Statin Therapy, Memory, and Cognition: An Updated Narrative Review

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Abstract

Concerns that statins may impair memory and cognition have persisted since regulatory agencies highlighted rare post-marketing reports of confusion and reversible cognitive symptoms. At the same time, growing observational and meta-analytic evidence suggests that statin therapy may reduce the risk of dementia, particularly Alzheimer's disease, via vascular and pleiotropic mechanisms. This narrative review synthesizes updated data on the relationship between statin use, cognitive performance, and incident dementia, focusing on randomized controlled trials, large prospective cohorts, meta-analyses, and recent mechanistic work. Overall, randomized trials and systematic reviews have not demonstrated consistent evidence of statin-induced cognitive decline, even with intensive low-density lipoprotein cholesterol (LDL-C) lowering. Several large observational studies and recent meta-analyses report a neutral or modestly protective association between statins and dementia risk, with some signals that hydrophilic statins and long-term exposure may confer greater benefit. Small subgroups, including highly susceptible individuals or those on specific lipophilic agents at high doses, may experience idiosyncratic, largely reversible cognitive symptoms. Current evidence supports continuation or initiation of statins when indicated for cardiovascular prevention, with individualized assessment of cognitive complaints, vascular risk, age, frailty, and patient preferences.

Keywords: statins, memory, cognition, dementia, Alzheimer's disease, LDL cholesterol, hydrophilic statins, lipophilic statins

Introduction

Statins are among the most widely prescribed drugs worldwide, primarily for reduction of LDL-C and prevention of atherosclerotic cardiovascular disease. Because most statin users are middle-aged and older adults, who are also the group at highest risk for cognitive decline and dementia, the potential cognitive effects of statins have received intense scrutiny (Jamshidnejad-Tosaramandani et al., 2022). Early case reports and small trials prompted regulatory warnings about possible memory loss and confusion, but the overall clinical significance of these signals has remained controversial.

Over the past decade, a large body of evidence has accumulated from randomized controlled trials (RCTs), prospective cohorts, and meta-analyses. Many of these studies suggest that statins are cognitively neutral or even protective with respect to dementia risk, while a minority report possible harm in selected subgroups (Swiger et al., 2013; Samaras et al., 2019; Zhou et al., 2021; Olmastroni et al., 2022; Du et al., 2025). At the same time, mechanistic work illustrates how statins might plausibly exert both beneficial and adverse effects on brain structure and function, through cholesterol-dependent and cholesterol-independent pathways (Jamshidnejad-Tosaramandani et al., 2022).

This review summarizes updated evidence on statins, memory, and cognition, with particular emphasis on data published in the last 5–7 years. We integrate mechanistic insights, regulatory perspectives, short-term cognitive outcomes, and long-term dementia risk, and we highlight implications for clinical decision-making in older adults.

Biological Rationale: Why Statins Might Harm or Help Cognition

Statins inhibit HMG-CoA reductase, decreasing cholesterol synthesis and downstream isoprenoid production. Because cholesterol is critical to synaptic integrity, myelin structure, and neuronal membrane microdomains, profound lipid lowering could theoretically impair neuronal function or plasticity. Experimental work suggests that excessive cholesterol depletion can alter synaptic vesicle formation, impair protein trafficking, and promote aggregation of misfolded proteins in neuronal models.

Conversely, statins also exert numerous pleiotropic effects that might protect the brain. They improve endothelial function, enhance nitric oxide bioavailability, reduce oxidative stress, and attenuate neuroinflammatory signaling. Statins may lower cerebrovascular event risk, decrease white matter lesion burden, and improve cerebral perfusion, thereby indirectly preserving cognition (Jamshidnejad-Tosaramandani et al., 2022). Some preclinical data also indicate that statins can reduce amyloid-β production, enhance clearance, and modulate tau phosphorylation, potentially reducing Alzheimer-type pathology.

Whether the net effect of statins on cognition is harmful, neutral, or beneficial likely depends on several modifying factors: baseline vascular risk, age at statin initiation, statin potency and lipophilicity, treatment duration, genetic background (for example, APOE ε4), blood–brain barrier integrity, and concomitant medications. These variables help explain why early pharmacovigilance signals and small trials suggested harm, while later large-scale studies generally do not.

Regulatory Perspective and Early Safety Concerns

In 2012, the U.S. Food and Drug Administration (FDA) updated statin labels to include information about rare, non-serious, and generally reversible cognitive adverse events such as memory loss, forgetfulness, and confusion. This action was based largely on spontaneous post-marketing reports and small studies rather than definitive trial data. Subsequent review of randomized trial and observational datasets by the FDA concluded that available evidence did not suggest that statin-associated cognitive changes were common or led to progressive or clinically significant decline.

The regulatory message has gradually shifted from concern to reassurance: clinicians are advised to be aware of the possibility of idiosyncratic cognitive symptoms, to consider statins as a potential cause in patients with new-onset confusion or memory complaints, and to manage these cases pragmatically (for example, dose reduction, switch to a different agent, or trial discontinuation). However, broad avoidance or discontinuation of statins because of cognitive fears is not supported by the bulk of evidence.

Short-Term Cognitive Outcomes in Randomized and Prospective Studies

1 Randomized trials and short-term cognition

A landmark systematic review and meta-analysis of RCTs and prospective studies by Swiger and colleagues found no consistent evidence of short-term cognitive harm from statins. Across 25 randomized trials, statin therapy did not significantly impair global cognition, attention, or memory performance compared with placebo (Swiger et al., 2013).

More recently, an American Heart Association scientific statement reviewing 25 RCTs including more than 46,000 participants concluded that statin use was not associated with increased adverse cognitive events or worsening of cognitive test scores compared with placebo (Goldstein et al., 2023). These results apply across a range of LDL-C reductions, including intensive lowering.

The ASPREE cognitive ancillary analyses, reported by Zhou and colleagues, examined older adults (≥65 years) followed for approximately 5 years. Statin use was not associated with differences in incident dementia,

Table 1. Proposed beneficial and adverse mechanisms of statins on cognition

Domain	Potential Beneficial Effects	Potential Adverse Effects
Vascular	Reduced LDL-C, less atherosclerosis and stroke, improved cerebral perfusion	Excessive LDL-C lowering in very low-risk patients might not yield additional benefit
Neuroinflammation	Reduced microglial activation and inflammatory cytokines	Possible immune modulation with uncertain long-term effects
Amyloid and tau	Reduced isoprenoids, less amyloid precursor protein processing to Aβ; possible tau modulation	Very low membrane cholesterol could theoretically disrupt synaptic handling of proteins
Synaptic function	Improved endothelial health, better neurovascular coupling	Excessive cholesterol depletion may impair synaptic vesicle formation and plasticity
Mitochondria and energetics	Improved vascular supply may support neuronal metabolism	Reduced coenzyme Q10 and mitochondrial function at high doses in susceptible individuals

Table 2. Representative randomized or prospective studies of statins and short-term cognition

Study/Type	Population	Exposure	Main Cognitive Finding
Swiger et al., 2013 (meta-analysis of RCTs)	Adults without baseline cognitive disease	Various statins vs placebo	No significant short-term cognitive impairment
Samaras et al., 2019 (prospective cohort)	Older adults, 6-year follow-up	Statin ever-use vs never-use	No worsening of memory or global cognition; similar brain volumes
Zhou et al., 2021 (ASPREE cohort)	Adults ≥65 years	Baseline statin therapy vs none	No difference in incident dementia, MCI, or cognitive decline
Evolocumab add-on RCT	High-risk CVD patients on statins	Evolocumab vs placebo	No adverse effect on cognitive performance despite very low LDL-C

mild cognitive impairment (MCI), or decline in individual cognitive domains versus non-use (Zhou et al., 2021). Cognitive trajectories over time were similar in statin users and non-users.

2 Brain imaging and structural outcomes

Samaras and colleagues evaluated statin use, cognition, and brain volumes in an elderly cohort over 6 years, with MRI assessments at baseline and 2-year follow-up. Statin users did not experience greater decline in memory, global cognition, or hippocampal and parahippocampal volumes than non-users; in some analyses, statin exposure was associated with trends toward slower decline in certain cognitive measures (Samaras et al., 2019).

Other analyses of aggressive LDL-C lowering, including the addition of non-statin agents such as PCSK9 inhibitors, likewise have not demonstrated detrimental cognitive effects. For example, in a large RCT of evolocumab added to statin therapy, there were no significant differences in cognitive outcomes between treatment and placebo groups despite very low LDL-C levels.

Long-Term Dementia Risk: Observational Evidence and Meta-Analyses

Because dementia develops over many years, most data on statins and long-term cognitive outcomes come from observational cohorts and meta-analyses rather than RCTs with dementia endpoints.

A 2022 meta-analysis of observational studies by Olmastroni and colleagues, including 36 studies and more than one million individuals, found that statin use was associated with a lower risk of all-cause dementia (odds ratio [OR] ≈ 0.80) and Alzheimer's disease (OR ≈ 0.68) compared with non-use. These findings were broadly consistent across sexes and statin types, although residual confounding cannot be excluded.

A more recent systematic review and updated meta-analysis published in 2025 reported that statin therapy was associated with a 21% reduction in risk of all-cause dementia and a 29% reduction in Alzheimer's disease. Protective effects were more pronounced in long-term users, individuals with diabetes, and certain Asian populations (Du et al., 2025; Westphal Filho et al., 2025). Hydrophilic statins (for example, pravastatin, rosuvastatin) may have slightly stronger protective associations than lipophilic statins, possibly because of differing blood–brain barrier penetration and vascular selectivity (Jamshidnejad-Tosaramandani et al., 2022; Belessiotis-Richards et al., 2025). However, other analyses, including the ASPREE study, have not found large differences between lipophilic and hydrophilic agents.

At the same time, not all cohort data show benefit. Some recent emulated target trials and large health system cohorts have reported essentially neutral associations between statin initiation and dementia incidence, suggesting that any protective effect is modest and potentially confined to specific subgroups (Zimmerman et al., 2025).

Statin Properties and Modifying Factors

1 Lipophilic versus hydrophilic statins
Lipophilic statins (for example, simvastatin, atorvastatin) more readily cross the blood–brain barrier and penetrate neuronal tissue, whereas hydrophilic statins (for example, pravastatin, rosuvastatin) are more hepatoselective. It has been hypothesized that lipophilic drugs might carry greater risk of cognitive side effects but also greater potential for direct neuroprotection.

Meta-analytic and cohort data offer mixed results. Some studies suggest hydrophilic agents are associated with slightly larger reductions in dementia risk than lipophilic agents, while others find no meaningful difference. Clinically, in patients who report cognitive complaints on a lipophilic statin, switching to a hydrophilic agent is a reasonable pragmatic strategy.

2 Dose, potency, and treatment duration
Higher-intensity statin regimens lower LDL-C more effectively and provide greater cardiovascular protection, but questions have been raised about whether very aggressive LDL-C lowering might harm the brain. The 2023 AHA statement reviewing RCTs of high-intensity statins and non-statin add-on therapies concluded that studies have not demonstrated increased risk of cognitive impairment, Alzheimer's disease, or hemorrhagic stroke with very low LDL-C.

Observational work suggests that longer statin exposure is associated with larger relative reductions in dementia risk, consistent with a cumulative vascular benefit model. Very short-term use, by contrast, is unlikely to meaningfully alter dementia trajectories and may be more susceptible to reporting bias regarding adverse events.

3 Age, vascular risk, and APOE genotype
Age at statin initiation is crucial: midlife statin therapy in the context of high LDL-C and other vascular risk factors is more likely to influence long-term brain health than late-life initiation in very old, frail individuals with established neurodegeneration. The benefit–risk balance may shift toward de-intensification or deprescribing in patients with advanced frailty, limited life expectancy, or advanced dementia.

Genetic factors such as APOE ε4 status may also modify response, although findings are inconsistent. Some data suggest that APOE ε4 carriers derive greater benefit from LDL-C lowering with respect to dementia risk, while others show no substantial interaction (Jamshidnejad-Tosaramandani et al., 2022).

Statins Across the Cognitive Spectrum

1 Normal cognition and subjective cognitive complaints
In cognitively normal adults, multiple RCTs and prospective studies indicate that statins do not accelerate decline in memory, executive function, or global cognition. However, a small subset of patients may experience subjective memory problems or confusion temporally related to statin initiation or dose escalation. In such cases, a trial of dose reduction, switch to a different statin, or temporary discontinuation can help clarify causality. If symptoms clearly resolve and recur with rechallenge, long-term avoidance or alternative lipid-lowering strategies may be appropriate.

2 Mild cognitive impairment
Data on statins in individuals with MCI are mixed. Some cohort studies suggest that statin use is associated with slower conversion from MCI to dementia, particularly in patients with substantial vascular comorbidity, while others show no effect. The heterogeneity of MCI etiologies (vascular, degenerative, mixed) complicates interpretation. In practice, most guidelines do not recommend discontinuing statins solely because of MCI; instead, they emphasize vascular risk reduction as part of a multifactorial dementia-prevention strategy.

Table 3. Selected meta-analyses and large observational studies of statins and dementia risk

Study	Design	Key Result
Swiger et al., 2013	Systematic review/meta-analysis (RCTs and cohorts)	No evidence of cognitive harm; some suggestion of reduced dementia risk
Olmastroni et al., 2022	Meta-analysis of observational studies	Statins associated with reduced dementia (OR ≈ 0.80) and AD (OR ≈ 0.68)
Du et al., 2025	Updated meta-analysis of 42 cohorts	Statin use linked to 21% lower dementia risk and 29% lower AD risk
Westphal Filho et al., 2025	Systematic review/meta-analysis	Statin users had lower dementia risk (HR ≈ 0.86); hydrophilic statins slightly more protective
Large Korean cohort 2025	Population-based study with LDL-C stratification	Low LDL-C associated with 26% lower dementia risk; statin use added ~13% additional risk reduction

3 Established dementia

In patients with established Alzheimer's disease or vascular dementia, statins are primarily used for cardiovascular indications, not as disease-modifying cognitive therapies. Observational studies of statin continuation in dementia suggest neutral to modestly favorable associations with cognitive trajectory and mortality, but confounding by indication and survivor bias are substantial. Recent work has reported that statin use in some cohorts of Alzheimer's patients is associated with higher cognitive scores at diagnosis, suggesting a "healthy user" or vascular benefit effect rather than direct neuroprotection (Petek et al., 2025).

In advanced dementia or end-of-life care, deprescribing statins may be appropriate when the time to cardiovascular benefit exceeds expected survival, or when pill burden and swallowing difficulties pose challenges.

Remaining Controversies and Emerging Data

Despite the overall reassuring picture from RCTs and most observational studies, several lines of evidence keep the debate active:

• Pharmacovigilance signals have repeatedly detected an over-representation of cognitive adverse event reports with some lipophilic statins, particularly atorvastatin, compared with other drugs, although causality is uncertain.(@ WalshMedical)

• Mendelian randomization analyses have suggested potential negative associations between genetically proxied HMG-CoA reductase inhibition and specific cognitive test scores in some populations, though these effects have not translated into increased Alzheimer's disease risk.(SpringerLink)

• Mechanistic models indicate that extreme cholesterol depletion may promote aggregation of certain neuronal proteins and alter membrane mechanics, raising theoretical concerns in highly susceptible individuals.(arXiv)

At the same time, powerful new meta-analyses and population-based cohorts are strengthening the case for a modest protective effect of statins against dementia, particularly when started in midlife and used for many years (Du et al., 2025; Westphal Filho et al., 2025; Goldstein et al., 2023). These apparently conflicting signals underscore the need for adequately powered RCTs with prespecified cognitive endpoints, long follow-up, and careful phenotyping of dementia subtypes.

1 Evidence Suggesting Potential Long-Term Cognitive Harm

Although large randomized trials and most meta-analyses are broadly reassuring, a number of more recent clinical and genetic studies have suggested that long-term statin exposure could be linked to subtle cognitive impairment in some patients. These findings do not overturn the overall evidence base, but they are important signals that deserve careful discussion.

One prospective single-arm study in routine clinical practice evaluated 213 adults on moderate- or high-intensity statin therapy using the Modified Mini-Mental State Examination (3MS). Cognitive impairment (3MS < 79) was present in 17.8% of participants, a prevalence higher than expected for the general U.S. population of similar age (mean 55.4 years) (Roy et al., 2020). High-intensity statin users had markedly more cognitive impairment than moderate-intensity users (41.7% vs. 5.7%), and there was a weak but significant negative correlation between duration of statin therapy and cognitive score, suggesting worse performance with longer exposure ($r = -0.28$) (Roy et al., 2020). Although the study lacked a non-statin control group and was vulnerable to confounding by indication, it remains one of the clearest clinical signals that long-term, high-intensity statin use may be associated with measurable cognitive deficits.

Other observational data have also pointed toward a possible adverse association. A retrospective analysis cited in Roy's paper reported that 39.9% of statin-treated patients met criteria for dementia or cognitive impairment compared with 18.9% of non-users in a sample of 3,500 patients, although causality could not be established and adjustment for vascular risk factors was limited (Roy et al., 2020). Several earlier cohort studies likewise described higher rates of cognitive symptoms or psychological disturbances in statin users, including associations with depressive symptoms and, in very small samples, behavioral changes such as irritability or aggression, but these findings were inconsistent and often methodologically weak (Jamshidnejad-Tosaramandani et al., 2022).

Genetic evidence has also raised questions about long-term cognitive safety. A Mendelian randomization study using variants in the HMGCR gene as a proxy for lifelong statin-like inhibition found that genetically predicted HMG-CoA reductase inhibition was associated with modestly worse performance on certain neurocognitive traits, such as reaction time and fluid intelligence, while PCSK9 inhibition was not clearly harmful (Rosoff et al., 2022). A more recent multi-omic Mendelian randomization analysis of HMGCR and LDLR inhibition similarly reported small adverse associations with some cognitive measures, again with effect sizes that were statistically significant but modest and of uncertain clinical relevance (Wen et al., 2025). These genetic findings suggest that, in theory, very long-term pharmacologic inhibition of HMG-CoA reductase might have subtle adverse cognitive effects in some individuals, even if such effects are difficult to detect in conventional clinical trials.

Emerging imaging and preprint data also contribute to the discussion. For example, a voxel-based morphometry study (preprint) reported structural brain differences and poorer cognitive performance in some statin users compared with non-users, though the authors found no clear dose–response effect and emphasized that reverse causation and confounding could not be excluded (Liew et al., 2025). A nuclear medicine abstract suggested that cognitively impaired subjects using lipophilic statins with normal total cholesterol experienced a faster progression to dementia on PET imaging than non-users in a small sample (Padmanabham et al., 2024). These reports are provocative but preliminary, with limited sample sizes and short follow-up.

From a mechanistic standpoint, several experimental studies summarized by Jamshidnejad-Tosaramandani and colleagues have found that statins can reduce levels of brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), and irisin, all of which support synaptic plasticity and neuronal survival (Jamshidnejad-Tosaramandani et al., 2022). Excessive depletion of membrane cholesterol in neuronal cultures has also been shown to disrupt lipid rafts, impair neurotransmitter release, and reduce long-term potentiation, providing a plausible biological mechanism for cognitive side effects in particularly susceptible brains.

A recent narrative review by Kazibwe and colleagues explicitly highlights this duality: early and some newer studies suggest an increased risk of cognitive impairment or dementia with statins, while more recent, larger epidemiologic analyses predominantly show neutral or protective associations (Kazibwe et al., 2024). The authors conclude that long-term statin use may have heterogeneous cognitive effects: neutral or beneficial in most patients, potentially adverse in a small subgroup defined by genetic, metabolic, or pharmacologic vulnerability.

Overall, the "negative" literature supports three cautious conclusions. First, high-intensity, long-duration statin therapy may be more likely to be associated with subtle cognitive impairment than short-term or low-intensity regimens, particularly with certain lipophilic agents (Roy et al., 2020). Second, there may be a biologically plausible link between long-term HMG-CoA reductase inhibition and small decrements in specific cognitive domains over decades, as suggested by Mendelian randomization studies (Rosoff et al., 2022; Wen et al., 2025). Third, current RCTs may be underpowered or too short to detect such subtle effects in the highly selected populations they enroll.

However, it is equally important to acknowledge substantial methodological limitations. Many of these negative studies lack appropriate non-statin control groups, are cross-sectional rather than longitudinal, or are vulnerable to confounding by indication: patients on intensive statin therapy are usually at higher vascular risk, which itself predisposes to cognitive decline. Genetic studies, while powerful, rely on assumptions that may not fully hold in complex neurocognitive phenotypes and often show very small effect sizes. Consequently, even authors of these "negative" studies generally do not recommend avoiding statins when there is a clear cardiovascular indication; instead, they advocate careful monitoring, individualized risk–benefit assessment, and more targeted long-term trials.

Practical Clinical Implications

On the basis of current evidence, several practical principles can guide clinicians:

1. Do not withhold statins solely because of fear of cognitive harm when there is a clear cardiovascular indication. The weight of evidence suggests that statins are cognitively neutral or modestly protective for most patients.

2. Assess and monitor cognition pragmatically. Ask about new memory complaints, confusion, or concentration difficulties after statin initiation or dose escalation. If symptoms arise, consider dose reduction, switching to a hydrophilic agent, or temporary discontinuation, especially when temporal association is strong.

3. Prioritize vascular prevention in midlife and early late life. Statin therapy as part of a comprehensive vascular risk-reduction strategy (blood pressure control, diabetes management, smoking cessation, physical activity) may be one of the most effective ways to reduce dementia risk at the population level.

Table 4. Selected recent evidence suggesting possible long-term cognitive harm from statins

Study / Type	Population & Exposure	Main Finding	Key Limitations
Roy et al., 2020 (clinical study)(jocmr.org)	213 adults on moderate- or high-intensity statins; 3MS testing	17.8% had cognitive impairment; higher prevalence with high-intensity therapy; weak negative correlation between duration and cognition	No non-statin control; cross-sectional; confounding by vascular risk and comorbidities
Retrospective cohort cited by Roy(jocmr.org)	~3,500 patients, statin users vs non-users	Dementia/cognitive impairment more common in statin users (39.9% vs 18.9%)	Retrospective; incomplete adjustment; indication bias likely
Rosoff et al., 2022 (Mendelian randomization)(JACC)	Genetic proxies for long-term HMGCR and PCSK9 inhibition	HMGCR inhibition associated with slightly worse reaction time and fluid intelligence; PCSK9 neutral	Genetic assumptions; small effect sizes; unclear clinical meaning
Wen et al., 2025 (Mendelian randomization)(ScienceDirect)	Genetic proxies for LDLR and HMGCR inhibition	Some adverse associations with cognitive traits	Observational genetic design; pleiotropy; minimal absolute effects
Liew et al., 2025 (preprint imaging study)(MedRxiv)	Statin users vs non-users; MRI voxel-based morphometry	Structural brain differences and somewhat worse cognitive performance in some statin users	Preprint; small sample; confounding; no clear dose–response
Padmanabham et al., 2024 (PET abstract)(SNM Journals)	Cognitively impaired subjects using lipophilic statins	Faster progression to dementia in some lipophilic statin users on PET imaging	Abstract only; small sample; observational; cannot infer causality

Table 5. Suggested clinical approach to statins and cognition

Scenario	Suggested Approach
Middle-aged patient with high LDL-C and multiple vascular risks	Initiate statin per guidelines; reassure about cognitive safety; emphasize lifestyle interventions
Older adult with subjective memory complaints after statin initiation	Review timing; check other causes (sleep, mood, polypharmacy); consider dose reduction or switch to hydrophilic statin; monitor
Patient with MCI and high vascular risk	Continue/initiate statin if indicated; focus on overall vascular risk reduction; monitor cognition
Advanced dementia, limited life expectancy	Discuss goals of care; consider deprescribing statin if cardiovascular benefit unlikely within remaining lifespan

4. Individualize decisions in very old, frail, or cognitively impaired patients. In individuals with advanced dementia or limited life expectancy, deprescribing statins may be appropriate, especially when pill burden is high or cardiovascular risk is relatively low.
5. Consider statin type and intensity. For patients who report cognitive symptoms, a trial of switching from a lipophilic to a hydrophilic statin at the lowest effective dose is reasonable. Intensive LDL-C lowering appears safe cognitively in high-risk patients but should be weighed against overall goals of care.

Conclusion

The relationship between statins, memory, and cognition is complex but increasingly well characterized. Early safety concerns, based largely on spontaneous reports and small studies, led to regulatory warnings and substantial public anxiety. However, a large body of evidence from randomized trials, prospective cohorts, and meta-analyses now indicates that statin therapy does not cause clinically important cognitive decline for the vast majority of users. Indeed, many observational studies suggest that statins may modestly reduce the risk of dementia and Alzheimer's disease, likely through their beneficial effects on vascular risk factors and possibly through direct neurobiological mechanisms.

Some individuals may experience idiosyncratic, largely reversible cognitive symptoms related to statin use, and these cases should be managed pragmatically with dose adjustment or alternative therapies. In very old or frail patients, the decision to initiate or continue statins should be individualized, balancing cardiovascular benefits, cognitive status, frailty, and patient priorities.

Overall, current evidence supports the continued use of statins for appropriate cardiovascular indications, with thoughtful attention to cognitive complaints, shared decision-making, and integration of statin therapy into broader strategies for brain-healthy aging.

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