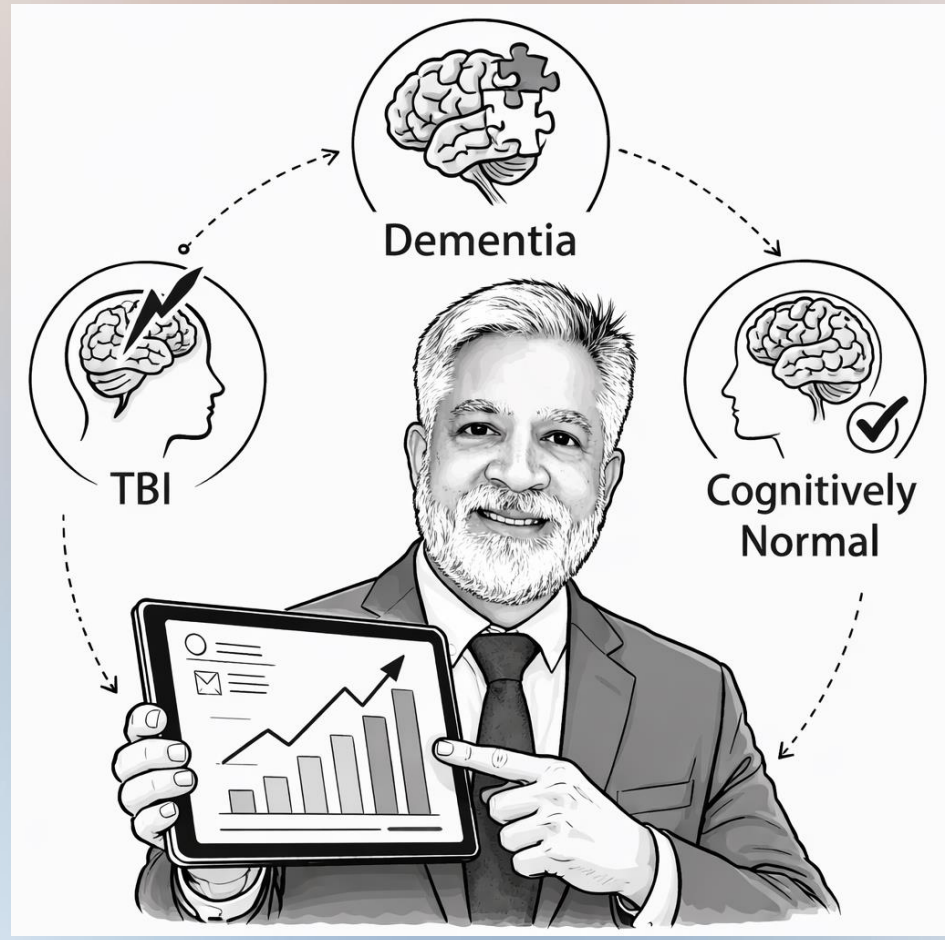

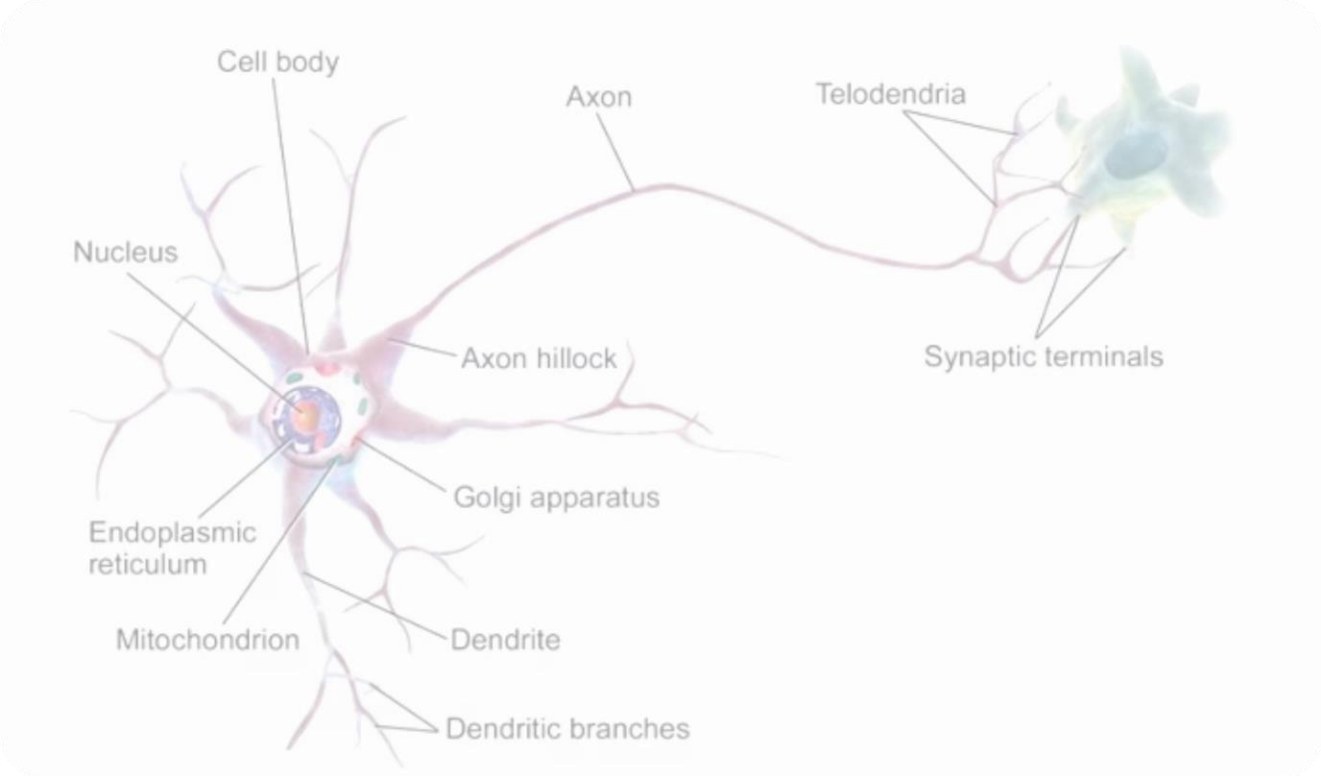

Optimizing Nutritional Support To the Military service members

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Headache, TBI & Cognitive Research Institute · Shreveport

Disclosure Statement

Principal Investigator

Dr. Suresh Kumar Practicing Neurologist, researcher and developer of RegainMemory 360 and serves as Principal Investigator at the Headache, TBI & Cognitive Research Institute.

Institutional Roles

President, Neurology & Headache Center, Inc. · CEO, Regain Memory. Forty percent of clinic & Regain Memory LLC revenue is reinvested into clinical research and development.

Intellectual Property

regainmemory360.com

Headache, TBI & Cognitive Research Institute

A clinical practice–based research institute dedicated to advancing the understanding and treatment of traumatic brain injury and cognitive impairment through rigorous, IRB-supervised investigation.

Clinical Research Mission

Create awareness through evidence-based research on TBI and cognitive science, bridging laboratory findings with real-world patient outcomes.

Research Focus

Post-traumatic headaches, cognitive impairment following acquired brain injury, and dementia — with emphasis on nutritional and lifestyle interventions.

Quality Assurance

An Internal Review Board maintains the standard of all research activities, ensuring ethical rigor and methodological consistency across studies.

Our Journey Of 20 yrs Cognitive Recovery….

I have dedicated more than 20 years of relentless research and clinical practice to improving the diagnosis and treatment of memory impairment, while advancing the understanding of memory disorders, cognitive decline, traumatic brain injury, and brain recovery.

1

AI –Self Cognitive Testing

AI-powered, self-administered online 12 domains cognitive assessment test helps identify patterns and guides personalized improvement.

2

Nutritional & Sleep Optimization

Guide and works with your **primary care physician**
To identify Neurometabolite deficiency
Identify possible underlying or secondary contributors of memory loss

3

Personalized Cognitive Therapy

A personalized online brain training program.
Targets weaker cognitive domains
Visual progress charts to track changes over time
Structured exercises designed around principles of **neuroplasticity**

Important Disclaimer
SDMAC/RM360 supports cognitive screening and monitoring and does not replace professional medical diagnosis or treatment. **Individual results may vary.**

Is Memory Loss & Recovery is Nutritional Dependent

Many patients' experiencing forgetfulness, brain fog, or poor concentration may be lacking essential nutrients the brain depends on every day. Before assuming cognitive decline is irreversible, clinicians should evaluate modifiable nutritional contributors.

Vitamin B12

Critical for nerve health and myelin integrity. Deficiency — especially common in vegetarians — can produce confusion and memory symptoms that mimic dementia.

Folate (B9)

Essential for DNA repair and neurotransmitter synthesis. Deficiency contributes to cognitive decline, low mood, and reduced mental clarity.

Vitamin D

Supports brain health, mood regulation, and immune function. Low levels are linked to memory impairment and increased Alzheimer's risk.

Mg, Zn & Fe

Trace elements vital for synaptic communication. Even mild deficiencies cause fatigue, poor concentration, and slowed cognitive processing.

Vitamin D, Homocysteine & Cognitive Recovery

A clinical study of 373 patients evaluated for memory concerns over five years examined the relationship between key nutritional biomarkers and cognitive performance as measured by the Montreal Cognitive Assessment (MoCA).

Methods

Patients stratified by MoCA score: mild (25–17), moderate (16–10), or severe (<10) impairment. Serum vitamin D, B12, folate, and homocysteine levels obtained as part of a comprehensive memory panel. Regression analyses assessed associations with MoCA scores.

Key Findings

- Each 1 μM increase in homocysteine → 0.22-point decrease in MoCA ($t = -3.81$, $p < 0.001$)
- Higher vitamin D inversely associated with homocysteine ($t = -2.40$, $p = 0.017$)
- Each 10 ng/mL increase in vitamin D → ~0.8-point higher MoCA score ($\beta = 0.083$, $p = 0.042$)
- Cognitive impairment persisted even at vitamin D levels of 40 ng/mL, underscoring the role of co-factors

Conclusion: Vitamin D and homocysteine interact through methylation pathways linked to beta-amyloid regulation — vitamin D is a potentially modifiable factor in cognitive health.

Folic Acid, Homocysteine & Cognitive Risk

The Homocysteine–Dementia Link

Folic acid and vitamin B12 are cofactors in homocysteine metabolism. A **5 µmol/L increase** in plasma homocysteine increases AD risk by **40%** — even after adjusting for age, sex, ApoE genotype, and B-vitamin levels. Elevated homocysteine and reduced folate are independent predictors of both dementia and Alzheimer's disease.

Folic acid deficiency may also reduce acetylcholine concentrations — the primary deficient neurotransmitter in AD — directly impairing cholinergic neurotransmission.

Cognitive Domains Affected

Elias et al. verified a significant inverse association between homocysteine levels and multiple cognitive abilities in individuals ≥60 years, including:

- Abstract reasoning and executive performance
- New verbal learning and memory
- Visual memory and organization
- Concentration, scanning, and tracking
- Object naming and language

Intervention Evidence

Smith et al.: supplementation with folic acid (0.8 mg/day), B12 (0.5 mg/day), and B6 (20 mg/day) for 2 years produced a **significant reduction in brain atrophy rate** in elderly with mild cognitive impairment.

Vitamin D: The Neuroactive Hormone

Prevalence of Deficiency

Approximately **1 billion people** — ~15% of the elderly population — are classified as deficient (<20 ng/mL) or insufficient (<30 ng/mL) in 25-hydroxyvitamin D.

Brain-Specific Actions

Vitamin D receptors and 1-α-hydroxylase are expressed throughout the brain — in neurons, glial cells, macrophages, spinal cord, and peripheral nervous system — confirming its role as a **neuroactive hormone** with autocrine and paracrine functions.

Neuroprotective Mechanisms

- Reduces calcium toxicity and nitric oxide synthesis
- Modulates glutathione metabolism and cytokine release
- Stimulates neurotrophins and neuritogenesis
- Promotes Aβ clearance and phagocytosis by macrophages

Annweiler et al

Serum <10 ng/mL associated with worse cognitive decline in French women ≥75 years.

Slinin et al

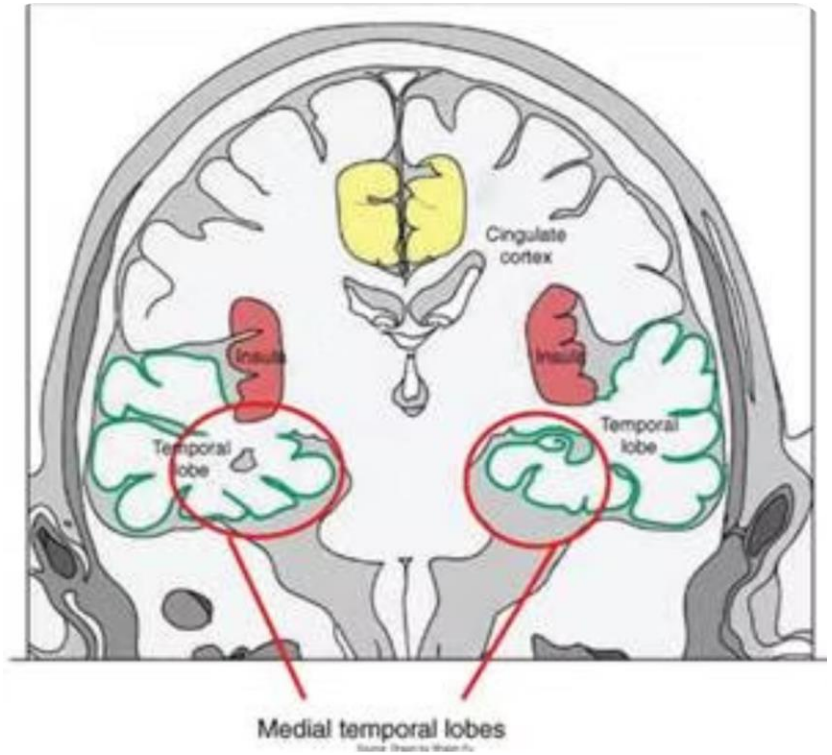
Low serum D (<20 ng/mL) linked to cognitive decline in 1,600+ North American men ≥65 years over 4.6 years.

Llewellyn et al

Serum <10 ng/mL yielded a **relative risk of 1.61** for cognitive decline vs. ≥30 ng/mL over 6 years.

Dosing: Vitamin D3 (cholecalciferol) preferred. Recommended range for elderly with AD: **800–4,000 IU/day**.
Supraphysiological doses do not improve cognitive outcomes.

Nutrition of Neurons



Mesial Temporal Cortex & Dementia

The mesial temporal cortex — critical for memory formation and eating behavior regulation — is directly affected by dementia. As this region degrades, patients experience appetite loss, refusal to eat, and declining food intake, creating a vicious cycle of worsening nutritional status.

Micronutrients as Neuroprotective Agents

The mesial temporal cortex cannot function optimally without adequate balance of trace elements, vitamins, and electrolytes. Micronutrients enhance and sustain neural plasticity while attenuating ongoing neurodegenerative processes — making nutritional optimization a core therapeutic strategy, not merely supportive care.

Folic Acid & Homocysteine

The Homocysteine–Dementia Link

Folic acid and vitamin B12 are cofactors in homocysteine metabolism. Elevated homocysteine is a **strong independent predictor** of dementia and AD — each 5 µmol/L increase in plasma homocysteine raises AD risk by **40%**, independent of age, sex, ApoE genotype, and B-vitamin status.

Cognitive Impact

Elias et al demonstrated a significant inverse association between homocysteine and multiple cognitive domains — abstract reasoning, verbal and visual memory, executive performance, and language — in adults ≥60 years.

Smith et al: Supplementation with folic acid (0.8 mg/day), B12 (0.5 mg/day), and B6 (20 mg/day) for 2 years significantly reduced the rate of brain atrophy in elderly patients with mild cognitive impairment.

A Hidden Epidemic: Nutritional Risk in Asian Indians

As a neurologist, I frequently identify four major reversible contributors to cognitive decline in Asian Indian patients — both in India and abroad. These are often overlooked, yet highly treatable.

Vitamin B12 Deficiency

Predominantly vegetarian diets lack sufficient B12 — a nutrient found mainly in animal-based foods and essential for nerve and brain function.

Vitamin D Deficiency

Despite sunny climates, indoor occupations, cultural clothing, and darker skin pigmentation significantly reduce vitamin D synthesis.

MTHFR Genetic Variation

Relatively common in South Asian populations; impairs folate processing and raises homocysteine, increasing vascular and cognitive risk.

Undiagnosed Sleep Apnea

Many patients snore or feel exhausted after 7–8 hours of sleep. Repeated nocturnal oxygen drops cause brain fog, memory loss, and long-term vascular injury.

The good news: these causes are treatable and often reversible. Test, don't guess — check B12, vitamin D, folate, homocysteine, and consider a sleep study.

"Doctor, What Should I Eat to Help My Memory?"

Oxidative stress — driven by transition metals such as Fe, Cu, and Zn — catalyzes reactive oxygen species (ROS) formation. Amyloid-β protein further inhibits the mitochondrial electron transport chain, amplifying ROS production and neurotoxicity. Targeted dietary antioxidants are a rational first-line defense.

Antioxidant Enzymes

Catalase and glutathione peroxidase are endogenous defenses against cytotoxic ROS; supported by dietary selenium and vitamin E.

Vitamins C & E

Free-radical scavengers that act synergistically to neutralize ROS and protect neuronal membranes from oxidative damage.

Omega-3 Fatty Acids

DHA and EPA modulate membrane fluidity, synaptic plasticity, and anti-inflammatory prostaglandin production — essential for neuronal health.

Trace Minerals

Zinc, iron, copper, and selenium maintain antioxidant defenses and regulate gene expression critical for neuronal integrity.

Selenium: A Neuroprotective Trace Element

Selenium (Se) exerts its neuroprotective effects primarily through selenoprotein P (SePP), glutathione peroxidase (GPx), and thioredoxin reductase. These selenoproteins constitute a powerful antioxidant system that defends neurons against amyloid-driven oxidative damage.

Selenium & Cognitive Function

Smorgon et al. found a direct correlation between plasma Se and cognitive performance. AD patients showed significantly reduced plasma Se concentrations vs. healthy controls (Cardoso et al.; Vural et al.).

SePP Neuroprotection

Takemoto et al. showed neuronal cells exposed to amyloid plaque oxidants were protected in the presence of SePP. Lu et al. and Miller et al. note SePP levels trend upward in aging and AD.

GPx Activity Deficit in AD

Cardoso, Vural, and Parudariu et al. each verified that GPx activity is significantly lower in AD patients and those with mild cognitive impairment compared to healthy elderly individuals.

Dietary source: Just two Brazil nuts daily for 12 weeks significantly improved Se status in healthy individuals (Thomson et al.). Se content in Brazil nuts ranges from 8–83 µg/g and is highly bioavailable.

Vitamins C & E: Synergistic Antioxidant Protection

Mechanism of Synergy

Vitamin E (tocopherols and tocotrienols) is the primary membrane-bound fat-soluble antioxidant. Oxidized α-tocopherol is regenerated by ascorbate (vitamin C), enabling continuous protection of low-density lipoproteins from oxidative damage — creating a self-renewing antioxidant cycle.

Neuroprotective Evidence

In vitro studies demonstrate that vitamins C and E may prevent hyper-phosphorylated tau protein dysfunction and reduce Aβ-induced neuronal death in hippocampal and cortical cell cultures. The Cache County Study found the **combined** use of vitamins C and E was associated with reduced AD occurrence — isolated intake of either vitamin alone showed no significant benefit.

Critical Clinical Note

OTC vitamin E supplements typically contain only synthetic α-tocopherol — one of eight natural isoforms. Neuroprotection appears to require the full combination of tocopherol and tocotrienol forms found in whole foods: wheat germ oil, olive oil, almonds, hazelnuts, sunflower seeds, dark leafy greens, avocado, and fish.

Zinc: Essential Mineral for Cognitive Integrity

Zinc is among the most important minerals for human metabolism, playing indispensable roles in carbohydrate, protein, lipid, and nucleic acid processing. In the brain, adequate zinc levels are foundational to neuronal health and cognitive function.

Gene Expression & Transcription

Zinc contributes to polynucleotide transcription and control of gene expression — fundamental mechanisms for neuronal development and repair.

Membrane Integrity & Antioxidant Defense

Zinc maintains membrane structural integrity, supports antioxidant defenses, regulates immunity, and is essential for normal growth and appetite maintenance.

Dietary Sources

Seafood (especially oysters), red meat, nuts, seeds, and whole grains are the richest dietary sources of bioavailable zinc.

B Vitamins: Niacin, Thiamin & B12

Niacin, thiamine, and vitamin B12 have well-established relationships with mental status deterioration. Each deficiency syndrome produces distinct — and often severe — neurological and cognitive consequences that are frequently misattributed to primary dementia.

Wernicke–Korsakoff Syndrome

Thiamine (B1) deficiency. Damages central and peripheral nervous systems, producing ophthalmoplegia, ataxia, and subsequent memory loss, confabulation, and hallucinations — a reversible cause of apparent dementia if recognized early.

Pellagra

Niacin deficiency. Classic triad of dementia, diarrhea, and dermatitis. CNS manifestations include neurasthenia, psychosis, disorientation, memory loss, and confusion — all potentially reversible with niacin repletion.

B12-Associated Dementia

Vitamin B12 deficiency. Impairs myelin synthesis and DNA methylation, producing subacute combined degeneration, megaloblastic anemia, and cognitive decline — especially prevalent in vegetarians and the elderly.

Omega-3 Fatty Acids & Neuronal Membrane Health

Omega-3 long-chain polyunsaturated fatty acids — ALA, EPA, and DHA — are incorporated into cell membranes and participate in anti-inflammatory cascades. **DHA** is the most abundant lipid in the neuronal membrane; **EPA** modulates membrane fluidity, synaptic plasticity, and receptor affinity.

1

Anti-Inflammatory Action

EPA is the substrate for anti-inflammatory prostaglandin-3 and competes with arachidonic acid at cyclooxygenase sites, reducing neuroinflammation.

2

Amyloid Modulation

Gu et al.: highest omega-3 intake was associated with **lower Aβ40 and Aβ42 concentrations**, even after adjusting for age, ethnicity, ApoE genotype, and caloric intake.

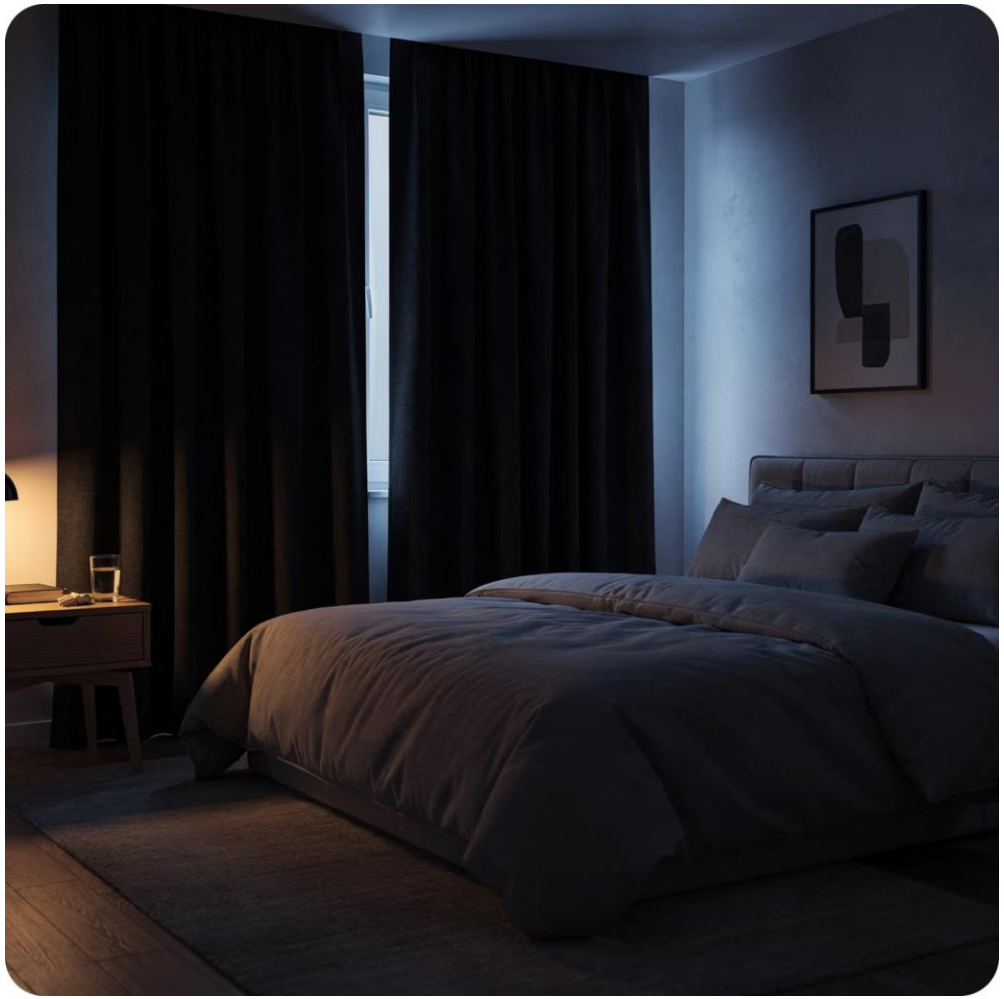
3

Population Evidence

Barberger-Gateau et al. (n=8,085; The Three City Cohort Study): weekly fish consumption associated with reduced AD risk (HR = 0.65; 95% CI 0.43–0.994; p = 0.047).

ALA must be obtained from diet (soy, canola, flaxseed oils, nuts). DHA and EPA are primarily found in fatty fish (salmon, tuna, trout) and fish oils. Balance with omega-6 intake is critical.

Melatonin, Sleep & Memory Consolidation



Melatonin's Neuroprotective Roles

Melatonin (5-methoxy-N-acetyltryptamine), synthesized by the pineal gland, serves as a neuroprotector, antioxidant, anti-inflammatory agent, and free-radical scavenger. It regulates circadian sleep-wake cycles — and its production declines with age, disrupting restorative sleep.

Sleep Deprivation & Memory Pathology

Sleep deprivation decreases hippocampal neurogenesis, increases amyloid-β generation, and directly causes memory dysfunction. Deep sleep is when the brain consolidates short-term memories into long-term storage — a process that is severely impaired by sleep fragmentation from apnea or melatonin insufficiency.

Clinical Evidence

While no large RCTs confirm direct pro-memory effects of melatonin supplementation, its positive impact on sleep quality indirectly boosts daytime cognitive performance, attention, and executive function. Melatonin also demonstrates neuroprotective effects after TBI and spinal cord injury.

Dopamine Supplement -Driven BDNF Activity: The Biology of Motivation, Memory, and Neuroplasticity

Dopamine-dependent BDNF activity plays a critical role in activity-driven neuroplasticity by linking motivation, reward, and attention with synaptic strengthening, memory formation, and brain recovery.

L Tyrosine

Direct amino-acid precursor for dopamine, norepinephrine, and epinephrine; may help cognitive performance under stress, though results vary..

L-Phenylalanine

Precursor to tyrosine, which then supports catecholamine synthesis. Avoid in **PKU**

L-Glutamine

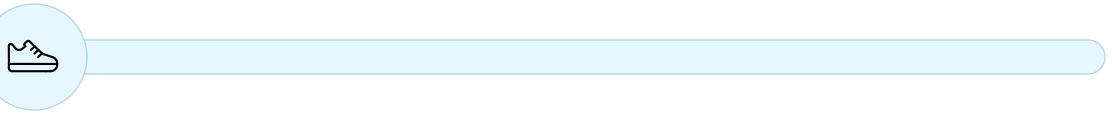
Related to the glutamate/GABA–glutamine cycle than direct dopamine production; may support brain metabolism and neurotransmitter balance, but it is **not a direct dopamine precursor**..

Exercise + cognitive training

Often stronger than supplements for increasing BDNF activity and neuroplasticity..

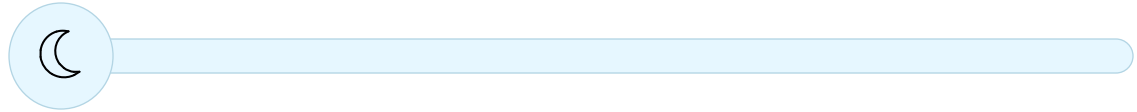
BDNF: The Brain's Growth Hormone

Brain-Derived Neurotrophic Factor (BDNF) acts as a fertilizer for the brain — supporting neuronal survival, stimulating new neuron formation, and strengthening synaptic connections. As BDNF levels decline with age (accelerated by poor sleep, stress, and inactivity), learning and memory suffer. Encouragingly, BDNF is modifiable.



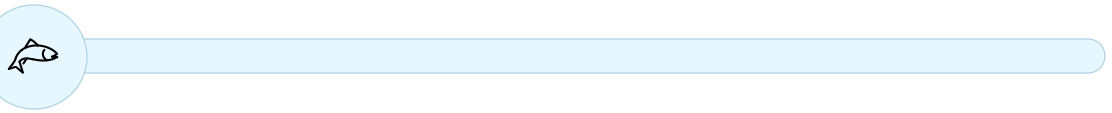
Physical Activity

30 min/day of aerobic exercise (walking, jogging, cycling) is one of the strongest natural BDNF stimulators.



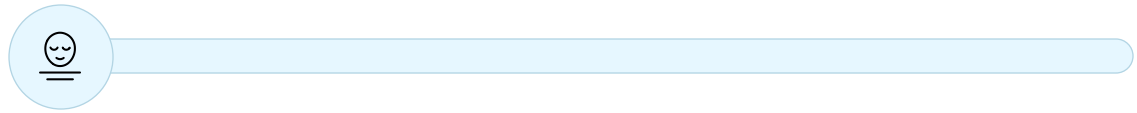
Deep Restful Sleep

7–9 hours of quality sleep strengthens synapses and sustains BDNF activity. Avoid screens 1 hour before bed.



Brain-Supporting Nutrition

Omega-3s, berries, nuts, and magnesium-rich foods support BDNF. Intermittent fasting may also stimulate production.



Stress Management & Medical Treatment

Chronic stress raises cortisol and suppresses BDNF. Treat sleep apnea, depression, insulin resistance, and vitamin deficiencies promptly.

Ginkgo Biloba: Evidence & Limits

Ginkgo biloba extract (EGb) contains flavonoids and terpenoids (ginkgolides, bilobalide) with varying degrees of antioxidant activity. It is one of the most widely studied herbal interventions for cognitive decline.

JAMA Clinical Trial

309 patients with dementia randomized; 78 in the EGb group and 59 in placebo completed the full 1-year study. Dosage: 120 mg daily. The Ginkgo group showed **no cognitive deterioration** compared to placebo — but no measurable improvement was reported either.

Clinical Interpretation

The evidence supports a **stabilizing rather than restorative** effect. Some patients report improved alertness with EGb. Ginkgo may be considered as an adjunct in a comprehensive nutritional protocol — not as monotherapy for cognitive recovery.

Ginkgo biloba's mechanism likely involves antioxidant activity, improved microcirculation, and mild anti-platelet effects — benefits that complement, but do not replace, foundational nutritional correction.

Aspirin & NSAIDs in Alzheimer's Disease

Cochrane Review Evidence

Jaturapatporn et al, *Cochrane Database Syst Rev.* 2012 — 14 RCTs (15 interventions) meeting inclusion criteria were analyzed across three treatment arms:

352	138	1745
Aspirin	**Steroids**	**NSAIDs**
Participants	Participants	Participants

Clinical Conclusion

The efficacy of **aspirin, corticosteroids, and NSAIDs** (both traditional NSAIDs and COX-2 inhibitors) in the treatment of Alzheimer's disease is **not proven**.
Despite the biological rationale for anti-inflammatory approaches in AD — given the neuroinflammatory processes implicated in amyloid pathology — current RCT evidence does not support routine use of these agents for cognitive preservation or treatment.

⚠ Clinicians should weigh the well-documented risks (GI bleeding, cardiovascular events, renal toxicity) against the **absence of demonstrated cognitive benefit**.

Key Takeaways: Nutritional Therapy in Memory Loss

Micronutrients Matter

Selenium, vitamins C, D, E, B-complex, zinc, and omega-3s each address distinct mechanisms in neurodegeneration.

Food First

Dietary sources consistently outperform isolated supplements — especially for vitamin E and omega-3 fatty acids.

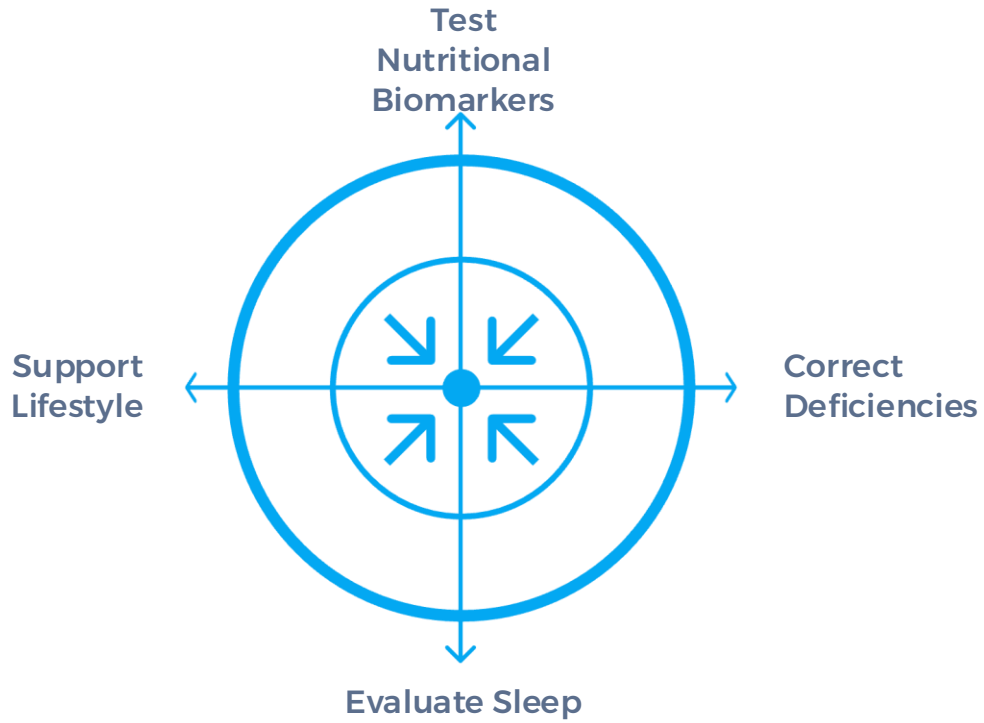
Evidence Is Evolving

Botanicals like Ginkgo, Ginseng, and Huperzine A show promise, but large-scale RCTs are still needed.

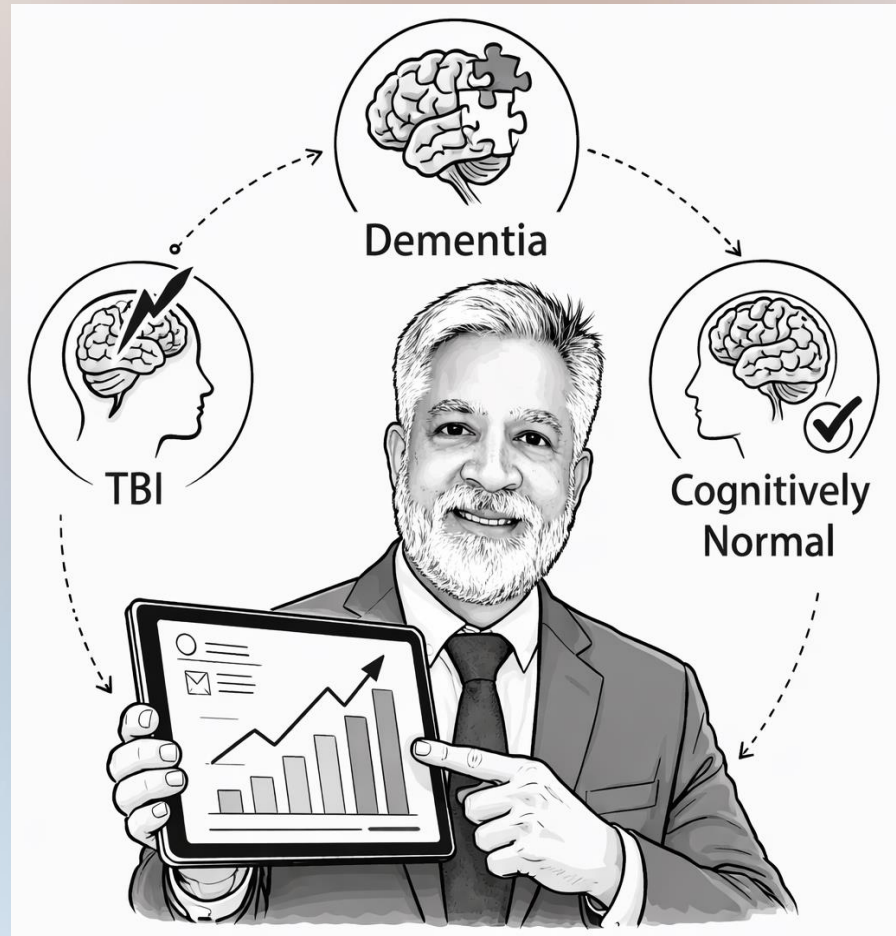
Sleep & Lifestyle

Melatonin and sleep quality are integral to cognitive resilience — restorative sleep amplifies the benefits of nutritional interventions.

Restoring the Brain: Key Clinical Takeaways



Before labeling a patient's symptoms as irreversible aging or primary dementia, a systematic evaluation of modifiable nutritional and lifestyle contributors is both clinically warranted and ethically imperative. Correcting these deficiencies can produce measurable cognitive gains.



Our Study support for Brain Body Optimization- Restore the Brain. Live Fully.

> Your brain deserves the right fuel. Correct the deficiency — restore the function — and reclaim your mental clarity.

373

Patients Studied

Over a five-year clinical period at the neurology clinic

40%

AD Risk Rise

For every 5 µmol/L increase in plasma homocysteine

~0.8

MoCA Points Gained

For each 10 ng/mL increase in vitamin D (age-adjusted)

4

Reversible Causes

B12, vitamin D, MTHFR variants, and sleep apnea — all treatable

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