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Paradoxical Effects of Stimulants: Increases Concentration but does Reduces Learning Ability

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Abstract: Attention deficit hyperactivity disorder (ADHD) and attention deficit disorder (ADD) are commonly treated with stimulant medications such as methylphenidate and amphetamines. These substances are known to enhance concentration, alertness, endurance, well-being, and self-esteem. However, paradoxically, they have been shown to impair critical cognitive processes related to learning. This article reviews the current scientific literature on this paradox, including findings from neurological, psychological, and psychiatric research. It proposes a theoretical framework explaining the neurobiological mechanisms underlying these effects. A key study by Volkow et al. (2008)1 demonstrated that methylphenidate reduces glucose metabolism in brain regions involved in cognitive processing. This reduction in metabolic activity provides a potential explanation for the paradoxical cognitive effects of stimulants.

Paradoxically, they have been shown to impair critical cognitive processes related to learning. This article reviews the current scientific literature on this paradox, including findings from neurological, psychological, and psychiatric research. It proposes a theoretical framework explaining the neurobiological mechanisms underlying these effects.

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Method: This review aims to investigate the well-known paradoxical effects of stimulants. Various search engines were used to identify articles that could provide insights into the mechanisms underlying these effects.

Searches were conducted in PubMed, EMBASE, PsycINFO, and Google Scholar, with non-sponsored research being assigned greater scientific value than sponsored studies, as previous research has documented systematic bias in industry-sponsored trials. The methodological quality of the studies was assessed with a particular focus on the funding source, given that sponsored studies are frequently associated with overestimations of efficacy and underreporting of adverse effects2-9. To account for this, the findings in this review were categorized based on the funding source, and methodological quality was evaluated using criteria derived from the Cochrane Risk of Bias Tool 10 and Grading of Recommendations, Assessment, Development and Evaluation (GRADE) [11]

The objective of this review is not to assess the overall effectiveness or adverse effects of stimulants, but rather to explore their paradoxical mechanisms of action in order to identify potential explanations and directions for future research. With this aim, articles that had the potential to contribute to this understanding were selected for inclusion. The authors' own interpretations of their results are not taken into account; instead, the results are used independently of the proposed interpretations. Even with the use of these tools, assessment can be challenging; however, if a sponsored study presents a strongly contradictory evaluation of efficacy or safety compared to nonsponsored studies, it will be considered critical.

Results: Stimulants increase alertness, endurance, and self-esteem; however, they also appear to impair essential cognitive processes necessary for academic and intellectual development in individuals with ADHD/ADD as well as in those without these diagnoses. The explanation may lie in the fact that these substances enhance activity in monoamine networks while reducing metabolism in other critical brain networks.

The reduction in the impact of the habenular nuclei on monoamine systems may potentially affect certain forms of learning, preventing them from functioning properly when stimulants are used. However, it is likely that the lack of learning should also be associated with other systems in the brain as well.

Conclusion: The paradoxical effects of stimulants raise significant questions about their role in cognitive enhancement and learning. While these drugs increase alertness, endurance, and self-esteem, they appear to impair essential cognitive processes necessary for academic and intellectual development. Neuroimaging studies suggest that reduced metabolic activity and disrupted functional connectivity in key learning networks may underlies the lack of learning improvements and enhancement of academic skills.

When the influence of the habenular nuclei on the monoamine systems is reduced, it leads to a decrease in learning associated with negative experiences. However, this only explains some of the learning difficulties linked to the use of stimulants, and it does not account for the significant findings reported by Volkow and colleagues.

Future research should further investigate how stimulants affect brain function and their long-term consequences on cognitive development, as they seem to increase the risk of impaired executive functions, psychosis, and other neurological disorders—especially in individuals dependent on these medications, but also in those who receive them by prescription over extended periods.

Introduction

Pharmacological treatment remains the predominant approach for managing ADHD/ADD, despite official guidelines advocating psychological interventions and social or educational support as firstline treatments. Various stimulant medications, including methylphenidate, amphetamine, and methamphetamine,

function by increasing neurotransmitter activity—primarily dopamine, norepinephrine, and serotonin—via reuptake inhibition and enzymatic degradation suppression [12-19].

In addition to the most classic forms of amphetamine and methylphenidate, other substances used include methamphetamine, methylphenidate hydrochloride, lisdexamfetamine dimesylate, amphetamine sulfate, mixed amphetamine salts, a combination of dexmethylphenidate hydrochloride and serdexmethylphenidate chloride, selective norepinephrine reuptake inhibitors (atomoxetine, viloxazine), and alpha-2 adrenergic receptor agonists (guanfacine hydrochloride, clonidine hydrochloride). The substances most commonly used for treating ADHD/ADD symptoms remain the original stimulant forms: methylphenidate and amphetamine [20].

The substances later developed for the treatment of ADHD/ADD are, to varying degrees, simple derivatives of stimulants. They align to different extents with both stimulants and antidepressants, all of which enhance monoamine signaling—serotonin, dopamine, and norepinephrine—in different proportions and combinations.

Amphetamine, developed in 188721, and methylphenidate, synthesized in 194422-23, were primarily used as treatments for depression xxx og narcopleci until the 1960s24. Additionally, these substances were employed as performance-enhancing drugs in sports25-26 and academia. However, in the 1970s, they began to be used to improve concentration and academic performance in children with ADHD/ADD symptoms, with reference to Charles Bradley's early experiments from 1937. Bradley administered amphetamine to children with ADHD/ADD symptoms, yet at the time, other physicians opposed the use of narcotics in children.

This stance shifted in the 1970s, and stimulants are still considered the preferred first-line treatment for ADHD/ADD in many parts of the world. These substances have now been used for over five decades to manage ADHD/ADD symptoms, with ongoing claims that they remain the most effective and widely prescribed pharmacological treatment today.

The rationale for using these substances has been based on the assumption that they improve attention span, reduce distractibility, enhance memory function, minimize impulsivity, mitigate hyperactivity, and improve social skills. Consequently, it has been suggested that these effects should enhance learning ability and lead to better academic performance 29-30. However, even if these assumptions were correct, they do not appear to be supported in practice. The results of numerous studies are highly inconsistent and contradictory—even after excluding studies funded by pharmaceutical companies. There is no conclusive evidence that stimulants have beneficial effects on key outcome measures, particularly academic performance [31].

It is rare to observe such conflicting findings in other scientific disciplines as seen in psychiatry. This discrepancy is partly due to the complexity of psychiatric research and the variability in methodological approaches, which can contribute to divergent results. The highly contradictory findings must also be interpreted in light of the fact that industry-sponsored studies often yield significantly different conclusions than independent research. However, when examining the effects of stimulants specifically, these inconsistencies seem to stem from other underlying factors that, upon closer analysis, may provide a clearer understanding of their impact [32].

The Paradoxical Effects of ADHD/ADD Medication

Research on the effects of ADHD/ADD medication reveals the following paradox: Why does ADHD/ADD medication not improve academic performance, despite

increasing wakefulness, endurance, attention, and concentration?

Some of the researchers who have specifically investigated this phenomenon are Claire Advokat and Mindy Scheithauer from Louisiana State University Baton Rouge, USA. In a 2013 article, they describe the issue as follows: “Recent increases in attention deficit hyperactivity disorder (ADHD) diagnoses, and the escalation of stimulant prescriptions, has raised concern about diversion and abuse of stimulants, as well as the ethics of using these drugs as “cognitive enhancers. “Such concern appears misplaced in the face of substantial evidence that stimulant drugs do not improve the academic performance of ADHD-diagnosed students. Moreover, numerous studies have found little or no benefit of stimulants on neuropsychological tests of ADHD-diagnosed as well as normal, individuals”. [29]

In this study by Claire Advokat and Mindy Scheithauer from the Department of Psychology at Louisiana State University Baton Rouge, USA, the apparent paradox is examined: why do stimulants enhance "attention" but not academic performance in students diagnosed with ADHD/ADD?

Advokat and Scheithauer were unable to identify a definitive explanation for this phenomenon. However, their findings indicated that stimulant use promotes "risky behaviour" and increases susceptibility to "environmental distractions." Despite this, they could not pinpoint a clear underlying mechanism.

In an earlier study from 2011, Advokat and colleagues investigated the relationship between ADHD/ADD medication, study habits, and academic performance among university students with an ADHD/ADD diagnosis. The majority of students with ADHD/ADD who used stimulant medication believed it helped them. However, they also rated themselves as being worse than other students at planning, completing assignments, and avoiding distractions. Although the study habits of students with ADHD/ADD did not significantly differ from those of the control group, their average grades from both high school and university, as well as their standardized test scores, were significantly lower. They also withdrew from significantly more courses compared to students who did not take medication. Interestingly, preliminary data from the study suggested that good study habits alone— without stimulant medication—could compensate for the performance gap among students with ADHD/ADD [33].

Another study by Advokat and colleagues from 2008 further demonstrated that ADHD/ADD medication does not significantly enhance cognitive abilities. The study examined individuals diagnosed with ADHD/ADD who were using stimulants for academic purposes and compared them with individuals who also took similar stimulants but did not have an ADHD/ADD diagnosis34. The results showed that the group with ADHD/ADD achieved significantly lower grades than those taking the same stimulants without an ADHD/ADD diagnosis.

This finding suggests that ADHD/ADD medication does not mitigate the cognitive deficits associated with ADHD/ADD symptoms compared to individuals without the diagnosis. These results are also consistent with the well-known studies conducted by Judith Rapoport and colleagues in 1978 and 2002, which concluded that stimulants do not have a differential effect on individuals with ADHD/ADD symptoms compared to those without [35-39].

Furthermore, these studies dismissed the hypothesis that ADHD/ADD symptoms are linked to low dopamine levels.

Nevertheless, some researchers continue to argue for a connection between dopamine and ADHD/ADD symptoms. However, their ability to do so relies on the

fact that while stimulant medication produces measurable effects, these effects do not ultimately result in meaningful cognitive improvements.

Stimulants enhance various brain functions associated with increased attention, wakefulness, activity, self-confidence, mood/euphoria, and social interaction, particularly when used in low doses over a short period. This effect occurs primarily because stimulants increase dopamine signalling, which plays a central role in these functions.

However, the benefits of stimulant medication may not be as significant as many pharmaceuticalsponsored studies suggest. This is evident in a study by Schein et al., which examined ADHD/ADD and the side effects of ADHD/ADD medication among adults in the United States. Their findings concluded: “Symptoms associated with ADHD and treatment-related side effects are common and have a significant negative impact on quality of life, as well as reducing patients’ likelihood of gaining employment” [40].

This is despite the fact that stimulants initially enhance concentration, activity levels, endurance, and mood. Most studies that highlight such positive effects primarily focus on the short-term impact of these drugs. Nevertheless, there is no doubt that substances such as amphetamine and methylphenidate increase attention, activity, wakefulness, endurance, and self-esteem while fostering a subjective sense of improved social functioning, as demonstrated in a study by Harpin et al.41 Individuals who take stimulants like amphetamine and methylphenidate tend to overestimate their performance. Additionally, research suggests that these substances can increase talkativeness42, which may be indicative of euphoria and/or a greater sense of ease in social situations.

In another study, researchers investigated the acute administration of varying doses of methylphenidate (10 mg, 20 mg, 40 mg, and placebo) on a broad range of cognitive functions in healthy young individuals. Their findings led to the following conclusion:
"According to recent literature, stimulants such as methylphenidate enhance performance when cognitive processes are functioning below an optimal level, which was not the case for the participants in the present study. We propose that the impression that methylphenidate improves cognitive performance in healthy young individuals—and thereby justifies its use—may be due to enhancements in subjective well-being induced by the drug. [43].

Ved længere tids påvirkning, så viser undersøgelser at brugerne bliver dårligere på en lang række områder, som f.eks. hukommelse, fastholdelse af opmærksomhed, social opmærksomhed, sociale færdigheder, empati m.m. [44-47].

The lack of actual cognitive benefits is further supported by evidence showing that individuals who use these stimulants struggle to integrate into the workforce or achieve a satisfactory social life, despite extensive medication with relatively high doses of these substances. Furthermore, research indicates that stimulant drugs can impair cognitive abilities and emotional responses relatively quickly. When taken in excessive amounts or at higher doses, these effects can escalate to the point of psychosis [48-77].

An increased risk of psychotic disorders and the potential development of schizophrenia and psychosis have also been linked to long-term stimulant use [50,57,78-84].

A study examining vulnerable adolescents and children of parents with psychiatric disorders found that 62.5% of young individuals who had been treated with stimulants exhibited psychotic symptoms, compared to 27.4% of vulnerable adolescents who had never taken stimulants [81].

Individuals who are already predisposed to psychosis

face an increased risk of 30–40% of developing psychotic symptoms when using stimulants82-83. A similar pattern has been observed in individuals diagnosed with bipolar disorder, further emphasizing the potential risks associated with long-term stimulant use85. Man ser da også forandringer i nogle områder af hjernen, som kan ligne skizofreni [44].

Overall, it can be concluded that stimulants have a significant impact on executive functions, enhancing some cognitive processes while impairing others. These substances increase attention processes, wakefulness, and alertness; however, paradoxically, they also lead to poorer learning outcomes.

A research team led by Jurjen van der Schans and colleagues from the University of Groningen, Netherlands, investigated children who used methylphenidate compared to those who had never taken the drug. Their study linked data from a pharmacy prescription database with standardized test results from primary school students in the Netherlands and reached the following conclusion:

"Our study shows that children who use methylphenidate still perform worse in school compared to their peers. Our findings also suggest that an earlier initiation of methylphenidate treatment is associated with lower academic performance compared to children who start treatment later." [31]

A study has shown an improvement in academic skills among children with ADHD/ADD who received stimulant treatment, compared to those with ADHD/ADD who did not receive such treatment. However, they never reached the same level as their peers without ADHD/ADD. Notably, this study was conducted by researchers who received funding from the pharmaceutical industry [85].

On the other hand, there is no doubt that stimulants enhance self-esteem, attention, and endurance while also influencing thinking patterns and emotional states in complex ways that are not yet fully understood. These substances exhibit seemingly contradictory and paradoxical effects—some beneficial, others harmful.
Further research indicates that stimulants can cause significant damage to the brain, leading to cognitive decline and stimulant use has been linked to neuronal death in dopamine-producing cells, and the risk of developing Parkinson's disease increases by 200–850% at a young age [86-91]. Additionally, the risk of dementia rises by 400% [92]. This strongly suggests that stimulants have a profound effect on cognitive function and that excessive or prolonged use can be highly detrimental to executive functions.

These various effects may influence how stimulants impair cognitive functions over time and may contribute to a reduction in learning ability. Paradoxically, however, they may also enhance concentration, endurance, and self-esteem. Yet, stimulants reduce learning ability from the very beginning of treatment. The effect that creates the paradoxical nature of stimulants from the moment the substance is first time consumed will be examined further in the following sections.

Neurological Mechanisms Underlying the Paradox

A particularly important study sheds light on why stimulants increase concentration, activity levels, endurance, and self-esteem, yet simultaneously impair critical cognitive processes involved in learning.

In this study by Volkow and colleagues, Positron Emission Tomography (PET) scanning was utilized to visualize glucose consumption in the brain. PET scanning is an advanced neuroimaging technique based on measuring radioactive decay from a tracer, allowing for detailed imaging of metabolic activity. Tomography, in general, refers to any imaging method that produces cross-sectional layers of an object using penetrating radiation or waves.

Statistical Parametric Mapping (SPM) was applied to data from various brain imaging techniques, including fMRI (functional Magnetic Resonance Imaging), PET, and EEG/MEG (Electroencephalography/Magnetoencephalography), to determine regions with the highest metabolic activity. Statistical tests were conducted on each voxel (a three-dimensional pixel in the brain imaging data) to assess whether significant changes in neural activity occurred between different conditions, such as a cognitive task versus a control state. Brain regions with statistically significant differences were then highlighted and visualized as color-coded areas on brain maps.

The study found that while methylphenidate increased concentration, it did not improve task performance compared to individuals who had not taken the drug. However, Volkow and colleagues made a surprising conclusion:

"Since the brain required approximately 50% less glucose to perform the task at the same level, this provides evidence that one of the mechanisms behind methylphenidate's effect is the ability to focus neural activation and make the brain more efficient." [1]

At rest, the brain's relative activity level was the same regardless of whether methylphenidate had been administered or not. The study shows that when performing a task after taking methylphenidate, brain activity increased by 11% and 22%, whereas without the drug, brain activity increased by 22%. The results of task performance remained at a relatively similar level.

Volkow and colleagues speculated that stimulants might reduce "neural noise", thereby enhancing cognitive efficiency in the adults they studied. However, the notion that 50% of the increased neural activity should be attributed to "neural noise" seems implausible. Instead, a more reasonable interpretation would be that stimulants suppress learning-related neural systems, rather than merely reducing noise.

This consideration is only valid if one examines the results in isolation. However, when the findings are contextualized with the well-documented fact that stimulants do not significantly enhance learning, the interpretation shifts in a different direction, making the results more meaningful. Volkow and colleagues also provide a more plausible explanation, suggesting that methylphenidate reduces the activity of what is known as the "default network" (DN).

The default network (DN) is active during unfocused mental states, such as relaxation, reflection, and metacognition, and is also associated with daydreaming and other internally directed cognitive processes. Additionally, DN plays a critical role in long-term memory and the integration of new information.

DN can be divided into core regions and two distinct subsystems. The core regions include the anterior medial prefrontal cortex, posterior cingulate cortex, bilateral angular gyrus, lateral temporal lobes, and superior frontal gyrus. The dorsomedial subsystem consists of the dorsal medial prefrontal cortex, temporoparietal junction, lateral temporal cortex, temporal pole, and inferior frontal gyrus [93].

Recent studies have shown that brain regions within DN remain active during task performance when cognition and behavior benefit from memory processes. For instance, DN activation is observed when decisions rely on previous trial information [94-96], when task-relevant stimuli are supported by long-term memory [97], when participants retrieve task context from memory, or when they encode rules upon which their actions are based [98].

Chandra Sripada and colleagues from the University of Michigan provide additional insight into why methylphenidate enhances attention while simultaneously suppressing certain neural systems. Their findings, derived from a support vector machine analysis, indicate that methylphenidate reduces

connectivity between the visual and somatomotor networks while also diminishing DN activity during task execution. The researchers suggest that this suppression may be a key mechanism behind methylphenidate's ability to enhance attention during cognitive tasks, but it also likely impairs other neural systems involved in learning [99].

Discussion

The results are in good agreement with previous research, where no significant improvements in learning have been observed, and where these substances may even reduce learning ability over time.

Conversely, it is relatively evident that stimulants increase wakefulness, concentration, endurance, euphoria, and self-esteem. The increased focus and attention that arise when taking methylphenidate or other stimulants may potentially be explained by an effect that suppresses competing networks, allowing energy to be utilized more efficiently. Volkow and colleagues have suggested that inhibition of the default network (DN) could be a contributing factor1, and this hypothesis has been pursued by other researchers.

Chandra Sripada and colleagues from the University of Michigan provide further insight into why methylphenidate enhances attention while simultaneously suppressing certain neural systems. Their findings, derived from a support vector machine analysis, indicate that methylphenidate reduces connectivity between the visual and somatomotor networks, while also decreasing activity in the default network (DN) during task performance. The researchers suggest that this suppression may be a key mechanism behind methylphenidate’s ability to enhance attention during cognitive tasks 93, but it may also likely impair other neural systems involved in learning. This aspect was not addressed in their study. Consequently, it would be pertinent to investigate the various systems that may be suppressed by stimulants.

In addition to DN, other networks that warrant further investigation include: central executive network (CEN), cognitive control network (CCN), dorsal attention network (DAN), executive control network (ECN), executive network (EN), frontoparietal network (FPN), working memory network (WMN), task positive network (TPN), ventral attention network (VAN), Salience Network, Procedural Memory System (PMS) and Declarative Memory System (DMS).

Understanding which neural systems are downregulated during stimulant use in learning and problem-solving contexts is crucial for both optimizing ADHD/ADD treatment strategies and assessing the impact of stimulant use among individuals with substance use disorders, as the inhibition of these networks may contribute to addiction-related mechanisms and other mechanisms where one may become overly motivated, hyper-focused, and prone to taking excessive risks.

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The networks associated with the two small habenular nuclei are specific networks that are affected by stimulants both directly and indirectly.

The habenular nuclei, a structure in the midbrain, can be referred to as our "depression" or "demotivation center." Its purpose is to suppress processes related to motivation under negative circumstances, in situations where the effort of a behavior does not seem to yield a

reward that makes it worthwhile, or during states of surrender—when nothing can be done.

In the midbrain, or diencephalon, lies the epithalamus. The epithalamus constitutes a small part of the diencephalon and consists of three smaller structures: the pineal gland, the habenular nuclei, and the stria medullaris thalami, along with the third ventricle. Most of the epithalamus is located near the caudal and dorsal parts of the thalamus. It is believed that the habenula has evolved in close interaction with the pineal gland [100-101]. The stria medullaris receives and transmits input to the habenular nuclei, while the fasciculus retroflexus serves as the output pathway from the habenular nuclei, consisting of axons from the habenula [102].

This larger complex plays a central role in our experience of demotivation, reduced energy, sadness, heaviness, and surrender. On the other hand, it is also associated with fatigue and sleep, where the pineal gland is the dominant structure, while the habenula is central to the experience of demotivation, surrender, giving up.

The habenular nuclei consist of two nuclei [103]: the lateral habenular nuclei and the medial habenular nuclei. The lateral habenular nuclei receive input from the basal ganglia, limbic areas, and cognitive regions, encompassing motor, emotional [104-107], and cognitive input [108].

The habenular nuclei are involved in learning and memory related to unpleasant experiences, which serve to demotivate us [104,109]. This structure is also activated during decision-making under negative influences and stress [110]. Additionally, the habenula is highly active when we make mistakes and process those errors104. Damage to the habenula impacts cognitive processes and has consequences for learning, memory, and attention abilities [104, 111]. Furthermore, damage to the habenula also leads to sleep disturbances [112].

When the habenula is activated, it suppresses the monoamine systems, including dopamine, norepinephrine, serotonin, and histamine [113-117]. This suppression lowers motivation and increases uncertainty, and strong activation results in fatigue, heaviness, and a sense of surrender. This system is crucial for evaluating ambiguous, uncertain, and potentially dangerous situations. It is also essential for assessing what is worthwhile or wise to pursue. The habenula plays a significant role in reflection related to uncertainty, ambiguity, self-assessment, and other metacognitive processes.

These processes are particularly important in situations that are ambiguous or in other situations where we need to experience uncertainty. It is likely that the increased release of monoamines in the brain when stimulants are used counteracts the impact of the habenula on conscious processes. This, in turn, may enhance motivation, energy levels, and self-confidence, thereby reducing uncertainty and doubt, but it can also have a negative effect on learning.

Most individuals who take stimulants report thinking less, having fewer distracting thoughts, being more focused, feeling less uncertain, and having greater confidence in their decisions. These effects are likely due to the habenula's inability to suppress the monoamine systems when the structure is stimulated. However, this does not explain the findings of Volkow and colleagues, who observed a 50% reduction in glucose consumption. Therefore, it is highly likely that other systems are also being suppressed.

Stimulants also have a direct impact on neurons in the habenula. It has been demonstrated that continuously administered amphetamine has a neurotoxic effect on dopamine terminals in the caudate and that both amphetamine and cocaine, when administered continuously over a period of 3–5 days, induced a

highly specific pattern of axonal degeneration extending from the lateral habenular nucleus along the fasciculus retroflexus toward the ventral tegmental area [118].

The effect of chronic amphetamine administration is also observed in relation to the expression of the D2 dopamine receptor, which was immunohistochemically analyzed in the caudate-putamen and the lateral habenular nucleus. A significant reduction in the number of immunopositive neural cells was observed in both regions, suggesting that chronically administered amphetamine alters the function of the D2 dopamine receptor in the dorsal diencephalic conduction system. This may be involved in the development of schizophrenia, stereotypic behavior, and neurological disorders in individuals who consume psychostimulants on a daily basis [119].

White matter is also reduced in the connections between the habenula and the prefrontal cortex [120], which is highly significant for the functional capacity of individuals who continuously use stimulants.

These direct effects can have profound consequences for human learning ability and may result in lasting damage, as well as maladaptive behaviour, where the ability to reconsider one's thoughts is impaired.

Conclusion

The paradoxical effects of ADHD/ADD medication raise significant questions about their role in cognitive enhancement and learning. While these drugs increase alertness, endurance, and selfesteem, they appear to impair essential cognitive processes necessary for academic and intellectual development. Neuroimaging studies suggest that reduced metabolic activity and disrupted functional connectivity in key learning networks may underlie the lack of learning improvements and enhancement of academic skills.

The reduction in the impact of the habenular nuclei on monoamine systems may potentially affect certain forms of learning, preventing them from functioning properly when stimulants are used. However, it is likely that the lack of learning should also be associated with other systems in the brain.

Future research should further investigate how stimulants affect brain function and their long-term consequences on cognitive development, as they seem to increase the risk of impaired executive functions, psychosis, and other neurological disorders—especially in individuals dependent on these medications, but also in those who receive them by prescription over extended periods. The use of stimulants as a treatment should be approached with a high degree of restriction and caution.

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