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Linking ADHD Treatment to Lifespan Brain Health: Evaluating Combined Stimulant and Cognitive-Behavioral Interventions in Dementia Risk

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Abstract

This narrative review examines evidence linking Attention-Deficit/Hyperactivity Disorder (ADHD) and long-term brain health, with a focus on how stimulant medication and cognitive-behavioral therapy (CBT) may influence cognitive aging. ADHD involves dysregulated dopamine signaling that impairs reward processing and executive control, contributing to lifelong cognitive and behavioral challenges. Large epidemiological studies indicate that adults with ADHD have a higher incidence of mild cognitive impairment (MCI) and dementia, even after accounting for psychiatric comorbidities. However, individuals treated with stimulant medications show no such increase in dementia risk, suggesting possible neurologically protective effects. Across randomized controlled trials and meta-analyses, combined treatment with stimulant medication and CBT consistently yields greater improvements in attention, organization, and emotional regulation as compared to either approach alone. These findings support the hypothesis that multiple therapies targeting both dopamine signaling and behavioral regulation may not only optimize ADHD treatment but also reduce the likelihood of late-life cognitive decline. Integrating pharmacological and behavioral interventions may therefore represent a sustainable option for promoting cognitive health across the lifespan.

Keywords

ADHD, Brain Health, Childhood, Dementia Risk, Cognitive Behavioral Therapy

1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental condition characterized by inattention, hyperactivity, and impulsivity. It is among the most common childhood conditions, affecting an estimated 5 - 7 percent of children worldwide [1] and approximately 9.8 percent of children in the United States between the ages of 3 and 17 years [2]. ADHD often persists into adulthood. Affected children often experience academic difficulties such as lower grades, higher rates of grade retention, and learning disorders [3]. They may also show behavioral problems, including impulsive rule breaking, classroom disruption, and social difficulties such as peer rejection, family conflict, or low self-esteem [4].

One leading hypothesis is that ADHD arises from dysregulated dopamine signaling. Neuroimaging and genetic studies consistently associate the disorder with reduced dopamine transporter and receptor availability [5]. Dopamine is a neurotransmitter that plays a central role in reward processing, motivation, and executive functions such as attention and working memory. In the typical brain, dopamine is released into the synaptic cleft and binds to postsynaptic receptors, reinforcing behaviors that lead to positive outcomes and supporting sustained focus on goal-directed tasks. When dopamine signaling is reduced, whether from lower dopamine synthesis, fewer receptors, or excessive transporter activity, the result is diminished reward sensitivity and impaired regulation of attention and impulse control. This state makes it difficult for individuals with ADHD to maintain focus on delayed or repetitive tasks, contributing to symptoms such as distractibility, restlessness, and impulsivity. Broader neurobiological models emphasize that dysfunction in both dopaminergic and noradrenergic systems is central to ADHD pathophysiology and treatment response [6].

Because reduced dopamine signaling is thought to play a key role in ADHD, current first-line treatments include stimulant medications that enhance dopamine transmission. These drugs, including methylphenidate and amphetamine-based compounds, primarily act by blocking dopamine and norepinephrine reuptake transporters, preventing these neurotransmitters from being recycled too quickly [7] [8]. As a result, dopamine remains available in the synaptic cleft longer, increasing its synaptic availability and strengthening connections within circuits responsible for attention, motivation, and inhibitory control. Neuroimaging studies show that stimulant use restores activation in frontostriatal pathways, particularly the prefrontal cortex and striatum, regions that are critical for executive function and reward-based learning [6]. By enhancing dopamine activity in these networks, stimulants can temporarily normalize the neural deficits underlying ADHD symptoms, leading to measurable improvements in focus, planning, and behavioral regulation. However, these effects are often short-term, and the mechanism by which long-term modulation of dopamine signaling affects brain development or cognitive outcomes later in life remains unclear.

Although stimulants reliably reduce symptoms in the short term, ADHD's effects often extend well beyond childhood, raising questions about its long-term

impact on brain health. Large population studies have found that adults with ADHD face a significantly higher risk of developing mild cognitive impairment (MCI) or dementia [9] [10]. These associations weaken after adjusting for psychiatric comorbidities such as depression and substance use, yet they persist, suggesting a potential link between ADHD-related neurobiology and cognitive aging. Importantly, however, current evidence does not indicate that stimulant therapy increases this risk. In fact, exploratory subgroup analyses consistently report no increased incidence of dementia among individuals treated with stimulants, implying that dopamine-enhancing interventions may help stabilize long-term cognitive function. Supporting this possibility, small clinical trials in dementia populations have demonstrated that methylphenidate can improve apathy, attention, and daily functioning, potentially modulating neurochemical pathways involved in cognitive decline [11] [12].

2. Scope and Rationale: Linking ADHD Treatment to Long-Term Brain Health

This review examines whether a combined treatment strategy using low dose stimulant medication and CBT can provide effective short-term symptom control in ADHD while also supporting long-term brain health. CBT refers to a structured, skills-based approach that targets sleep, mood, daily routines, and overall functioning. These domains are central to ADHD management and are also connected to the concept of cognitive reserve, the brain's ability to maintain function despite age-related or pathological changes, shaped by factors such as education, lifestyle, activity level, and behavioral coping strategies.

The discussion integrates evidence from three complementary areas of research. First, randomized trials and meta-analyses have consistently demonstrated that stimulant medications reduce ADHD symptoms [7] [8]. More recent studies show that CBT can also be effective for adults with ADHD, including both internet-based delivery [13] and structured combined treatment with medication, which yields greater improvements than either treatment alone [14].

Second, large population cohort studies indicate that ADHD is associated with an increased risk of later cognitive decline, including MCI and dementia [9] [10]. Although factors such as depression and substance use partly weaken this relationship, the association persists, suggesting that ADHD itself or its underlying neurobiology may influence long-term brain health.

Third, small mechanistic studies in dementia populations suggest that dopamine and norepinephrine-enhancing stimulants, particularly methylphenidate, can improve apathy, attention, and daily functioning in Alzheimer's disease [11] [12]. These findings provide proof-of-concept that stimulant medications may influence not only symptoms but also disease related markers in aging brains.

The scope of this review spans adolescence through adulthood and considers both clinical symptoms (inattention, hyperactivity, impulsivity) and functional outcomes such as academic, work productivity, daily routines, sleep, mood, and

activity levels. It also addresses modifiable dementia risk factors, including depression, sleep disruption, and physical inactivity, as potential pathways linking ADHD treatment to long-term cognitive health. While no ADHD intervention has been proven to prevent dementia, the available evidence suggests that a dual approach combining low dose stimulants with CBT targeting sleep, mood, and activity is more sustainable than medication alone and offers a promising framework for reducing long-term cognitive risk.

3. Epidemiological Evidence Linking ADHD to Dementia Risk

Several large population studies indicate that ADHD is associated with an increased risk of developing dementia later in life, although the strength and nature of this link remain debated. MCI, a transitional stage between normal aging and dementia, is also relevant because it represents early cognitive decline that may eventually progress to full dementia.

Evidence from an Israeli cohort of 109,218 adults born between 1933 and 1952, followed for an average of 17 years found that an adult ADHD diagnosis was linked to a significantly higher likelihood of developing dementia. Individuals with ADHD were about 3.6 times more likely to develop dementia before adjustments (unadjusted hazard ratio (HR), 3.62), and about 2.8 times more likely after adjusting for 18 different confounding factors such as age, sex, and health conditions (adjusted hazard ratio 2.77, 95% confidence interval [CI] of 2.11 - 3.63). An HR above 1.0 indicates increased risk, and a CI shows the range in which the true value is likely; because the CI does not cross 1.0, this association is considered statistically significant. Additional analyses suggested that the findings were unlikely to result from reverse causation, meaning that early dementia symptoms were not mistaken for ADHD. Importantly, the study found no increased risk of dementia among adults with ADHD who had received stimulant medication; however, this pattern should be interpreted cautiously, as it may reflect residual confounding, such as differences in healthcare access, illness severity, or socioeconomic status, rather than a true protective effect of medication. Early cognitive decline may also reduce the likelihood that individuals are prescribed or remain on stimulants, making reverse causation a plausible explanation for the observed association. Despite limitations such as reliance on diagnostic codes rather than symptom severity, the long follow up period, national registry data, and rigorous methods strengthen the conclusion that ADHD itself may be an independent risk factor for dementia [9].

A Swedish population study of 3.59 million individuals born between 1932 and 1963 yielded similar but more nuanced results. This study also found that ADHD was linked to both dementia (HR 2.92, 95% CI 2.40 - 3.57) and MCI (HR 6.21, 95% CI 5.25 - 7.35), suggesting an especially strong association with early cognitive decline. However, after adjusting for psychiatric comorbidities such as depression, anxiety, bipolar disorder, and substance use, the association between ADHD and dementia weakened substantially (HR 1.62, 95 percent CI 1.32 - 1.98).

This finding indicates that part of the observed risk may be explained by overlapping mental health conditions rather than ADHD alone. The study's strengths include its large sample size and detailed statistical modeling, but it also faced limitations such as potential diagnostic misclassification, residual reverse causation, and a relatively young median age at follow up, which may capture earlier onset rather than late onset dementia.

Recent longitudinal research continues to support and refine these earlier findings. A 2023 nationwide Israeli cohort study involving more than 109,000 adults aged 51 to 70 at baseline followed participants for up to 17.2 years and found that adult ADHD was associated with a 2.77-fold higher risk of incident dementia after adjusting for 18 confounding factors [10]. A 2024 15-year prospective cohort extended this evidence by showing that adult ADHD was independently associated not only with overall dementia but also with Lewy body disease, non-amnestic mild cognitive impairment, and cognitive decline across aging trajectories [15]. These studies reinforce that ADHD may confer increased vulnerability to multiple neurodegenerative pathways and highlight the importance of long-term follow-up across diverse diagnostic categories.

Together, the Israeli study suggests that ADHD itself may directly increase dementia risk, while the Swedish study indicates that much of the risk may instead come from psychiatric comorbidities. Both studies agree that ADHD is associated with higher risk of dementia and MCI, but they differ in interpretation of the underlying cause. These findings reveal important gaps in knowledge: it remains unclear whether ADHD contributes to dementia through direct neurobiological mechanisms or functions more as a marker of psychiatric vulnerability. The potential protective role of stimulant medication also warrants further study, and future research should include diverse populations to determine whether these results extend beyond Israel and Sweden [16].

4. Stimulant Medication and Long-Term Cognitive Outcomes

Several studies have examined whether stimulant medications for ADHD worsen, protect, or have no effect on long-term cognition and dementia risk. Current evidence does not show that stimulant medications increase dementia risk, and some findings suggest a possible protective effect. However, findings remain inconsistent and limited by study design, leaving the core question unanswered.

In the Israeli cohort, secondary analyses found no elevated dementia risk among adults with ADHD who had been prescribed stimulants, in contrast to the overall higher dementia risk observed in untreated ADHD cases [9]. This raises the possibility of a protective effect, although the finding may also reflect confounding factors such as access to care, illness severity, or incomplete exposure data. However, this pattern should be interpreted cautiously, as differences in healthcare access, illness severity, socioeconomic factors, or diagnostic scrutiny may account for the observed association rather than any true protective effect of stimulant treatment. Early cognitive decline may also reduce the likelihood that

individuals initiate or continue stimulant therapy, making reverse causation a plausible explanation. Only about 22 percent of the ADHD group had documented medication use, and details about dosage and duration were limited.

Beyond dementia outcomes, cognitive studies provide additional insight. A two-year prospective study reported that continued methylphenidate use produced sustained improvements in working memory, suggesting ongoing neurocognitive benefits during treatment [17]. In contrast, a randomized controlled trial (RCT) lasting sixteen weeks found that improvements in reaction time and working memory did not persist after medication discontinuation, indicating that benefits may be largely symptomatic and present only during active treatment [18].

Together, these studies suggest that stimulant medications neither accelerate nor clearly prevent dementia risk. The epidemiological data are reassuring in showing no harm, but subgroup analyses alone cannot establish causality. The cognitive studies highlight that stimulants do improve executive functioning while in use, but that durable, off-medication benefits remain questionable. Thus, stimulants may help optimize short to mid-term brain function and reduce secondary risks such as depression or reduced activity that indirectly affect dementia outcomes, but there is no evidence yet pointing to direct neuroprotection.

(OPTION 2) Mechanistically, chronic modulation of catecholamine signaling could plausibly influence neurodegeneration through multiple pathways: dopamine oxidation and subsequent neuromelanin formation can generate reactive quinones, induce mitochondrial dysfunction, and promote α-synuclein aggregation [19], whereas preserved noradrenergic tone has been shown to suppress microglial activation and enhance amyloid clearance [20].

Overall, the findings lean toward cautious interpretation: stimulant use appears to be at least neutral, and possibly beneficial, with no indication of increased dementia risk. The Israeli cohort's observation that treated ADHD patients did not share the elevated dementia risk seen in untreated individuals aligns with this interpretation, while smaller cognitive studies clarify that the observed advantages are most evident during active treatment. In summary, current evidence points toward short to mid-term cognitive benefits and potential indirect protection through improved mood, sleep, and activity levels, though definitive proof of long-term neuroprotection remains unseen.

While stimulant medications remain the most effective short-term treatment for ADHD, their benefits appear to wane once medication use stops, and evidence of long-term neuroprotection is still absent. This limitation underscores the need for broader treatment strategies that not only reduce symptoms but also strengthen the cognitive and behavioral mechanisms that support attention, organization, and emotional regulation over time. Behavioral interventions such as CBT are particularly promising in this regard. By targeting sleep, mood, and daily routines, domains closely linked to both ADHD outcomes and cognitive aging, CBT may complement pharmacologic therapy and promote lasting brain health

through modifiable pathways.

5. Cognitive Behavioral Therapy and Combined Treatment Approaches

Although stimulant use may be associated with reduced dementia risk, important limitations remain. Most existing studies rely on nonrandomized or short duration designs, which weakens causal interpretation. Dementia incidence has not been directly evaluated in stimulant trials, and most clinical studies focus on younger or middle aged adults rather than older populations where dementia risk is most relevant. In addition, comorbid conditions, lifestyle factors, and medication adherence are rarely controlled in full. Future research should therefore test whether stimulants, when combined with nonpharmacologic interventions such as CBT, exercise, and sleep optimization, could provide sustained long-term cognitive protection. This raises the broader question of whether combined treatment approaches, rather than medication alone, offer the most promising path toward safeguarding brain health across the lifespan.

CBT is highlighted in this review because it is the most thoroughly evaluated behavioral intervention for adults with ADHD. Multiple randomized controlled trials and meta-analyses show that CBT produces consistent improvements in attention, organization, and emotional regulation when compared with other behavioral approaches such as psychoeducation, coaching, or mindfulness training. [21] Its structured, skills-based format helps individuals develop daily routines and coping strategies that extend beyond immediate symptom management and may support long-term cognitive and emotional health. Traditional in-person CBT tends to yield slightly stronger outcomes and lower dropout rates, especially when personalized feedback is important, whereas internet-delivered programs show similar symptom improvement with better accessibility and sustained engagement [13] [22]. Taken together, these findings support CBT as the most empirically grounded and adaptable behavioral framework for integration with medication in ADHD treatment.

Current evidence suggests that CBT, particularly when combined with low-dose stimulant medication, improves ADHD symptom control more than medication alone and may plausibly contribute to long-term brain health through indirect pathways such as improved sleep and mood. However, most trials focus on symptom reduction rather than dementia outcomes, so the implications for brain health remain preliminary.

A 2024 meta-analysis of six RCTs found that adults receiving CBT alongside medication experienced greater reductions in ADHD symptoms compared to those receiving medication alone, with benefits lasting at least three months and declining by six to nine months [14]. Complementary studies of internet-delivered CBT also reported improvements sustained for up to twelve months on validated symptom scales [13]. Although these effects are clinically meaningful, they are based on relatively small and heterogeneous samples and symptoms, rather

than broader neurocognitive decline.

Beyond ADHD-specific outcomes, evidence from sleep and lifestyle interventions adds support for CBT's broader relevance to brain health. Trials of CBT for insomnia in older adults demonstrate improved sleep and modest gains in cognitive performance [23], while multidomain prevention programs such as the Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGER) show that coordinated lifestyle modification, which includes physical activity, blood pressure control, smoking cessation, and depression treatment, can preserve or improve cognition over two years [24].

Together, these studies support the idea that CBT and combined CBT plus low-dose medication enhance near-term functioning, and may indirectly strengthen long term cognitive resilience. ADHD-specific trials show improved symptom control and daily functioning, while sleep and lifestyle interventions address established dementia risk factors. Both lines of evidence suggest that CBT may help build "cognitive reserve", the brain's ability to withstand aging and pathology [8] [14]. However, these findings are indirect. in organization, sleep, and mood plausibly reduce risk, but direct evidence of dementia prevention remains absent.

Major gaps remain. Most ADHD CBT trials are short in duration, modest in size, and focused on younger adults, leaving many dementia related variables untested. Sleep and multidomain interventions are more promising but still rarely extend beyond two years, and few track dementia incidence directly. In addition, heterogeneity in CBT format and intensity complicates comparisons, and the role of combining CBT with pharmacotherapy in older ADHD populations remains unexamined. Future research should therefore involve pragmatic mid- to late-life trials that integrate CBT, low-dose medication, physical activity, and vascular-risk management, using cognitive endpoints to determine whether combined approaches yield lasting benefits for brain health.

6. Summary and Future Directions

ADHD, long recognized as a childhood neurodevelopmental disorder, may also carry implications for brain health across the lifespan. Large-scale epidemiological studies consistently link ADHD to an increased risk of MCI and dementia, though interpretations differ. Evidence from the Israeli cohort suggests that ADHD itself may elevate risk [9] [10], while findings from Sweden's registry point to psychiatric comorbidities as the primary contributors [16]. Stimulant medications, the cornerstone of first-line treatment, show no indication of increasing dementia risk and may even provide limited protection. Apparent protection remains unproven and could reflect residual confounding or reverse-causation artifacts. Although cognitive gains are reliable during active use, they do not persist once medication is discontinued, suggesting that stimulants primarily offer symptomatic benefit [18].

CBT, especially when combined with low dose stimulants, has been shown to improve symptom control, sleep, mood, and daily functioning while indirectly

addressing modifiable dementia risk factors [13] [14] [21] [22]. This dual strategy is especially compelling for individuals with ADHD, who face an elevated risk of later cognitive decline. By managing core symptoms and targeting sleep and mood, combined treatment may help strengthen cognitive reserve and promote brain resilience. Taken together, current evidence supports the idea that although no ADHD therapy has been proven to prevent dementia, a combined approach using stimulants and CBT represents a more sustainable and theoretically protective strategy than medication alone. represents a more sustainable strategy for symptom control and a theoretically plausible route to influencing modifiable risk factors, though causal neuroprotection has not been established.

Addressing the remaining gaps will require longitudinal studies that follow mid- to late-life ADHD populations, integrate cognitive endpoints, and examine how targeting sleep, depression, and lifestyle factors influences brain health trajectories. Such research could not only clarify the ADHD-dementia link but also establish whether combined interventions can shift brain-health outcomes across the lifespan. One promising design would be a randomized, longitudinal trial testing the combined effects of CBT and low-dose stimulants on measures of cognitive aging. By comparing combined therapy with medication alone and CBT alone, researchers could determine whether dual treatment enhances executive function, preserves attention networks, and reduces early markers of neurodegeneration. This approach would move the field beyond symptom management toward understanding whether ADHD treatment strategies can play a preventive role in cognitive decline and dementia.

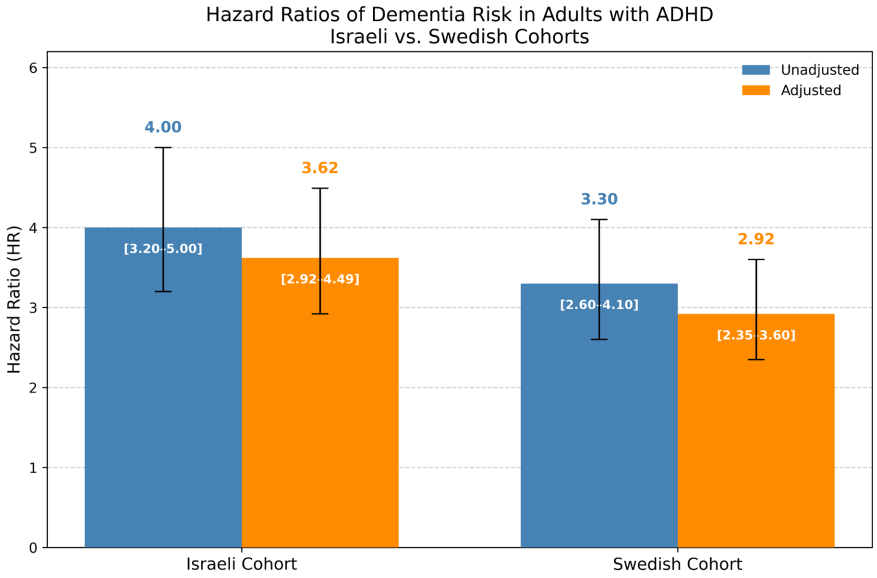

Figure 1. Hazard ratios of dementia risk in adults with ADHD: Israeli vs. swedish cohorts (Unadjusted vs. Adjusted).

This bar chart (**Figure 1**) compares unadjusted (blue) and covariate-adjusted (orange) hazard ratios (HR) for dementia in adults with ADHD from two large

national cohorts. Error bars represent 95% confidence intervals, showing that while adjustment attenuates the estimates, both populations demonstrate a robust association between ADHD and increased dementia risk.

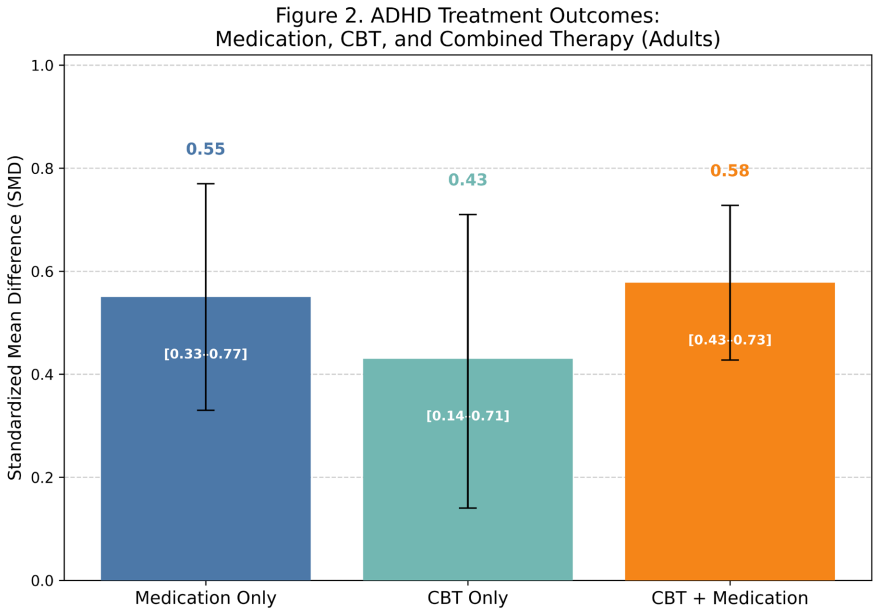

Figure 2. ADHD treatment outcomes: Medication, CBT, and combined therapy in adults.

Bar graph (**Figure 2**) showing standardized mean differences (SMD) with 95% confidence intervals (CI) for symptom improvement across three treatment modalities. Medication alone produced a moderate effect (SMD = 0.55; 95% CI 0.33 - 0.77), CBT alone yielded a comparable improvement (SMD = 0.43; 95% CI 0.14 - 0.71), and the combination of CBT with medication demonstrated the strongest overall efficacy (SMD = 0.58; 95% CI 0.43 - 0.73). Data were derived from meta-analyses and randomized controlled trials [25]-[32]. The figure highlights that multimodal therapy combining pharmacological and behavioral interventions yields the most substantial improvement in adult ADHD symptomatology.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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