughout 2006–2020 in Sweden. Complementarily, in a comparative effectiveness study in a national cohort of patients with borderline personality disorder, treatment with ADHD medications (mainly psychostimulants) was associated with a reduced risk of suicide attempt or death (HR≈0.82), while other drug classes showed no benefit or were linked to increased risk (3). This pooled evidence has been discussed in editorials and reviews that emphasize that, when used appropriately, psychostimulants can produce "real-world outcomes"[1] —that is, reduce serious harm and improve functional opportunities— although the extension of prescribing to less severe populations raises the need to periodically evaluate the risk/benefit ratio and to complement pharmacotherapy with psychosocial interventions (3–5).

Prescriptions for methylphenidate and amphetamine derivatives increased by 35.5% between 2008 and 2012 in the US. In 2016, the number of individuals over the age of 12 in the US who started NMU stimulants was 1.4 million, more than the number of people who started methamphetamine and cocaine combined (2).

However, the increase in prescriptions among the adult population (6) raises the controversy of possible overdiagnosis in this population and even questions ADHD as a nosological entity.

In parallel with this, the *non-medical use of prescription stimulants* (NMU) has sparked a debate about the ethical implications of prescribing them in educational contexts, questioning their use and their consequences for the physical and mental health of the population. Faraone *et al.* (2020) review the state of the art of UNM of psycho-

[1] Real-world outcomes refer to events and outcomes in everyday life that are socially and clinically relevant, such as self-harm and suicide, accidental injuries, traffic collisions, criminal convictions, substance use, and other functional consequences, and are typically recorded in administrative databases (hospital, police, or court records) or in large-scale observational studies.

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stimulants. According to this review, studies to date are only descriptive, and with the available evidence, a "risk profile" and a "motivation profile" can be constructed. Among the limitations of this review are the use of self-report questionnaires and little consensus on definitions related to abuse, use, and diversion, among others.

Regarding the epidemiology of PSU, 50-90% obtained the drugs through family or friends, 4-35% had a prescription, and 20% obtained the prescription by feigning symptoms of ADHD. In the school population, its greatest use is found among 8th to 10th graders. In addition, PSU of psychostimulants is associated with the concomitant presence of substance use disorder. Of that group, 64% would have alcohol use disorder (AUD), 43% marijuana use disorder, 10% cocaine use disorder, 8.8% hypnotic/sedative use disorder, and 8.4% hallucinogen use disorder. In one of the studies, it is estimated that one-third of PSU users met the criteria for stimulant use disorder and half met the criteria for alcohol use disorder (7).

Academic reasons were found to be the motivation for stimulant MUD. Based on the review conducted by Faraone *et al.* (2020), it is estimated that this group would be 50-89% of university students, with 40% reporting that they seek to be more productive (8). In another survey, between 38-57% sought to improve academic and work performance (9). Another group reported using stimulants for recreational activities or to *"get high*," ranging from 2-31%. Another group would lose weight, and this group would have a higher prevalence of a history of concomitant eating disorders.[2] Other motivations for use include: compensating for the effects of alcohol, helping with socialization, self-treatment of ADHD symptoms, and increased alertness and energy without necessarily being associated with cognitive improvement.

What are the consequences of non-medical use of psychostimulants? With regard to adverse effects, similar effects to those of prescribed use are described, namely: headache, abdominal pain, increased

[2] The treatment of eating disorders corresponds to a group of pathologies characterized by excessive concern about body image associated with eating disorders.

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irritability, increased emotional lability, decreased appetite, insomnia, tachycardia, etc.[3] In terms of academic consequences, the UNM of psychostimulants would not imply improved academic performance in those who do not have a diagnosis of ADHD (10–15).

In the same review conducted by Faraone *et al.* (2020), a survey of 826 pediatricians was reviewed, where only 46-44% educated about the clinical and health risks of stimulant use during college; and 40% did not provide education to students starting college. Furthermore, the view of UNM as a breach of academic integrity has mixed perspectives on US university campuses. In this review, the authors raise the importance of implementing preventive strategies focused on the non-patient at-risk population (secondary and higher education students); eradicating myths about its use and abuse; and referring the at-risk population or those with psychiatric comorbidity.

The first-line intervention for psychostimulant UNM is psychoeducation. Among the myths are: 1) challenging unrealistic expectations of improved performance and 2) overestimating the use of UNM among college peers ("everyone uses it to study").

3. The concept of ADHD and the use of psychostimulants

The history of psychostimulant use is closely linked to the history of ADHD

The concept has existed for a long time, under various names such as Moral Control Deficit, proposed in 1902, and Brain Damage Syndrome or Organic Behavior Syndrome, proposed by various authors (16,17). However, between 1937 and 1941, the first publications on the treatment of children with hyperactivity appeared. With an anecdotal positive response to the use of amphetamines by Charles Bradley, which was later replicated in subsequent studies, a positive

[3] The use of psychostimulants in the population with ADHD has been shown to have effects on symptoms associated with the condition, such as impulsivity and emotional dysregulation.

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relationship between behavioral improvement and performance was described (18).

Subsequently, the concept of Hyperkinetic Disorder proposed by Laufer gained increasing acceptance among specialists. In his article, he acknowledges Bradley's achievement, not only in finding a treatment, but also in that his study allowed the "neurophysiological and clinical facets of the condition" to be characterized. Based on the pharmacological response, etiological hypotheses are proposed and the condition is characterized as a "treatable" syndrome (19,20). This syndrome inspired the development of "hyperkinetic disorder" in children in the DSM-II in 1968. In parallel, methylphenidate (Ritalin) was developed in 1955 and approved by the FDA in 1961 (21).

In the 1970s, two models parallel to the DSM-II model emerged, which were also influenced by the use of psychostimulants. These are the Minimum Brain Damage Model and the Attention Deficit Model. Wender proposed the concept of *Minimum Brain Damage* (hereinafter: "MBD"), consisting of two cardinal symptoms: attention disorders and reinforcement system disorders, from which secondary symptoms derive (22). The etiological relevance of MBD would go beyond childhood disorder. The etiological hypothesis proposed (namely, atomic injury and/or neurochemical alterations) may serve as a precursor to various forms of adult psychopathology, including what he calls a "character disorder" (namely, impulsive and immature forms), sociopathy, and even schizophrenia. Among the etiological hypotheses established by Wender in his article, MBD would be the result of an alteration in gene expression that leads to metabolic alterations in the monoaminergic system. Secondly, alteration of the monoamine system would lead to two main cardinal symptoms: altered arousal levels[4] and decreased sensitivity of reinforcement

[4] Arousal: the term arousal, used by Wender to refer to attention, has had different meanings. On the one hand, there is behavioral arousal, that is, a person's level of activity. On the other hand, physiological arousal refers to the level of "activation" or alertness, mediated by brain structures such as the ascending reticular activating system, which is responsible for wakefulness. For educational purposes, we will use it as a synonym for "attention threshold." For more on this debate, see (23).

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systems (to reward). Although Wender argues that response to treatment should not be the appropriate way to determine etiology, the response to treatment of these patients suggests that there would be specific neurochemical alterations. Furthermore, he argues that in the case of these patients, there would be no "euphoric" effects, as hyperactivity is reduced and "conditioned avoidance behavior" is enhanced (22). Furthermore, based on the response to treatment in children, this hypothesis is extrapolated and studies are conducted in adults with similar symptoms (24). When compared to the current characterization of ADHD, this conceptualization has a number of problems. Among them, we find a large group of symptoms, with little specificity, which can currently be transferred to other conditions such as oppositional defiant disorder (hereinafter: "ODD"). Although this term fell into disuse due to its diagnostic ambiguity, it maintained the practice of treatment with stimulants as the central therapeutic axis (22,25).

At the same time, Douglas's model of "Attention Deficit" emerged. For Douglas and his group, the inability to maintain sustained attention and difficulties in controlling impulsivity can be considered the most common deficits in the hyperactive group (26). According to his model, he argues that these patients in childhood "manage to self-regulate," so the use of medication should be restricted to those cases where the symptoms are "extremely debilitating" (26). This was due to the apprehensions and doubts at the time about the use of stimulants (namely, their effects on children, the lack of clarity about their mechanism of action, the risk of addiction, and other adverse effects).

During the 1970s, there was a boom in the importance of environmental factors in scientific literature and popular media, where the role of upbringing and dietary factors such as preservatives as possible causal etiologies gained relevance (27). This, coupled with the rise of pharmacological therapy with psychostimulants, explains what Douglas calls in his review "the politicization of pharmacological therapy" (26), going so far as to propose "the myth of the

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disorder," for which they attributed responsibility to the current educational system (28).

In the late 1970s, Peter and Roslyn Glow proposed a model in which Impulsive Hyperkinetic Disorder is conceived as a disorder of intrinsic motivation. Intrinsic motivation is defined as interest in the environment, motivated by curiosity and an intrinsic need to interact with, understand, and control one's environment. They describe it as a motivation inherent in information processing (29).

Based on this model, given their lack of intrinsic motivation toward the external environment, individuals would be less able to analyze the contingencies between behavior and environmental events, with a lower conception of themselves as the cause, which would lead to social and emotional immaturity. Within this model, an explanation is proposed regarding the use of psychostimulants as treatment and improvement in performance (29). The scientific-academic discussion between Douglas' conceptualization and Wender's conceptualization will influence the discussion of what the future DSM-III will be.

The transition from DSM-II to DSM-III involved significant changes to the structure and objectives of the text. DSM-III proposed "to have a common language through which to communicate about disorders (...) The planning of a treatment program must begin with an accurate diagnostic assessment. In addition, its development was different: previous drafts of this version were widely disseminated for critical review and use by clinicians and researchers. This is stated verbatim at the end of the introductory paragraph: Thus, the final version of DSM-III is only a snapshot in a continuing process aimed at understanding mental disorders more accurately (30).

The transition from DSM-II's "hyperkinetic reaction of childhood" to Attention Deficit Disorder (ADD) in DSM-III is notable for the name change, as "attention difficulties are prominent and always present in children with this diagnosis, and hyperactivity persists into adolescence" (30).

In DSM-III, ADD is considered a disorder that begins in childhood and a type of "behavioral disorder." Three subtypes are distin-

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guished: 1) ADD with hyperactivity (where criteria for the *clusters* of inattention, impulsivity, and hyperactivity must be met); 2) ADD without hyperactivity (where the criteria for the inattention and impulsivity *clusters* are met); and 3) residual-type ADD (someone who met the criteria for ADD with hyperactivity in the past but no longer meets the criteria for this cluster) (30). This last type is relevant, as it is recognized as a prognostic course that persists in adolescence and/or adulthood.

The publication of DSM-III in 1980 marked a paradigm shift by adopting a descriptive and behavioral approach. In this new framework, ADHD was formalized as a specific clinical diagnosis, which facilitated its study through controlled trials, systematically demonstrated the efficacy of stimulants, and exponentially increased the use of medications such as methylphenidate and amphetamines (31–33). In Conners' words, it was this shift that allowed for "a true scientific union with genetics, neuroscience, and therapeutic trials" (31).

With the revision of the Manual in 1987 (DSM-III-R), the disorder was redefined under the term *Attention Deficit Hyperactivity Disorder*, and the clusters were unified into a single set of criteria, which was much more descriptive in nature. This change led to an increase in the number of people eligible for pharmacological treatment, widely legitimized the use of stimulant drugs, and opened the door to massive clinical trials on methylphenidate and amphetamines (31,34,35).

Subsequently, in 1992, the Multimodal Treatment of ADHD Study (MTA Study) was developed, funded by the US National Institute of Mental Health (NIMH) in collaboration with the Department of Education. This study showed that the group receiving combined treatment achieved better results in reducing ADHD symptoms compared to those receiving only pharmacotherapy or only behavioral therapy. In turn, the group treated exclusively with psychostimulants outperformed the group that received only behavioral treatment, especially in the dimensions of hyperactivity and impulsivity (36). Furthermore, it was shown that the structure and

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quality of the pharmacological treatment administered in the study (supervised, adjusted, with rigorous clinical follow-up) was significantly more effective than the typical medication received by children treated in the community, underscoring the importance of standardized therapeutic protocols. The MTA Study was key in establishing that the use of psychostimulants such as methylphenidate was not only clinically effective but also part of the optimal treatment approach when integrated with psychosocial interventions. These findings influenced international clinical guidelines and public policies on child mental health. The empirical validation of the multimodal therapeutic model, together with the confirmation of the significant effects of psychostimulants, consolidated the role of pharmacotherapy as a central component of ADHD treatment, especially in cases of greater symptomatic severity (31,36).

The DSM-IV (1994) further refined the diagnosis by introducing subtypes: ADHD with predominantly inattentive symptoms, ADHD with predominantly hyperactive-impulsive symptoms, and combined ADHD. It describes that symptoms may be exacerbated or attenuated (or even absent). Symptoms worsen in situations that require sustained attention or mental effort, which lack intrinsic interest or novelty (situational variability). They may be attenuated when under strict control, in a new situation, in activities of interest, in a one-on-one personal relationship, or with frequent rewards. Symptoms tend to occur more frequently in group situations. However, as a categorical diagnosis in clinical practice, the DSM-IV showed limitations: relatively rigid criteria, but with poor etiological anchoring, and a persistent separation—by age of onset and course—that tended to classify ADHD as primarily childhood, even when clinically observed sequelae in adolescence and adulthood (37).

This diagnostic refinement allowed for more precise adjustment of pharmacological treatments according to the patient's clinical profile. The new system was also key to selecting more homogeneous participants in clinical trials, which allowed for more robust demonstration of the differential efficacy of psychostimulants (31,32). In

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addition, the DSM-IV was accompanied by a growing body of neuroscientific evidence that indirectly validated the efficacy of stimulants by showing minimal but significant structural alterations in the brains of people with ADHD (38,39).

4. The peak in diagnoses and the current situation of ADHD

One explanation for the increase in ADHD diagnoses has to do with an alignment of incentives associated with growing knowledge about ADHD and stimulants. In the early 1990s, the *Supplemental Security Income* (SSI) program was modified in the United States to allow children from low-income families with mental disorders (including ADHD) to be eligible, which led to an almost threefold increase in new ADHD diagnoses in the first half of the 1990s (21). In addition, in 1991, the U.S. Department of Education made reforms to the *Individuals with Disabilities Education Act* (IDEA), which classified ADHD as a disability protected by this law, expanding eligibility for school accommodations and raising awareness among parents and educators about the existence of services for diagnosed children. This increased the search for diagnoses (21).

However, some theorists suggest that school accountability and education funding systems have created incentives for teachers and administrators to promote the identification of ADHD. For example, some studies show that in states where schools receive funding tied to standardized test results, ADHD diagnosis rates increase significantly shortly after such policies are implemented. In other words, when schools are given financial incentives to improve student success, students are more likely to be diagnosed with ADHD and medicated (40,41).

A crucial moment in the expansion of stimulant use occurred with the passage of the FDA Modernization Act of 1997, which extended patent exclusivity rights for new drugs. It included key

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measures that facilitated the research and marketing of new drugs in children. In particular, it introduced "pediatric exclusivity": companies that conduct clinical trials in pediatric populations receive a six-month extension of their product's patent. This created a direct economic incentive to research formulations in children and create new formulations of existing drugs (42).

This significantly encouraged the pharmaceutical industry to invest in new long-acting formulations of methylphenidate and amphetamines, as well as in modifications of previous compounds and the reuse of drugs that had failed in other clinical contexts (21). This legislation marked a notable increase in scientific publications related to these drugs, following a period of relative stagnation (21).

Regarding the role of the pharmaceutical industry, an analysis between 2014-2018 found that the industry spent more than $20 million on payments to physicians related to ADHD stimulants. The authors conclude that "pharmaceutical industry marketing may be contributing in part to the increase in stimulant prescriptions" (43).

Several converging lines of evidence and clinical-methodological reflection formed the basis for reconsidering the definition and scope of ADHD between DSM-IV and DSM-5. On the one hand, clinical and epidemiological studies suggested that a significant proportion of individuals maintain clinically relevant symptoms into adolescence and adulthood. This accumulation of evidence challenged the idea of an exclusive childhood disorder and called for diagnostic criteria that considered the continuum of development and the persistence of dysfunction. On the other hand, the concept of "neurodevelopmental disorders" began to emerge in the literature. This was due to growing evidence of phenotypic and genetic overlap between ADHD, ASD, and intellectual disability (44,45).

The transition from DSM-IV to DSM-5 was driven by converging scientific, clinical, and social pressures demanding a more biologically based and developmentally grounded explanation of the phenomenon of attention deficit hyperactivity disorder. Longitudinal clinical studies conducted during the 1990s and 2000s documented

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that a considerable proportion of children diagnosed with ADHD continued to exhibit clinically significant symptoms and functional impairment during adolescence and adulthood, calling into question the view of ADHD as a condition limited primarily to childhood (46,47). Simultaneously, advances in genetics and neuroimaging produced strong evidence of heritability and consistent differences in the frontostriatal neural circuits, supporting a neurodevelopmental formulation of the disorder (2,44). At the same time, social criticism of possible overdiagnosis, the medicalization of childhood behaviors, and the emergence of the neurodiversity movement urged caution against an uncritical expansion of diagnostic boundaries (48,49). Together, these empirical advances and socio-ethical debates prompted the DSM-5 to reconceptualize ADHD within a neurodevelopmental framework, while seeking a balance between validity, clinical utility, and protection against excessive pathologization (1).

Currently, ADHD is understood in the DSM-5 as a neurodevelopmental disorder. The DSM-5 defines a neurodevelopmental disorder as one that begins in childhood and continues throughout life, either meeting diagnostic criteria or in a milder form (1). In DSM-5, the diagnosis can be made in adulthood, provided that the symptoms must be present before the age of 12 to consider the diagnosis (American Psychiatric Association, 2013, pp. 31–60).

From a developmental perspective, neurodevelopmental disorders are disorders that increase the risk of other mental health conditions (45,50). The DSM-5 further expanded the scope of diagnosis by officially recognizing ADHD in adults, which established a new target population for pharmacological treatments. This inclusion was the culmination of longitudinal studies of the symptomatic continuity of symptoms into adulthood and the efficacy of psychostimulants in this population (2,51). The prevalence review by Polanczyk et al. has shown that these modifications partly explain the observed increase in prevalence (1,52).

In summary, the DSM-5 retained the clinical essence of the DSM-IV but expanded its diagnostic criteria, facilitating the inclusion of adolescents and adults (lowering the symptom threshold)

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and recognizing more contexts of the patient's life (severity and environment specifiers) (1).

Finally, some considerations on cognitive *enhancement*. In earlier times, plants containing caffeine and nicotine (coffee, tea, cocoa, tobacco) were predominantly used to increase alertness and resistance to fatigue, to which were later added early synthetic stimulants studied in animal models as learning enhancers (53–55). In the 1960s and 1970s, the concept of nootropics was born with piracetam, and cholinergic and other pro-cognitive drugs for dementia became established, while in the 1980s and 1990s, the situational use of beta blockers for performance under stress was explored, and the pro-cognitive role and limits of caffeine and nicotine as enhancers of attention and alertness were better characterized (56–58). Between 1990 and 2010, the spotlight shifted to psychostimulants: methylphenidate, amphetamines, and modafinil (the latter indicated for narcolepsy) began to be used in healthy subjects as "smart drugs," with small improvements and specific domains (working memory, attention, executive functions), but with a clear discrepancy between the subjective feeling of improvement and objective change in cognitive tests (53,59,60).

5. Discussion: the bioethical issue

On the one hand, the introduction of psychostimulants influenced the transition from a disorder focused on "hyperkinesis or increased movement" to one centered on "inattention," with the management of the latter being a way to secondarily control the former. On the other hand, the treatment-diagnosis relationship is raised, which is evident in Douglas' model, where he limits the use of drugs to the most severe cases.

The history of the concept and its historical-etiological description has fluctuated between two poles: from more biological to more socio-environmental proposals to explain the origin of ADHD; between hyperactivity and inattention; between diagnosis based on

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"different from the average" versus "diagnosis of the most severe case"; between a course that lasts until childhood and a condition that persists into adulthood.

Despite the clinical consolidation of the use of psychostimulants, the approach has been criticized. Some studies have pointed to overprescription, especially in educational settings, as well as the influence of the pharmaceutical industry in the expansion of the diagnosis (61). In addition, there is an ongoing debate about the long-term effects of continued use of these medications and the need for more integrative multimodal approaches (34).

In the therapeutic area, psychostimulants reduce the core symptoms of ADD (62). This is reflected in clinical guidelines, where the goal of pharmacological treatment is symptom reduction, in contrast to non-pharmacological measures that improve quality of life and functionality (63). It also helps reduce anxiety secondary to core symptoms (64) and promotes medium-term improvements in academic performance, risk reduction, and family functioning (65). In the long term, there has been a reduction in suicidal behavior and the development of substance use disorder (SUD) in people with ADHD (66). It has also been associated with a reduction in crime and traffic accidents in the ADHD population (67,68).

Psychostimulants are not without adverse effects. A meta-analysis on cardiovascular risk found that individuals treated with methylphenidate had an 87% higher probability of having a higher rate of cardiovascular events in the 6 months after starting treatment (69). In addition, patients with atherosclerotic disease are at greater risk of developing cardiovascular events with psychostimulants (70).

The non-medical use of psychostimulants has adverse consequences both for individuals and for public health. Between 2005 and 2010, emergency room visits for this reason among adults in the US increased by 200% (from 5,212 to 15,585), while those specifically related to amphetamines rose by 156%. In adolescents, about 35% of exposures due to non-medical use of methylphenidate or amphetamines are associated with clinically significant effects, including moderate or severe toxicity and deaths (71,72).

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With all of the above in mind, we are in a position to pose the bioethical dilemma: *To what extent is it ethically justifiable to prescribe or normalize the use of psychostimulants—such as methylphenidate or amphetamines—when, although they reduce harm and improve functionality in patients with ADHD, they are increasingly used by people without a medical diagnosis, in educational or work contexts, for reasons of performance and competitiveness?*

The use of psychostimulants such as methylphenidate and amphetamines has been clinically validated for the treatment of ADHD, but several authors warn that this efficacy does not necessarily translate into a sufficient scientific understanding of their mechanism of action or biological justification. Henry Middleton points out that "the quality of this scientific knowledge is very poor" (73) and that many psychotropic drugs are prescribed on a massive scale without evidence of an underlying neurochemical alteration (73). In the case of stimulants, he warns that without clear evidence that these children are chemically different, the effects could be due solely to increased activation (73). In other words, psychostimulants improve attention in both diagnosed and non-ADHD individuals, which precludes a specific etiology and calls into question the biomedical assumption that justifies their use.

Louis C. Charland has criticized the scientific validity of randomized clinical trials (RCTs) used to approve psychotropic drugs. He points out that "when the null hypothesis is rejected, it means that it is not correct to say that the treatment has no effect. This is not the same as saying that the treatment 'works'" (74). In many cases, the reported efficacy is marginal —on the order of 10% over placebo— and is based on a misleading statistical interpretation rather than causal verification. Therefore, the therapeutic benefit of psychostimulants is revealed to be epistemically ambiguous: there is functional improvement, but its neurobiological basis is uncertain. In bioethical terms, this point problematizes the principle of beneficence (75), since the intervention is not always based on solid knowledge, and that of non-maleficence, given the risk of adverse effects and dependence described by Middleton (73). Prolonged use of

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these drugs, it is claimed, produces "changes in the sensitivity and number of receptors, [...] modifying the way cells respond to both the drug and natural stimuli," leading to tolerance and withdrawal. Thus, the therapeutic evidence for ADHD cannot be considered neutral: efficacy does not equate to scientific understanding or moral justification for intervention.

For Charland, "certain aspects of thinking, feeling, and behavior, which were previously described without a medical conceptual framework, have been re-described using that conceptual framework" (74). This process has profound effects on the individual's self-understanding: "it affects the way individuals understand themselves" (74).

This author has stated that the DSM has been a central instrument in this expansion, generating what he calls "hyper-medicalization" (74). As diagnostic categories increase, the range of behaviors that can be considered pathological expands. In the case of ADHD, this process culminates in its reconceptualization in the DSM-5 as a "neurodevelopmental disorder" that can be diagnosed even in adults.

From an ethical standpoint, this expansion has normative and social consequences. Sadler argues that psychiatric diagnosis "is not a purely technical act, but a practice permeated by moral and cultural values" (76). The DSM classifications are not neutral descriptions, but normative judgments about what counts as 'deviation' or "normality." Therefore, he asserts that "the values underpinning diagnostic categories do not enjoy the moral consensus that characterizes other branches of medicine" (76).

The overdiagnosis of ADHD reflects this problem. As categories become more flexible, more individuals are incorporated into the therapeutic system, which carries risks of labeling and loss of autonomy. Sadler emphasizes that psychiatric labels generate "classification effects," shaping people's identities and behaviors (76). From this perspective, the diagnostic expansion of ADHD is not only a clinical phenomenon but also an ethical dilemma: at what point does it cease to be medical assistance and become a form of normative control over attention, behavior, and performance?

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As we have seen, there is a direct link between the process of medicalization and the non-medical use (NMU) of psychostimulants. As Middleton explains, these drugs do not correct a chemical deficit, but rather act by promoting the release of norepinephrine, serotonin, and dopamine (73), increasing the overall activation of the nervous system. Consequently, their use outside the therapeutic setting, for example, among college students or professionals seeking to improve their performance, does not differ physiologically from prescribed use, but it does shift the moral justification from treatment to optimization.

This shift raises a central ethical dilemma: the line between curing and enhancing. If psychostimulants can increase cognitive performance in healthy individuals, is it ethical to normalize their use in competitive educational and work contexts? According to Charland, contemporary psychiatry lacks sufficient autonomy to resist market pressures, given that it is closely allied with the interests of the pharmaceutical industry (74). This alliance contributes to normalizing non-medical consumption under the guise of efficiency and productivity.

The problem is exacerbated because, as Sadler shows, the values incorporated into DSM and psychiatric practices do not always respond to the patient's well-being, but rather to professional and social interests (76). Non-medical use thus becomes a form of medicalization of normality, where scattered attention or fatigue are interpreted as pathological deficits that require chemical correction.

From a care perspective (77,78), this phenomenon undermines the relationality of the person and replaces empathetic understanding of discomfort with the pursuit of performance. In this way, the non-medical use of stimulants turns attention and motivation into consumer goods, reducing self-care to a form of pharmacological self-optimization.

On the other hand, we find that the phenomenon of medicalization is closely linked to the commercial interests of drug manufacturers (pharmaceutical companies). This process is called "commercialization." This can be seen in the evolution of psychiatry. In its

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mature stage, discipline was dominated by commercial interests, giving rise to the phenomenon of *disease mongering*, that is, the expansion of the boundaries of disease for market purposes (79). Many of the new drugs are "me-too drugs," small variations of existing molecules that "offer marginal benefits" (79). This creates a scenario in which *scientific innovation* is subordinated to profit, while clinical trials focus on small, short-term populations, limiting knowledge about long-term effects. Along the same lines, Charland denounces the "lack of transparency derived from patent law" (74), which prevents public review of efficacy and safety data. Thus, psychiatric pharmacology operates within an epistemically closed and morally problematic structure, where scientific truth is replaced by commercial efficacy.

In the context of the institutional development of psychiatry, Sadler warns that the DSM-5 was developed under "conditions of excessive secrecy, with closed meetings and confidential discussions," which constitutes "not only a technical failure, but a moral problem" (76). Such obscurity surrounding the process of defining psychiatric pathologies calls into question the ethical legitimacy of psychiatry, by confusing the internal good of medical practice (healing and caring) with external goods such as professional prestige and economic benefits (80). Thus, the convergence of commercial interests, expansive diagnoses, and mass prescribing constitutes a loss of moral autonomy in medical science, with ADHD appearing as a paradigmatic case. The ethics of care and "well-ordered science" (81) offer an alternative path: reorienting scientific practice through deliberative transparency, conceptual plurality, and the primacy of patient well-being.

From a clinical point of view, there are also a number of tensions: first, the non-medical use of psychostimulants undermines the concept of ADHD: as we have seen, with the increase in diagnoses, criticism of the concept and suspicion of overdiagnosis or overmedicalization have also increased. This leads, as a consequence, to increased mistrust when prescribing these drugs. Second, the search for psychostimulants may be overshadowing other psychiatric or

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non-psychiatric conditions that can manifest with symptoms such as concentration problems and restlessness, which would only be detectable by a mental health professional. An example of this is the literature that addresses the search for differential diagnoses and/or comorbidities when consulting for "difficulties in concentration" (82,83). Third, there is an increased risk of exposure to adverse effects ranging from mild to severe. Among those already mentioned, people who do not have ADHD may develop tolerance and, in the long term, substance abuse. Finally, even assuming that this were not the case, and that we had no problems with adverse effects or other diagnoses at the individual level, a collective problem is nevertheless created. *The limits of productivity in society are changed*. People with resources, contacts, and information can access these drugs and produce more. But at the same time, our concept of functionality and performance is also changed, harming those who do not use them. Thus, clinicians become "accomplices" by increasing the limits of collective productivity. The non-medical use of psychostimulants violates the principle of justice from the ethics of care because it shifts the limits of "expected performance" based on market logic and individual competition, rather than organizing social life around relationships of interdependence and mutual responsibility. For Held, liberal justice and rights can only function within a pre-existing framework of care relationships and a "presumption of care" in society; when performance is governed by competitive and commercial norms, trust, solidarity, and concern for the needs of the most vulnerable are eroded, producing systematic exclusions under the guise of formal equality. Thus, the normalization of neuroenhancement through non-medical psychostimulants not only creates material inequality of access but also reconfigures social relations in such a way that expectations of success become unattainable without pharmacological resources, subordinating care and equity to the logic of individual advantage and betraying the moral priority that the ethics of care gives to the relationships and practices that sustain all members of the community. This discussion is already taking place in *e-sports*, where the use of these drugs is being debated, as well as in

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the university setting (84). In turn, among the collective effects are also the consecutive stock shortages that have occurred since the early 2020s (85). Although non-medical use is not the direct cause of the shortage, the high demand it generates contributes to the overall picture.

Based on what we have seen, we can provide an alternative to the bioethical dilemma posed at the beginning of the article: how can we ethically distinguish between treatment and performance optimization in a context where medical science is influenced by commercial interests, expansive diagnoses, and social expectations of productivity? How can we address these issues?

First, we must recognize that the process of medicalization is the result of an interrelationship between technology, interests, and social and ethical principles. Second, the scientific basis for the process of psychiatric medicalization is weak, and there is a risk of promoting neurochemical dependence precisely in the diagnosed and productive use of drugs associated with ADHD. Third, there is a need for institutional ethics that evaluate the processes of pathologization and medicalization associated with institutionally described and defined pathologies. Finally, it is always necessary to see how the market interrelates with institutional processes and how the commercialization of psychiatry, through the creation and promotion of new drugs, requires scientifically validated processes, which, in turn, require institutions that limit the influence of commercial interests for the internal good of medical activity.

From a clinical point of view, it is necessary, first of all, to be aware of the existence of the problem. Second, to emphasize a history of neurodevelopment and the onset of symptoms in childhood. Third, better calibrate the criterion of functionality, for example, with external functioning criteria. And finally, have stricter definitions and methods of measuring symptoms. For example, that inattention in ADHD must be present even under ideal conditions, or Douglas' model of treating the population with the most severe symptoms pharmacologically.

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6. Conclusion

The analysis of ADHD presented in these pages and the use of psychostimulants reveals a complex interrelationship between therapeutic advances, diagnostic transformations, social expectations, and commercial pressures. The available evidence confirms that psychostimulants can promote well-being in patients with ADHD; however, it also shows that their efficacy is based on neurobiological foundations that are still uncertain and on clinical trials whose results require cautious interpretation. The expansion of diagnosis, especially since the DSM-5, has brought new populations into the therapeutic sphere, intensifying the risk of overdiagnosis and reinforcing processes of medicalization that redefine everyday behaviors as pathologies.

The increase in the non-medical use of psychostimulants is the indicator where these tensions become most morally visible. When substances designed to treat a specific disorder are used to improve performance in competitive contexts, the boundary between treatment and optimization becomes blurred, and medical intervention can become involved in a logic of self-demand and productivity that is not its intended purpose. In response to this, it is necessary to strengthen institutional ethics that guarantee transparency in diagnosis, limit the influence of commercial interests, and reaffirm the internal good of medical practice. At the same time, social and political reflection is needed to recognize that attention disorders cannot be reduced exclusively to neurobiological dysfunction, but must be understood in their educational, relational, and cultural dimensions.

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