

BIOME GUIDE PROTOCOL

Cognitive Health & Dementia Prevention Protocol

Evidence-based probiotic, supplement, and dietary strategies to protect your brain from the inside out

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16 peer-reviewed citations

What This Protocol Addresses

Cognitive decline is not an inevitable part of aging. From subjective cognitive decline (SCD) to mild cognitive impairment (MCI) to Alzheimer's disease (AD), neurodegeneration follows a continuum where early intervention can slow or even prevent progression. AD alone affects over 55 million people worldwide, and numbers are projected to triple by 2050. Emerging research reveals that the gut-brain axis plays a central role in this process, with gut dysbiosis, neuroinflammation, and metabolite imbalances driving cognitive decline years before diagnosis.

The Gut-Brain Axis: Five Pathways to Cognitive Decline

Pathway	Mechanism	Evidence
Neuroinflammation / LPS	Gut-derived LPS crosses weakened BBB, activates microglia, triggers TNF-alpha/IL-6/IL-1beta cascade	Wang 2022: PSCI patients had higher Enterobacteriaceae, LPS, and inflammatory markers
SCFA / Butyrate depletion	Reduced butyrate weakens BBB integrity, impairs colonocyte energy, and reduces Treg function	Wang 2022: sodium butyrate rescued cognitive impairment in FMT mice
Neurotransmitter synthesis	Gut bacteria produce >90% of serotonin, GABA, dopamine precursors; dysbiosis disrupts supply	Chen 2023: Lactobacillus decreasing from SCD stage onward
Vagus nerve signaling	Bidirectional gut-brain communication; microbial metabolites modulate vagal afferents	Siripaopradit 2024: probiotics reduced TNF-alpha (SMD -2.08), IL-6 (SMD -2.98)
Amyloid-beta clearance	Gut dysbiosis impairs BBB P-glycoprotein transport and amyloid clearance	Fan 2025: Akkermansia muciniphila correlated with reduced amyloid burden

Early Warning: Dysbiosis Begins Before Diagnosis

Chen et al. 2023 (Eur J Neurol) analyzed 34 studies and found that gut microbiome abnormalities are present even at the subjective cognitive decline (SCD) stage, well before clinical MCI or AD diagnosis. Lactobacillus shows a decreasing trend starting at SCD, while Enterobacteriaceae progressively increase through MCI to AD. This means the gut offers a window for intervention years before cognitive symptoms become severe.

Gut Dysbiosis in Cognitive Decline

Three independent meta-analyses confirm that people with AD and MCI harbor a distinct, disrupted gut microbial profile compared to healthy age-matched controls. The pattern is consistent: reduced microbial diversity, depleted beneficial taxa (Bifidobacterium, Lactobacillus, Ruminococcaceae), and enrichment of pro-inflammatory species.

Meta-Analysis	Studies / Participants	Key Findings
Jemimah 2023 (PLoS One)	17 studies; 679 AD/MCI vs 632 controls	Decreased species richness in AD; Bacteroidetes geography-dependent (higher in US, lower in China); Phascolarctobacterium increased in MCI only -- dysbiosis starts early
Chen 2023 (Eur J Neurol)	34 case-control studies across SCD, MCI, AD	Chao1 and Shannon diversity significantly lower in AD; Firmicutes lower in AD and MCI; Enterobacteriaceae increasing, Ruminococcaceae/Lachnospiraceae/Lactobacillus decreasing through AD continuum
Hung 2022 (Aging)	11 studies; 378 HC vs 427 AD spectrum	AD patients had reduced diversity; more Bacteroidetes/Proteobacteria; less Firmicutes/Bifidobacterium/Lactobacillus; gradient changes from MCI to AD stage in key taxa

Why This Matters for Your Protocol

The consistent depletion of Bifidobacterium, Lactobacillus, and SCFA-producing taxa (Ruminococcaceae, Lachnospiraceae) across all three meta-analyses provides the rationale for targeted probiotic supplementation and prebiotic fiber to restore these beneficial populations. The fact that dysbiosis begins at the SCD/MCI stage means early intervention has the greatest potential impact.

Probiotics for Cognitive Function: The Evidence

Four meta-analyses of randomized controlled trials demonstrate that probiotic supplementation significantly improves cognitive function in people with existing cognitive impairment. The effect is most pronounced in those who already have MCI or AD, with consistent findings across independent research groups.

Meta-Analysis	Studies / Participants	Overall Effect	Key Subgroup Finding
Liu 2023 (Nutr Rev)	RCTs; cognitively impaired	SMD = 0.64 (95% CI 0.15-1.12)	AD/MCI subgroup: SMD = 1.34 (p < 0.01) -- large effect size
Guo 2025 (Eur J Clin Nutr)	21 RCTs; lifespan analysis	Older adults: SMD = 0.40 (p = 0.004)	Memory: SMD = 0.51 (p = 0.0002); processing speed: SMD = 0.37 (p = 0.006)
Peng 2025 (Gen Hosp Psychiatry)	15 RCTs; 994 MCI/AD participants	SMD = 0.57 (95% CI 0.19-0.94, p = 0.003)	12+ weeks: SMD = 0.73 (p = 0.0009); high dose: SMD = 0.93 (p < 0.01)
Liu 2025 (J Food Sci)	4 double-blind RCTs; 251 AD patients 65+	SMD = 0.67 (95% CI 0.14-1.19, p < 0.05)	Formulation type and baseline severity influence outcomes

Optimal Probiotic Parameters for Cognitive Health

Across all four meta-analyses, the strongest results come from: (1) Lactobacillus and Bifidobacterium multi-strain formulations, (2) supplementation for at least 12 weeks (Peng 2025: SMD nearly doubles from 0.57 to 0.73 at 12+ weeks), (3) doses of at least 10 billion CFU daily (Peng 2025: SMD = 0.93 with doses >1x10⁹ CFU), and (4) targeting people with existing cognitive impairment rather than healthy individuals (Liu 2023: probiotics may be ineffective in cognitively healthy populations).

Probiotics for Prevention vs Treatment

An important distinction: probiotics show their strongest cognitive benefits in people who already have MCI or AD. For cognitively healthy adults, the evidence for prevention is less clear. However, given that gut dysbiosis begins at the SCD stage, proactive supplementation combined with diet and lifestyle may help maintain microbial balance before decline sets in.

Spotlight Strain: Bifidobacterium breve MCC1274

Among the probiotic strains tested for cognitive health, B. breve MCC1274 (also known as A1) stands out with two dedicated RCTs demonstrating cognitive improvement and, critically, suppression of brain atrophy in MCI patients.

Landmark Trial: Asaoka et al. 2022 (J Alzheimers Dis)

Parameter	Detail
Design	24-week double-blind, placebo-controlled RCT
Participants	130 patients aged 65-88 with suspected MCI
Intervention	B. breve MCC1274 at 2×10^{10} CFU daily
Cognitive result	Significant improvement in ADAS-Jcog 'orientation' subscale at 24 weeks
MMSE result	'Orientation in time' and 'writing' improved in lower-baseline subgroup (MMSE <25)
Brain atrophy	VSRAD MRI scores worsened in placebo; probiotic SUPPRESSED brain atrophy progression
Significance	First RCT to demonstrate probiotic-mediated suppression of brain atrophy in MCI

Supporting Trial: Kobayashi et al. 2019 (Benef Microbes)

In an earlier 12-week double-blind RCT of 121 elderly with memory complaints, B. breve MCC1274 significantly improved immediate memory (RBANS subscale) and MMSE total score in subjects with lower baseline cognitive scores. Safety was confirmed with no adverse effects. This established the preliminary efficacy that the larger Asaoka 2022 trial then confirmed and extended.

Why B. breve MCC1274 Matters

This is the only probiotic strain with RCT evidence showing suppression of brain atrophy (measured by MRI VSRAD) in MCI patients. The mechanism likely involves reducing neuroinflammation and supporting BDNF production through the gut-brain axis. Available commercially as Morinaga's 'Memory Bifidus' in select markets, or as a component of multi-strain cognitive formulations.

The Gut-Brain Axis: Butyrate and LPS

Two key metabolites mediate the causal relationship between gut dysbiosis and cognitive impairment: lipopolysaccharide (LPS), a pro-inflammatory endotoxin from gram-negative bacteria, and butyrate, a neuroprotective short-chain fatty acid produced by beneficial gut bacteria.

Causal Evidence: Wang et al. 2022 (J Neuroinflammation)

This landmark study provided the first causal evidence linking gut microbiota to cognitive impairment. Among 83 stroke patients, those who developed post-stroke cognitive impairment (PSCI) had significantly higher gut Enterobacteriaceae, circulating LPS, and inflammatory markers, alongside lower fecal butyrate. When fecal microbiota from PSCI patients was transplanted into mice, the recipients developed BBB disruption, microglial activation, hippocampal neuronal apoptosis, and amyloid-beta deposition. Critically, sodium butyrate supplementation rescued all of these detrimental changes.

Metabolite	Role in Cognition	Intervention Strategy
LPS (harmful)	Crosses weakened BBB; activates TLR4 on microglia; triggers neuroinflammatory cascade; promotes amyloid-beta deposition	Reduce gram-negative bacteria via probiotics and prebiotics; strengthen gut barrier with L-glutamine and butyrate
Butyrate (protective)	Strengthens BBB tight junctions; reduces microglial activation; supports hippocampal neuronal survival; facilitates amyloid clearance	Sodium butyrate supplement (300-600 mg 2x/day); dietary fiber to feed butyrate producers; resistant starch from cooled potatoes, green bananas

Akkermansia muciniphila: A Protective Species

Fan et al. 2025 (Alzheimers Res Ther) used shotgun metagenomics on 119 MCI patients and 320 controls, identifying 59 key microbial species linked to AD biomarkers. Akkermansia muciniphila correlated with reduced amyloid burden, suggesting a neuroprotective role. Importantly, species within the same genus (e.g., different Bacteroides species) showed opposing effects, highlighting that genus-level analysis is insufficient -- species-level resolution matters.

Butyrate: The Neuroprotective Metabolite

Wang 2022 demonstrated that sodium butyrate supplementation rescued cognitive impairment caused by dysbiotic gut microbiota. Butyrate works through multiple mechanisms: strengthening the blood-brain barrier, reducing microglial activation, inhibiting hippocampal neuronal apoptosis, and facilitating amyloid-beta clearance. This provides direct evidence for including butyrate (via supplements or dietary fiber) in a cognitive health protocol.

Omega-3 Fatty Acids: DHA and EPA

Omega-3 fatty acids, particularly DHA (docosahexaenoic acid), are essential structural components of brain cell membranes. DHA comprises approximately 40% of polyunsaturated fatty acids in the brain and is critical for synaptic plasticity, neurogenesis, and anti-inflammatory signaling.

Landmark Evidence: Wei et al. 2023 (Am J Clin Nutr)

Analysis	Finding	Significance
ADNI cohort (6-year follow-up)	Long-term omega-3 users: 64% reduced AD risk (HR 0.36, 95% CI 0.18-0.72)	p = 0.004
Meta-analysis of 48 studies (103,651 participants)	DHA intake: RR 0.82 (I2 = 63.6%) for cognitive decline	p = 0.001
Dose-response analysis	Each 0.1 g/day of DHA or EPA: 8-9.9% lower cognitive decline risk	p < 0.0005
Blood biomarkers	Elevated plasma EPA and erythrocyte membrane DHA associated with lower cognitive decline risk	Moderate-to-high evidence

Omega-3 Dosing for Brain Health

Total omega-3: 1000-2000 mg DHA + EPA daily, with emphasis on DHA (aim for at least 500-1000 mg DHA). Take with a fat-containing meal for optimal absorption. Triglyceride-form fish oil or algae-based DHA for vegetarians. Fatty fish (salmon, sardines, mackerel) 2-3 times weekly provides additional dietary DHA. Long-term use (years) shows the strongest protective effect.

B Vitamins, Folate, and Homocysteine

Elevated homocysteine is one of the most well-established modifiable risk factors for dementia. B vitamins -- particularly folate, B12, and B6 -- lower homocysteine through one-carbon metabolism. A massive 2022 meta-analysis of 95 studies demonstrates that B vitamin supplementation slows cognitive decline, with folate emerging as the most critical nutrient.

Key Evidence: Wang et al. 2022 (Nutr Rev)

Finding	Data	Population
B vitamins slow cognitive decline (MMSE)	MD = 0.14 (95% CI 0.04-0.23)	6,155 participants across 25 RCTs
Effect with >12 months treatment	MD = 0.15 (95% CI 0.05-0.26)	3,814 participants -- duration matters
Effect in non-dementia population	MD = 0.15 (95% CI 0.04-0.25)	3,431 participants -- prevention works
Low folate and dementia risk	OR = 1.76 (95% CI 1.24-2.50)	6,654 participants -- 76% higher risk
High homocysteine and dementia risk	OR = 2.09 (95% CI 1.60-2.74)	12,665 participants -- over 2x risk
Higher dietary folate and incident dementia	HR = 0.61 (95% CI 0.47-0.78)	13,529 participants -- 39% risk reduction
B12 or B6 alone	Not significantly associated with reduced risk	Meta-analysis of cohort studies

Important: Folate Is the Key B Vitamin

While B vitamin complexes are commonly recommended, this meta-analysis reveals that folate is the primary driver of dementia risk reduction. B12 and B6 alone did not significantly reduce dementia risk. However, they work synergistically with folate for homocysteine metabolism. Recommendation: methylfolate (400-800 mcg/day) as the foundation, with B12 (500-1000 mcg methylcobalamin) and B6 (25-50 mg P5P) as supporting cofactors. Test homocysteine levels -- target below 10 mcmol/L.

The MIND Diet: Eating for Brain Health

The MIND diet (Mediterranean-DASH Intervention for Neurodegenerative Delay) combines elements of the Mediterranean and DASH diets, specifically targeting foods with the strongest evidence for neuroprotection. A landmark 2023 study in JAMA Psychiatry confirmed its association with significantly reduced dementia risk.

Landmark Evidence: Chen et al. 2023 (JAMA Psychiatry)

This study combined three prospective cohorts (Whitehall II, Health and Retirement Study, Framingham Heart Study Offspring) with 18,136 participants, plus a meta-analysis of 11 cohort studies totaling 224,049 participants (5,279 dementia cases). Higher MIND diet adherence was associated with 17% lower dementia risk (pooled HR 0.83, 95% CI 0.76-0.90). The association was consistent across sex, age, smoking status, and BMI subgroups.

MIND Diet: 10 Brain-Healthy Food Groups

Food Group	Recommended Intake	Why It Matters
Green leafy vegetables	6+ servings/week	Folate, lutein, vitamin K; strongest single food association with slower decline
Other vegetables	1+ serving/day	Diverse polyphenols and fiber for microbiome diversity
Berries	2+ servings/week	Anthocyanins cross BBB; reduce neuroinflammation and oxidative stress
Nuts	5+ servings/week	Vitamin E, healthy fats, polyphenols; anti-inflammatory
Olive oil	Primary cooking oil	Oleocanthal has ibuprofen-like anti-inflammatory action in the brain
Whole grains	3+ servings/day	Prebiotic fiber feeds SCFA-producing bacteria; sustained energy
Fish (non-fried)	1+ serving/week	DHA and EPA; strongest dietary source of brain-essential omega-3
Beans/legumes	3+ servings/week	Resistant starch, folate, fiber; feeds butyrate producers
Poultry	2+ servings/week	Lean protein; preferred over red meat
Wine (optional)	1 glass/day max	Resveratrol; moderation is key -- excess alcohol is neurotoxic

5 Food Groups to Limit

Food Group	Limit To	Rationale
Red meat	<4 servings/week	Pro-inflammatory; TMAO production from gut bacteria

Food Group	Limit To	Rationale
Butter/margarine	<1 tablespoon/day	Saturated fats promote neuroinflammation
Cheese	<1 serving/week	High saturated fat; pro-inflammatory in excess
Pastries/sweets	<5 servings/week	Blood sugar spikes; advanced glycation end-products damage neurons
Fried/fast food	<1 serving/week	Trans fats, oxidized oils; directly neurotoxic

The MIND Diet and the Microbiome

The MIND diet is inherently microbiome-supportive. Its emphasis on leafy greens, berries, whole grains, beans, and olive oil provides diverse prebiotic fiber and polyphenols that feed SCFA-producing bacteria (*Ruminococcaceae*, *Lachnospiraceae*) and *Bifidobacterium* -- the exact taxa depleted in cognitive decline. Chu et al. 2022 (*Neurosci Biobehav Rev*) confirmed that dietary patterns modulate cognitive impairment partly through gut microbiota composition.

Vitamin D and Dementia Risk

Vitamin D receptors are expressed throughout the brain, including the hippocampus and prefrontal cortex -- regions critical for memory and executive function. Low vitamin D status is consistently associated with higher dementia risk across multiple analyses.

Evidence Summary: Aghajafari et al. 2018 (BJPsych Open)

This umbrella review evaluated 11 systematic reviews (9 meta-analyses) on vitamin D, cognition, and dementia. Five out of six meta-analyses showed a positive association between low serum 25(OH)D concentrations and dementia risk. While the quality of evidence was rated moderate overall, the consistent direction of the association across independent analyses provides reasonable confidence that maintaining adequate vitamin D status is neuroprotective.

Vitamin D Action	Brain Health Mechanism
Neurotrophin regulation	Supports NGF and BDNF expression -- essential for neuronal survival and plasticity
Anti-inflammatory	Reduces neuroinflammatory cytokines (TNF-alpha, IL-6) in brain tissue
Amyloid clearance	Enhances macrophage-mediated phagocytosis of amyloid-beta plaques
Calcium homeostasis	Prevents excitotoxic neuronal damage from calcium dysregulation
Antioxidant	Upregulates glutathione and reduces oxidative stress in neurons

Vitamin D Dosing for Cognitive Protection

Aim for serum 25(OH)D of 40-60 ng/mL. Dose: 2000-4000 IU vitamin D3 daily with a fat-containing meal. Test levels at baseline and every 6 months. Up to 50% of older adults are vitamin D deficient. Vitamin D also supports the gut microbiome by strengthening intestinal barrier integrity and modulating immune responses -- creating a dual benefit for the gut-brain axis.

Curcumin and Cognitive Aging

Curcumin, the primary bioactive compound in turmeric, has potent anti-inflammatory and antioxidant properties with demonstrated ability to cross the blood-brain barrier. A 2025 meta-analysis evaluated its effects on cognitive outcomes in both preclinical and human studies.

Meta-Analysis Evidence: Yu et al. 2025 (J Prev Alzheimers Dis)

Study Type	Outcome	Effect Size
Preclinical (25 animal studies, 572 animals)	Acquisition memory in AD models	SMD = -1.78 (95% CI -2.12 to -1.43) -- strong improvement
Preclinical (25 animal studies)	Retention memory in AD models	SMD = 2.36 (95% CI 1.72-3.00) -- very strong improvement
Human RCTs (10 studies, 531 participants)	Global cognitive outcomes	SMD = 0.14 (95% CI -0.78 to 1.07) -- not significant overall
Human RCTs -- subgroup	Working memory	SMD = 1.01 (95% CI 0.15-1.87) -- significant improvement
Human RCTs -- subgroup	Processing speed	SMD = 0.37 (95% CI 0.07-0.67) -- significant improvement

Curcumin: Domain-Specific Benefits

While curcumin did not improve global cognition overall in human trials, it showed significant benefits for working memory (SMD = 1.01) and processing speed (SMD = 0.37). The strong preclinical evidence suggests the gap may be due to bioavailability limitations. Use a bioavailable formulation: curcumin phytosome (Meriva), nano-curcumin, or curcumin with piperine (20-fold absorption increase). Dose: 500-1000 mg/day of bioavailable curcumin.

Supporting Supplement Stack

Beyond the core interventions, several additional supplements support cognitive health through complementary mechanisms. This tiered approach allows you to start with the essentials and add based on individual needs and response.

Tier	Supplement	Dose	Mechanism / Notes
Tier 1 (Core)	Multi-strain probiotic (Lacto + Bifido)	20-50 billion CFU/day	Restores depleted taxa; 12+ weeks for cognitive benefit; B. breve MCC1274 if available
Tier 1	Omega-3 DHA/EPA	1000-2000 mg/day (DHA-dominant)	64% AD risk reduction with long-term use; structural brain component
Tier 1	B vitamin complex (methylfolate lead)	Folate 400-800 mcg + B12 500-1000 mcg + B6 25-50 mg	Homocysteine reduction; 39% dementia risk reduction with adequate folate
Tier 2	Vitamin D3	2000-4000 IU/day	5/6 meta-analyses link low D to dementia; target 40-60 ng/mL
Tier 2	Curcumin (bioavailable)	500-1000 mg/day	Working memory + processing speed improvements; anti-neuroinflammatory
Tier 2	Sodium butyrate	300-600 mg 2x/day	BBB protection; rescued cognitive impairment in FMT study (Wang 2022)
Tier 3	Prebiotic fiber (inulin/FOS/GOS)	5-10 g/day	Feeds SCFA producers; supports microbiome diversity; start low, increase gradually
Tier 3	Magnesium L-threonate	1000-2000 mg/day (144 mg elemental Mg)	Only form shown to cross BBB and increase brain Mg; supports synaptic density
Tier 3	Lion's mane mushroom	500-1000 mg/day	Stimulates NGF production; preliminary evidence for MCI improvement

Tiered Approach

Start with Tier 1 supplements for the first 4 weeks. Add Tier 2 in weeks 5-8 based on individual response and budget. Tier 3 supplements are optional additions for months 3+ to further optimize. All supplements should be taken consistently -- the evidence shows that duration of at least 12 weeks is needed for measurable cognitive benefit.

Phase 1: Foundation (Months 1-3)

The first three months establish the core protocol: restoring beneficial gut bacteria, reducing neuroinflammation, and providing the brain with essential nutrients for protection and repair.

Intervention	Specification	Timing
Multi-strain probiotic	Lactobacillus + Bifidobacterium; 20-50 billion CFU; include B. breve if possible	Morning, with or before breakfast
Omega-3 (DHA/EPA)	1000-2000 mg daily (DHA-dominant formulation)	With meals, split AM/PM if >1000 mg
B vitamin complex	Methylfolate 400-800 mcg + methylcobalamin 500-1000 mcg + P5P 25-50 mg	Morning with food
Vitamin D3	2000-4000 IU/day (test 25(OH)D; target 40-60 ng/mL)	With largest fat-containing meal
Curcumin (bioavailable)	500-1000 mg (phytosome or with piperine)	With food
Prebiotic fiber	Start 3 g/day, increase to 5-10 g over 2 weeks	Mixed into food or drink
MIND diet adoption	Emphasize leafy greens 6x/week, berries 2x/week, fish 1-2x/week, nuts daily	All meals
Cognitive engagement	30+ minutes daily of novel mental challenges (puzzles, language learning, music)	Consistent daily schedule
Physical activity	150 minutes/week moderate aerobic exercise (walking, swimming, cycling)	Most days of the week

Important: Medication Safety

If you are taking blood thinners (warfarin, apixaban), consult your physician before starting omega-3 or curcumin, as both have mild anticoagulant effects. If you are on cholinesterase inhibitors (donepezil, rivastigmine) or memantine for AD, this protocol is designed as adjunctive support and should not replace prescribed medications. Always inform your neurologist or primary care provider about supplements you are taking.

Phase 2: Optimize and Sustain (Months 4–6+)

After establishing the foundation, Phase 2 focuses on optimization, adding targeted supplements based on individual response, and building sustainable long-term habits.

Component	Phase 2 Adjustment	Rationale
Probiotic	Continue daily; consider adding sodium butyrate 300–600 mg 2x/day	Direct BBB and neuroprotective support beyond what probiotics alone provide
Omega-3	Maintain 1000–2000 mg; consider Omega-3 Index blood test	Target Omega-3 Index of 8–12% for optimal brain protection
B vitamins	Test homocysteine; adjust dose to keep below 10 mcmol/L	Personalized dosing based on biomarker response
Magnesium L-threonate	Add 1000–2000 mg/day if tolerated	Only magnesium form with evidence of crossing BBB; supports synaptic density
Lion's mane	Add 500–1000 mg/day (hot water extract preferred)	NGF stimulation for neuronal repair and growth
MIND diet	Deepen adherence; aim for MIND score 9–12 out of 15	Each 3-point increase = 17% lower dementia risk (Chen 2023)
Social engagement	Prioritize meaningful social interaction 3+ times/week	Social isolation is a major modifiable dementia risk factor
Sleep optimization	Target 7–8 hours quality sleep; consistent sleep/wake times	Glymphatic clearance of amyloid-beta occurs primarily during deep sleep

The Long-Term Perspective

Cognitive health is a lifelong investment. Wei et al. 2023 found that LONG-TERM omega-3 users had 64% lower AD risk, and Wang et al. 2022 showed B vitamin supplementation over 12+ months was needed for significant cognitive benefit. The MIND diet meta-analysis tracked participants for up to 24 years. Consistency over months and years, not quick fixes, is what protects the brain.

Tracking Your Progress

Monitoring cognitive function and relevant biomarkers helps you assess whether the protocol is working and guides adjustments. Track these at baseline and at regular intervals.

Marker	What It Tells You	Frequency / Target
MoCA (Montreal Cognitive Assessment)	Validated screening for MCI; tests memory, attention, language, visuospatial	Baseline + every 6 months; score 26+ is normal
MMSE (Mini-Mental State Exam)	Broad cognitive screening; widely used in clinical settings	Baseline + every 6 months; score 24+ is normal
Homocysteine	Amino acid level reflecting B vitamin status; strong dementia risk factor	Baseline + every 6 months; target <10 mcmol/L
Serum 25(OH)D	Vitamin D status	Baseline + every 6 months; target 40-60 ng/mL
hs-CRP	Systemic inflammation marker	Baseline + every 3-6 months; target <1.0 mg/L
Omega-3 Index	EPA+DHA as % of red blood cell membrane fatty acids	Baseline + annually; target 8-12%
Subjective cognitive tracking	Daily memory, focus, word-finding, mental clarity self-rating (1-10 scale)	Weekly journal; look for trends over months
Sleep quality	Duration, continuity, perceived restfulness	Nightly tracking; 7-8 hours with minimal waking

Timeline Expectations

Weeks 1-4: Digestive improvements; subtle energy and mood changes as microbiome begins shifting. Months 2-3: Improved mental clarity and focus; homocysteine may begin to decrease. Months 3-6: Measurable cognitive improvements on formal testing (based on 12+ week RCT data). Months 6-12+: Sustained neuroprotective benefit; reduced inflammatory markers; long-term risk reduction. Be patient. Brain health responds to consistent, sustained intervention over months, not days.

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Disclaimer

This protocol is provided for educational and informational purposes only. It is not intended as medical advice and should not replace consultation with a qualified healthcare provider. Cognitive decline and dementia are complex neurological conditions that may require prescription medications and specialist management. Always consult your neurologist, geriatrician, or primary care physician before starting any supplement regimen, especially if you are currently taking medications for Alzheimer's disease, cardiovascular conditions, or blood-thinning therapies. Individual results may vary. The information presented is based on peer-reviewed research available at the time of writing and may be updated as new evidence emerges.

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