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THE HUMAN BRAIN <https://medicalbits.transistor.fm/episodes/brain-health>

How to Build, Protect, and Preserve the Most Complex Object in Our Universe

Three pounds: that is approximately what the adult human brain weighs. Within those three pounds reside every face you recognize, every language you speak, every melody you remember, every fear, every joke, the many injustices you have witnessed, every hope you harbor, and the continuously edited story you call “myself.” Our brain is not simply another organ. It is the only organ capable of contemplating itself (although our new AI agents are becoming increasingly talented mimics!).

Our Earth and Space-based telescopes have observed incredible wonders, and our precise microscopes have discovered myriad biological complexities, but not an object more intricate than our brain. No computer has reproduced its full adaptability. Its roughly 90 billion neurons form trillions of connections. The remarkable part is not their number, but their dynamism: strengthened by use, weakened by neglect, remodeled after injury, and influenced by sleep, movement, blood pressure, glucose, hearing, vision, mood, education, observation, attention, friendship, and **purpose**.

BRAIN HEALTH IS BUILT ACROSS THE LIFE COURSE



“Every man can, if he so desires, become the sculptor of his own brain.”

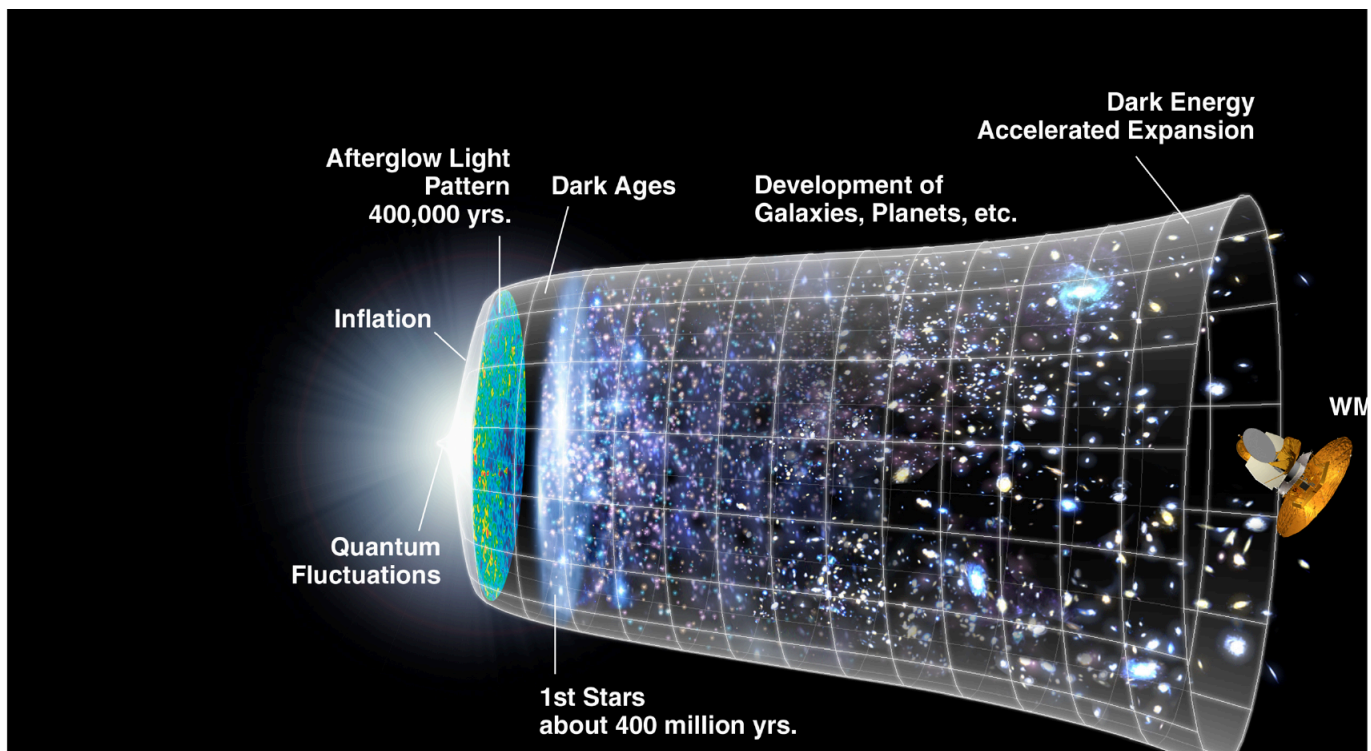
Santiago Ramón y Cajal

A BRIEF COSMOLOGICAL AND EVOLUTIONARY DETOUR

From a Universe of Energy to the Birth of Stars and Planetary systems.

On Feb 12, 2003, as I was enjoying the daily pleasures of [APOD \(Astronomy Picture of the Day\)](#), I was jolted into action by a simple picture and its description: WMAP resolves the Universe! I was beyond myself. Where had I been all those years!? How could it be that the Universe was resolved, and I had no idea!

Besides the [universe age](#), [data](#) and analyses of the [spots](#) on the [cosmic microwave background](#) bolstered indications that the universe is composed of a [strange](#) type of [dark energy](#) (73%), a mysterious type of [dark matter](#) 23%, and regular matter (baryonic) that we know and touch (4%) in familiar [atoms](#). A useful way to visualize this almost incomprehensible span of time is the [WMAP/NASA diagram](#). It compresses the history of the Universe into a single image: inflation at the extreme left, the afterglow of the cosmic microwave background, the dark ages, the first stars, the development of galaxies and planets, and the later accelerated expansion associated with dark energy.



[Inflating the Universe — WMAP Science Team, NASA. Astronomy Picture of the Day, March 23, 2006. The original WMAP-era diagram labels a 13.7-billion-year history; today the age of the Universe is generally rounded to about 13.8 billion years.](#)

The remarkable part of the figure is not simply its scale. That rapid early expansion called inflation is thought to have magnified tiny quantum fluctuations into the seeds of the large-scale structure we recognize as galaxies and clusters. Roughly 400,000 years after the Big Bang, the Universe cooled enough for light to travel freely; that fossil light is the cosmic microwave background measured with extraordinary precision by WMAP. Hundreds of millions of years later, the first stars appeared. Those stars and their descendants manufactured the heavier elements from which planets—and eventually biology—could be built.

The story of the brain begins long before brains existed.

Amazingly, our Universe is mostly energy with tiny amounts of matter. All the chemical elements of that matter, without exception, formed in the furnace of stars from humble atoms of hydrogen (H) and helium (He).

The solar system and our rocky planet Earth appeared roughly 4.5 billion years ago. For its first two billion years, Earth's atmosphere was anoxic (no O₂), and simple, unicellular life was dominated by anaerobic metabolism (fermentation and methanogenesis). The evolution of photosynthetic cyanobacteria in our primitive oceans—between 3.8 and 2.7 billion years ago—enabled the biological production of O₂ (initially remaining at low concentrations, due to rapid consumption and buffering by reduced minerals and gases), but 2.4 billion years ago, the Great Oxygenation Event marked the first substantial rise in atmospheric oxygen. Oxygen levels fluctuated for hundreds of millions of years before stabilizing above 10% of present atmospheric levels, with permanent oxygenation arriving around 2.2 billion years ago. A second major oxygenation event occurred in the Neoproterozoic (~800 million years ago), coinciding with the rise of complex multicellular life.

The cosmological timeline from the creation of our Universe to the rise of Oxygen in our atmosphere spans almost 12 billion years, with numerous biological and geochemical milestones promoting the evolution of oxygen-utilizing metabolic pathways along the way, resulting in complex biological forms. As oxygen became available, metabolic pathways evolved, and aerobic respiration emerged in response, making organisms much more efficient and capable of delivering a higher energy yield per molecule of substrate. Photosynthetic cyanobacteria eventually learned to split water using sunlight and released oxygen as a waste product. Oxygen was initially toxic, but surviving organisms evolved antioxidants, protective enzymes, and membranes resistant to oxidation.

The great biochemical prize was energy. Without oxygen, one molecule of glucose yields about two ATP molecules; aerobic metabolism yields roughly thirty-two.

Only life can produce oxygen in significant concentrations!

This enormous increase in efficiency helped make large, specialized, multicellular life possible. Mitochondria inside our cells are descendants of ancient bacteria, and the human brain—only about two percent of body weight—uses roughly one-fifth of the body's energy at rest.

Life existed for billions of years before anything resembling a brain appeared. Single-celled organisms sensed light, chemicals, and temperature without neurons. Later, specialized excitable cells evolved to transmit signals rapidly. Simple nerve nets allowed early animals to coordinate movement. As bodies became larger and more complex, sensory information began to converge toward a front end: cephalization, the evolutionary habit of putting the command center near the eyes, ears, mouth, and direction of travel.

Brains were not designed in one elegant moment. Evolution does not begin with a blueprint; it modifies whatever is already available. Newer systems were layered upon older ones. Basic circuits regulate breathing, temperature, hunger, arousal, and movement. What appears impossible in human timescales becomes inevitable in cosmic ones.

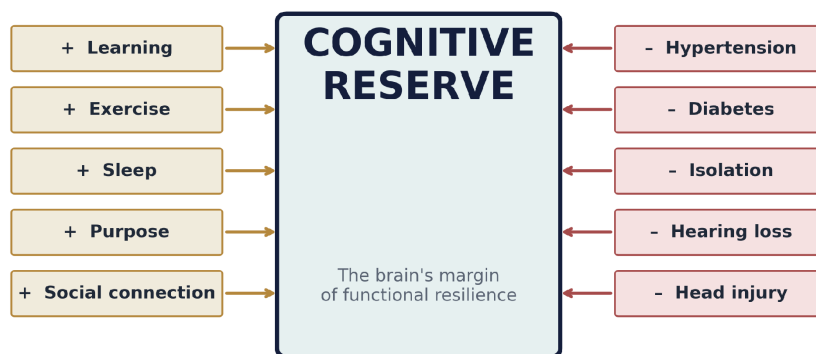
Every breath connects us to billions of years of biological innovation.

THE BRAIN CHANGES EVERY DAY

Plasticity, pruning, memory, forgetting, and cognitive reserve

The old metaphor of the adult brain as fixed wiring has been discarded. The brain is better understood as a living network that continually updates itself. Every meaningful experience changes its activity, and repeated experiences strengthen synapses.

One of the most intriguing observations in neurology is that pathology and symptoms do not always travel together. Two individuals may have comparable amyloid deposition, vascular injury, or age-related atrophy, yet one remains independent while the other struggles with daily tasks. Some brains appear able to tolerate more damage before function becomes visibly impaired. Reserve is not a hidden reservoir of extra neurons. It is a property of networks—the efficiency, flexibility, and number of alternative strategies available when the usual route is compromised. Education is associated with reserve, but years of schooling are an imperfect proxy. Occupational complexity, literacy, languages, music, social engagement, physical activity, and lifelong learning may all contribute. None provides immunity; they may delay the point at which pathology becomes clinically obvious.



[The Nun Study](#) helped popularize this concept. Some participants had extensive Alzheimer-type pathology at autopsy despite relatively preserved cognition during life. Their stories did not prove that education prevents Alzheimer disease, but they showed that the clinical expression of disease

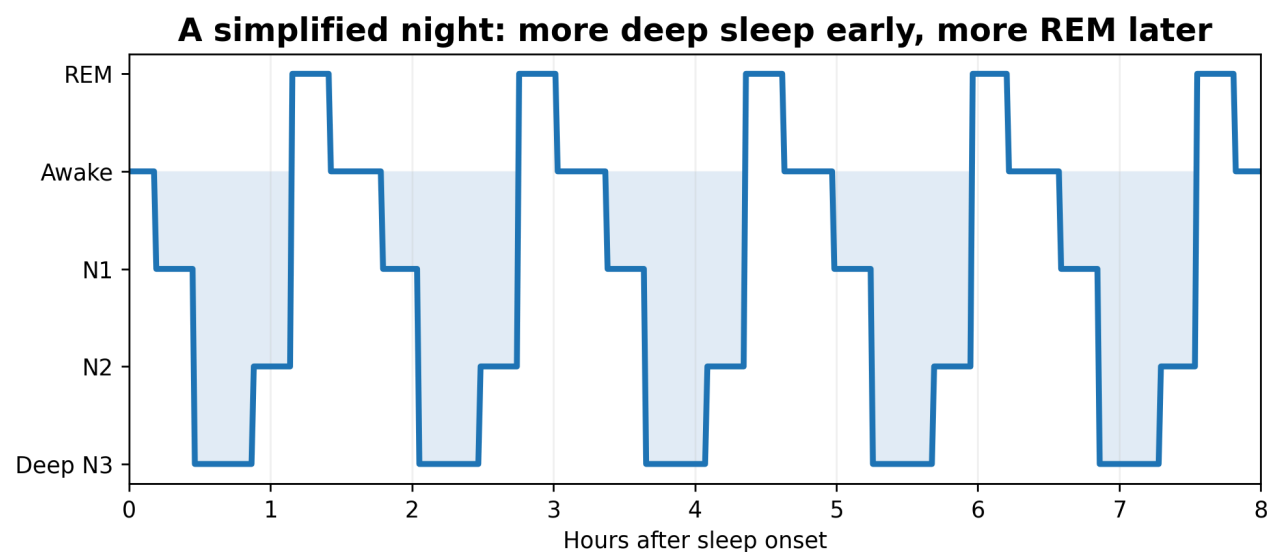
depends on more than plaques alone. The lesson is both hopeful and sobering: reserve may postpone disability, but once compensatory capacity is exhausted, decline can appear relatively quickly. Reserve is built through a varied life: reading outside one's field, sustained conversation, travel, teaching, learning an instrument, dancing, solving unfamiliar problems, and maintaining physical and sensory function. The best cognitive activity is one that is meaningful enough to continue and demanding enough to require adaptation. Make regular deposits in the cognitive reserve account. But no account is infinite. Learn something that makes you temporarily incompetent. That discomfort is evidence that the network is adapting.

SLEEP, HEARING, AND VISION

The brain's night shift: Memory consolidation and metabolic housekeeping.

We spend roughly one third of life asleep, an enormous evolutionary investment for an organism that becomes less vigilant and more vulnerable while doing it. Sleep must therefore perform functions important enough to justify the risk. It supports memory consolidation, emotional regulation, immune balance, metabolic control, and the recalibration of neural networks. Deep

non-REM sleep is prominent earlier in the night, while REM sleep becomes more abundant toward morning. Both matter!



Sleep deprivation alters amyloid and tau biology, while chronic sleep disruption is associated with poorer cognition and higher dementia risk. The most clinically important disruptor is often obstructive sleep apnea as repetitive airway collapse produces intermittent hypoxemia, sympathetic surges, blood-pressure variability, fragmented sleep, daytime impairment, with morning headache, resistant hypertension and even atrial fibrillation.

Treating apnea improves sleepiness and quality of life. Sleep medication requires the same caution as every other medical intervention. Sedating antihistamines and drugs with anticholinergic effects can worsen confusion, balance, and memory, particularly in older adults. Alcohol may shorten sleep latency but fragments the second half of the night.

Remember that cognition depends on input and therefore, any sensory loss is unhealthy. The brain does not experience the world directly. It constructs an internal model from sensory signals. When hearing declines, conversation requires more effort, subtle information is missed, and participation becomes tiring. The person may withdraw from restaurants, meetings, lectures, and family exchanges—not because interest has disappeared, but because decoding speech has become exhausting. Hearing loss is consistently associated with cognitive decline, but association does not prove that hearing loss directly causes dementia.

Sleep is not time “wasted”. It is part of the machinery that allows waking life to work.

EXERCISE: THE MOST RELIABLE BRAIN MEDICINE

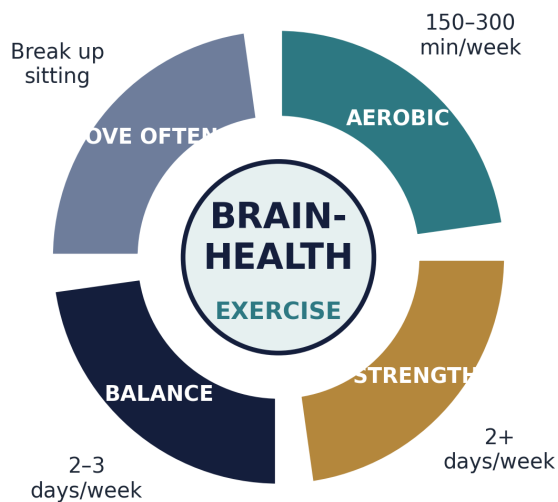
Movement changes vessels, metabolism, mood, muscle, and the brain itself

If a drug improved blood pressure, insulin sensitivity, sleep, mood, balance, muscle mass, bone density, cerebral perfusion, and cardiovascular risk, it would be considered extraordinary. Physical activity does all of these, but it’s impossible to patent! And requires your participation!

Remember that EXERCISE is not a single intervention.

Aerobic activity, resistance training, balance work, coordination, and interruption of prolonged sitting affect different systems. Aerobic exercise improves cardiorespiratory fitness and endothelial function.

Resistance exercise preserves muscle, glucose disposal, bone, and the ability to recover from illness. Walking is excellent, but walking alone may not preserve strength. Strength work is necessary, but strength alone may not preserve aerobic capacity. The brain benefits from all forms of exercise and from learning new tricks.



Mechanistic studies describe increased brain-derived neurotrophic factor, vascular adaptation, altered inflammation, and hippocampal plasticity. Clinical studies show that physically active adults tend to experience better cognitive outcomes, although healthier people may also be more able to exercise—a reminder that observational data can exaggerate causation.

Randomized trials support modest cognitive benefit, particularly from

sustained and multidomain programs, but there is no single perfect “brain workout.” A reasonable prescription is at least 150 minutes per week of moderate aerobic activity, two or more sessions of resistance training, and regular balance practice, adjusted for ability and medical status. The most effective program is the one continued long enough to matter.

Progress gradually, avoid injury, and become appropriately uncomfortable!

Muscle is not ornamental tissue. It is a metabolic organ and the brain’s most useful ally.

FOOD FOR THOUGHT—AND THE VASCULAR BRAIN

Dietary patterns, blood pressure, cholesterol, smoking, and small vessels

Dietary patterns matter more than superfoods, powders, and promises. Nutrition research is difficult. People do not eat isolated nutrients; they eat patterns of food embedded in culture, income, appetite, habits, and family. Dietary questionnaires are imperfect, adherence changes, and healthy eaters often exercise more and smoke less. These limitations explain why nutrition headlines repeatedly swing between enthusiasm and reversal.

Emphasize vegetables, legumes, fruit, nuts, whole grains, fish, and unsaturated fats; minimize tobacco, excess calories, refined carbohydrates, sugary drinks, and highly processed foods.

[Mediterranean and MIND-style](#) diets are associated with better cardiovascular and cognitive outcomes. The evidence is stronger for the overall pattern than for any “brain food.” Blueberries are nutritious; they are not a treatment. Olive oil is useful; it does not neutralize an otherwise

unhealthy diet. The MIND diet combines features of Mediterranean and DASH eating, with particular emphasis on green leafy vegetables, berries, nuts, beans, whole grains, and fish.



The [FINGER study](#) combined nutrition with exercise, cognitive training, social activity, and vascular risk management (the way risk factors occur in real life!) with more encouraging results.

As we always repeat, supplements deserve restraint! Correct vitamin B12 deficiency. Treat iron deficiency. Improve blood pressure control and cholesterol. Avoid smoking. Aiming to preserve the health of those invisible small brain vessels.

The brain receives about 20% of cardiac output and depends on an uninterrupted supply of oxygen and

glucose. Large strokes are obvious; small-vessel injury is quieter. Repeated microscopic infarcts, vessel-wall disease, impaired blood pressure autoregulation, and white-matter damage can gradually slow processing, impair gait, and reduce executive function. Many older brains contain mixed pathology - vascular injury alongside amyloid, tau, Lewy bodies and/or other degenerative changes.

Midlife hypertension is one of the strongest modifiable risk factors for later cognitive impairment. The danger is cumulative and often asymptomatic. The [SPRINT-MIND](#) study found that intensive blood-pressure management helps maintain cognitive function.

Eat food recognizable by your grandmother.

DIABETES, OBESITY, AND ALCOHOL

Metabolic stability matters more than short-term “biohacks”

Type 2 diabetes increases the risk of stroke, small-vessel disease, neuropathy, kidney disease, depression, and cognitive decline. High glucose, insulin resistance, and hypoglycemia can also injure cognition, and more so in older adults treated aggressively. The goal is not simply a lower number. It is durable metabolic control without recurrent lows, frailty, or muscle loss. Visceral and liver fat, cardiorespiratory fitness, muscle mass, sleep apnea, and glucose regulation may matter more than body-mass index alone. Weight loss improves blood pressure, diabetes, mobility, and sleep apnea, but rapid loss without resistance training and adequate protein can reduce lean muscle mass. Becoming smaller but weaker is not necessarily healthier.

GLP-1 receptor agonists have transformed the treatment of diabetes and obesity and have been shown to reduce cardiovascular events in selected populations. Observational studies and laboratory work have generated interest in neurologic benefits, including inflammation (“inflammaging”) and neurodegeneration. GLP-1 drugs are already capable of solving diseases

that shorten life, but we do not yet know whether they are true anti-aging drugs. For people with obesity, diabetes or cardiovascular/metabolic risk, the health benefits could plausibly translate into longer, healthier lives. **But taking GLP-1 medications solely for longevity or Alzheimer's prevention in an otherwise healthy person is not currently supported by evidence.** Ongoing trials may clarify whether any cognitive benefit exists beyond improvements in weight, diabetes, sleep apnea, and vascular risk.

The practical prescription is refreshingly conventional: maintain muscle, move after meals, reduce refined carbohydrates and sugary drinks, sleep adequately, treat apnea, and individualize diabetes therapy. The brain does not require a “biohack.” It requires stable physiology over many years. Metabolic health is not a six-week challenge. It is the long conversation between muscle, liver, fat, pancreas, vessels, and brain.

COMMON BELIEF

“A daily drink protects the heart and brain.”

CURRENT MESSAGE

Alcohol can worsen sleep, BP, AFib, falls, cancer risk and cognition at higher doses.

For brain health, less is generally better. Do not start drinking for prevention.

Light drinkers experienced less vascular disease and dementia than abstainers. But abstainer groups may include former heavy drinkers and people who stopped because of illness. Moderate drinkers may differ in income, diet, social connection, and access to care. When analyses address these differences more carefully, the apparent [benefit shrinks](#). We must remember that alcohol is neuroactive and likely neurotoxic, but it's

also a social “lubricant”. Dose matters. Higher intake is associated with hypertension, atrial fibrillation, stroke, sleep fragmentation, neuropathy, falls, depression, several cancers, and structural brain changes. Binge drinking is particularly harmful. Older adults are more sensitive because total body water decreases, medications interact, balance worsens, and the liver clears alcohol less predictably. The safest message for brain health is neither moralistic nor complicated: do not begin drinking for prevention.

If you drink alcoholic beverages, less is better (none is best).

SOCIAL CONNECTION, MOOD, AND MEMORY CHANGES

The brain is a social organ, not an isolated computer

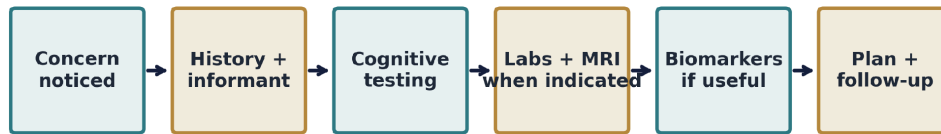
Human brains evolved in groups. Language, facial expression, cooperation, reputation, attachment, and shared memory are not accessories to cognition; they are central functions. Social isolation is associated with depression, inactivity, poorer sleep, vascular risk, and cognitive decline. Yet loneliness is subjective. A person may live alone and feel connected, or live in a crowded household and feel profoundly unseen. Depression can impair concentration, processing speed, motivation, sleep, and memory. In later life, it may imitate dementia, coexist with dementia, or represent an early manifestation of neurodegenerative disease.

[Treating depression](#) is essential for quality of life and may improve cognition, although antidepressants are not established dementia-prevention drugs. Medication choices should consider anticholinergic burden, falls, sodium levels, sleep, and interactions. Purpose is difficult to randomize in a clinical trial but easy to recognize clinically. Normal aging, mild cognitive impairment, delirium, and dementia are not the same. Normal aging may slow retrieval. A name

returns later; multitasking becomes less efficient; new technology requires more repetition. Concerning change is different: repeated questions, missed payments, medication errors,

getting lost in familiar places, impaired judgment, personality change, or loss of previously mastered skills.

EVALUATION WITHOUT PANIC



Treat reversible contributors • assess safety • establish a baseline • avoid premature labeling

The key comparison is not with a twenty-year-old brain but with the person's own prior level of function. Evaluation begins with history, preferably with someone who knows

the patient well. Mood, sleep, hearing, alcohol, medications, pain, recent illness, and daily function matter. Cognitive screening creates a baseline but does not diagnose a specific disease.

A healthy brain needs challenge, but it also needs witnesses—people with whom a life is remembered and shared.

COGNITIVE DYSFUNCTION

Patients and family members (who are the first ones to realize that memory may be failing), frequently ask about reversible causes of Cognitive Dysfunction. They should be investigated first, as 10% of dementia cases are potentially reversible.

Before attributing cognitive decline to a neurodegenerative process, due diligence is warranted:

- Medications/polypharmacy: Anticholinergics (think Benadryl), benzodiazepines, opioids, and sedatives are common culprits — affects one-third of persons >60 years.
- Hypothyroidism (or hyperthyroidism): TSH screening.
- Vitamin B12 deficiency: Serum B12 (and folate).
- Depression ("pseudodementia"): cognitive impairment may resolve with treatment.
- Sleep-disordered breathing: OSA contributes to cognitive impairment; screen with [STOP-BANG questionnaire](#).
- Metabolic derangements: Renal failure, hepatic dysfunction, hypercalcemia, hyponatremia, hyperglycemia.
- Normal pressure hydrocephalus (NPH): Gait disturbance, urinary incontinence, and cognitive impairment — potentially curable with CSF shunting.
- Structural lesions: Subdural hematoma, brain tumor — identified on brain MRI or CT
- Infections: Neurosyphilis, HIV, Lyme disease — test based on risk factors
- Hearing loss: Sensorineural hearing loss is a modifiable contributor
- Vitamin D deficiency: Emerging evidence as a treatable contributor

Recommended evaluation: CBC, CMP (glucose, calcium, electrolytes, renal/hepatic function), TSH, vitamin B12, folate. Brain MRI without contrast is recommended to exclude structural causes and assist with subtype classification.

Up to 90% of Cognitive Decline Are Related to Major Neurodegenerative Dementia Subtypes:

1. Alzheimer Disease (AD) — 40–45% of cases

- Recognition: Insidious onset of episodic memory loss (especially recent events), followed by difficulty remembering names, spatial difficulties, executive dysfunction. MRI shows hippocampal and temporoparietal atrophy in advanced disease. PET scan shows low cingulate and temporoparietal metabolism. Amyloid PET +.

2. Vascular Dementia — 15% of cases

- Recognition: Stepwise decline temporally related to cerebrovascular events, though gradual onset is also possible. History of vascular risk factors (HTN, DM, AF). Occasional focal neurologic deficits. MRI with lacunar infarcts, encephalomalacia. Subcortical microhemorrhages suggest cerebral amyloid angiopathy.

3. Dementia with Lewy Bodies (DLB) — 5% of cases

- Recognition: Visual hallucinations, cognitive fluctuations (alertness and attention), parkinsonism (bradykinesia, rigidity), and REM sleep behavior disorder (often precedes cognitive symptoms by years). Marked spatial impairment with relatively preserved memory early on. MRI initially normal; PET shows generalized hypometabolism including visual cortex ("cingulate island" sign). DaTscan (dopaminergic imaging) assists diagnosis.

4. Parkinson Disease Dementia (PDD)

- Recognition: Dementia developing ≥ 1 year after established motor parkinsonism (the "1-year rule" distinguishes PDD from DLB). Similar cognitive and neuropsychiatric features to DLB.

5. Frontotemporal Dementia (FTD) — ~1% of cases.

- Recognition: Early age of onset (often 45–65 years with two variants: **Behavioral Variant**: Personality changes, disinhibition, apathy, loss of empathy, socially inappropriate behavior, with relatively preserved memory. **Language Variant** (primary progressive aphasia): Impaired speech fluency, semantic paraphasias, progressive aphasia. Imaging: MRI shows frontal and anterior temporal atrophy. FDG-PET shows frontal/anterior temporal hypometabolism. Amyloid PET is negative (key differentiator from AD).

6. Normal Pressure Hydrocephalus (NPH) — Potentially Reversible

- Recognition: Classic triad of wide-based magnetic gait (>90% of cases), urinary urgency/incontinence, and frontal-subcortical cognitive pattern (slowed processing, impaired executive function, preserved naming). Gait usually presents first. Imaging: MRI shows ventriculomegaly out of proportion to cortical atrophy (Evans index >0.3), effaced high-convexity sulci with enlarged Sylvian fissures, aqueductal flow void.

7. Mixed Dementia — Up to 40% of cases

The most common combination is AD + cerebrovascular disease. Amyloid PET helps distinguish mixed dementia from pure vascular dementia. Treatment targets both AD pathology and vascular risk factors.

Subtype	Hallmark Features	First-Line Treatment	Key Caution	
Alzheimer disease	Episodic memory loss, slow insidious onset	Cholinesterase I, ± memantine; lecanemab/donanemab for early AD	ARIA with anti amyloid therapies	
Vascular dementia	Stepwise decline, vascular risk factors, focal def	Vascular risk factor management	No proven pharmacotherapy	
Lewy body dementia	Visual hallucinations, fluctuations, RBD, parkinsonism	Cholinesterase inhibitors	Avoid typical antipsychotics	
PD dementia	Dementia ≥1 yr after motor PD	Rivastigmine (FDA-approved)	Neuroleptic sensitivity	
Frontotemporal dementia	Behavioral changes aphasia; younger	SSRIs for behavior only	Cholinesterase inhibitors may worsen	
NPH	Gait-urinary-cognitive triad	VP shunt	Rule out comorbid AD (~20–57%)	

ALZHEIMER DISEASE: PATHOLOGY BEFORE SYMPTOMS

Amyloid, tau, inflammation, synapses, and a disease that begins quietly

As discussed, Alzheimer disease is the most common cause of dementia, but the clinical syndrome is the end of a long biological process. Amyloid deposition begins often decades before obvious symptoms. Tau pathology spreads more closely with neuronal dysfunction and clinical decline. Synapses fail, networks disconnect, inflammation changes, and vulnerable regions such as the hippocampus lose function.

ANTI-AMYLOID THERAPY IS A MONITORED PATHWAY



Potential benefit is modest; ARIA risk, APOE genotype, anticoagulation and patient goals matter.

The amyloid hypothesis has been both productive and oversold. Amyloid is clearly part of Alzheimer biology and removing it

can modestly slow decline in selected early disease. Yet amyloid alone does not explain the full disorder. Some people carry substantial amyloid without dementia; others have mixed pathologies. Tau, vascular injury, immune response, lipid metabolism, genetics, and resilience all influence the clinical picture.

APOE is the most important common genetic risk locus. Carrying APOE-e4 raises risk and tends to shift onset earlier, but it is not destiny. Many carriers never develop dementia, and many patients with Alzheimer disease do not carry e4. Genetic testing in an asymptomatic person can create anxiety without providing a proven prevention strategy; it should be undertaken only after considering what the result will change.

NEW ALZHEIMER TREATMENTS: CAUTIOUS PROGRESS

Lecanemab, donanemab, modest benefit, and meaningful risk

Lecanemab and donanemab are monoclonal antibodies that reduce brain amyloid and have demonstrated statistically significant slowing of cognitive and functional decline in early symptomatic Alzheimer disease.

Their approval marks genuine progress: after decades of failed trials, disease biology can be altered in a way that changes the clinical trajectory. But “slowing” is not “stopping,” and neither drug restores lost memory. In the lecanemab trial, decline over eighteen months was moderately less than with placebo. Donanemab similarly slowed progression over seventy-six weeks, with larger relative effects in participants with lower tau burden. Translating group averages into an individual prediction is difficult. The practical benefit may be measured in additional months of function, and patients differ in how they value that possibility. Both drugs can cause amyloid-related imaging abnormalities—brain edema or microhemorrhage known as ARIA. Risk is higher in APOE-e4 carriers, particularly homozygotes, and requires MRI monitoring. Anticoagulation, extensive microbleeds, vascular disease, access to infusion centers, transportation, caregiver support, and willingness to accept uncertainty all matter. Treatment is therefore a pathway, not a prescription written after a brief visit. These treatments are intended for mild cognitive impairment or mild dementia due to Alzheimer disease with confirmed amyloid pathology—not for normal forgetfulness, moderate or advanced dementia, or general prevention. Their emergence should produce neither dismissal nor triumphalism. They are useful but imperfect first-generation therapies and a foundation for combinations that may eventually target amyloid, tau, inflammation, and synaptic resilience together.

The first disease-modifying treatments are a beginning, not the “destination”.

BLOOD BIOMARKERS AND PREVENTION

Powerful tools require careful questions; risk reduction is real, certainty is not

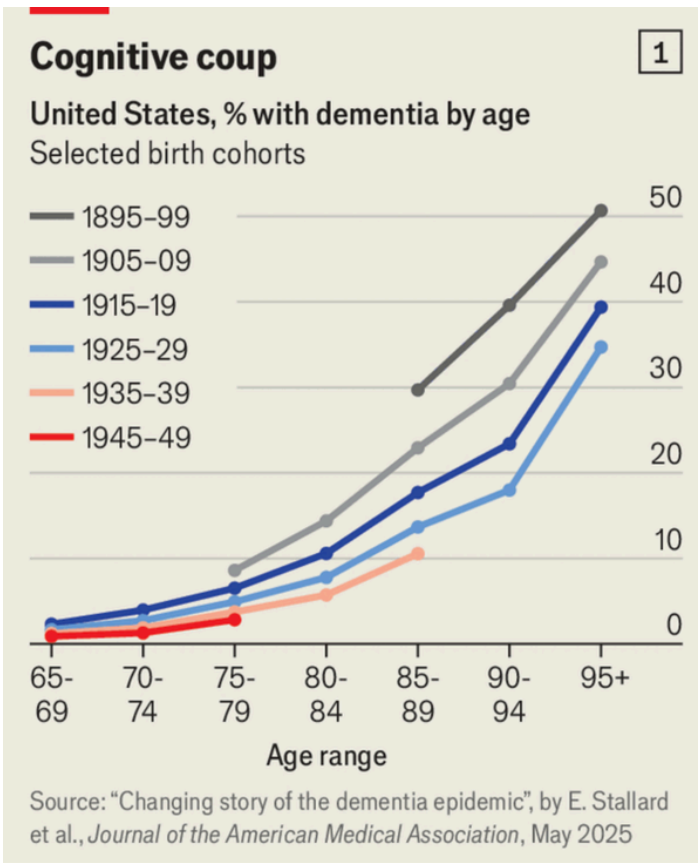
Until recently, confirming Alzheimer pathology generally required cerebrospinal fluid obtained by lumbar puncture or an expensive amyloid PET scan. Blood biomarkers—especially phosphorylated tau species such as p-tau217—are changing that landscape. In 2025, the FDA cleared the first blood test intended to aid identification of amyloid pathology in symptomatic adults. A positive result suggests amyloid pathology; it does not prove that Alzheimer disease is

the sole cause of symptoms. An indeterminate result may require additional testing. The promise is substantial. Plasma p-tau217 is the best-performing blood-based Alzheimer biomarker, achieving diagnostic accuracy comparable to CSF and PET reference standards, outperforming the standard clinical evaluation by primary care physicians.

AN EPIDEMIC—OR A CHANGING RISK?

Total case numbers can rise while age-specific risk falls

Few conditions are feared as deeply as dementia. Its threat is not only death or disability, but



the gradual erosion of identity, judgment, language, independence, and trust in oneself. The novelist [Sir Terry Pratchett](#), who developed a rare form of Alzheimer's disease, described a world receding from him in every direction. Families often experience a related grief: the person remains physically present while familiar parts of personality and shared history become harder to reach. The demographic headlines are frightening. As populations grow older and live longer, the total number of people with dementia is projected to rise sharply. Those projections are important for health systems and caregivers, but can obscure more hopeful trends: In several wealthy countries, age-adjusted dementia incidence can fall even while total case numbers rise. That distinction matters. Dementia risk rises steeply with age, so even a modest delay in average onset can have an enormous effect. Delaying onset by approximately five years could, in principle, reduce the number of people living with dementia by roughly half.

That is not a promise to any individual; it is a reminder that prevention need not be perfect to be profoundly valuable. The news is not uniformly reassuring. Declines have been observed mainly in higher-income countries and may be threatened by diabetes, obesity, inactivity, unequal access to care, and worsening cardiovascular risk in younger generations.

The correct conclusion is neither complacency nor panic. Dementia remains a formidable challenge, but the trajectory is not fixed. The total burden can rise while the age-specific risk may fall. Both can be true.

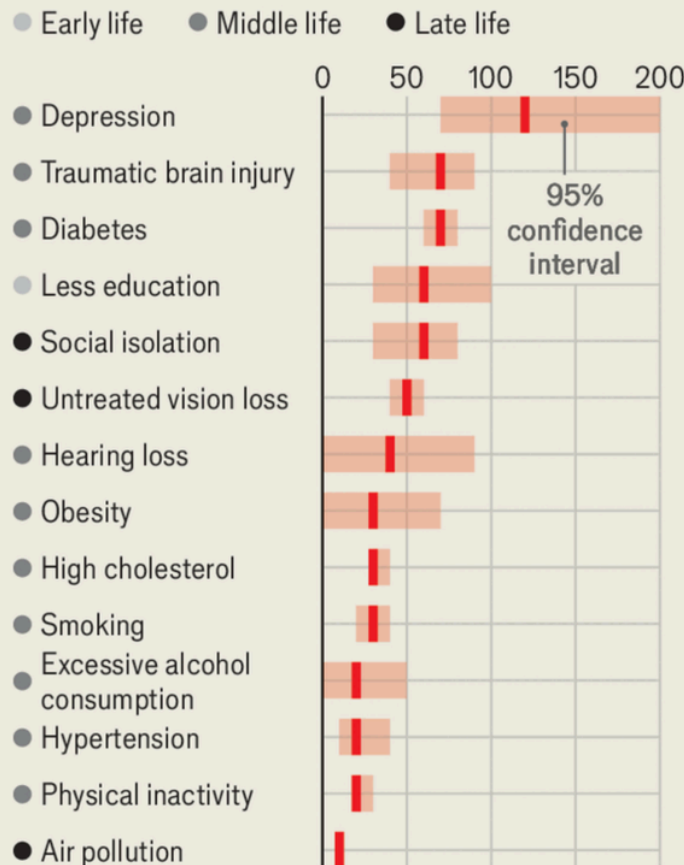
PREVENTION AND AN UNEXPECTED CLUE

Modifiable risks—and an intriguing shingles-vaccine signal

Mind map

2

Potentially modifiable risk factors for dementia, higher relative risk, %



Source: "Dementia prevention, intervention and care",
Lancet Commission report, 2024

The 2024 Lancet Commission estimated that as many as 45 percent of dementia cases might be delayed or prevented by addressing fourteen potentially modifiable factors across the life course. The estimate is a population-level model, not a guarantee for any individual. It reminds us that modest effects on common risks—hearing loss, high LDL cholesterol, hypertension, physical inactivity, diabetes, depression, social isolation, smoking, excessive alcohol, visual loss, traumatic brain injury, air pollution and limited education—can add up.

Medical discoveries sometimes emerge from natural experiments. In Wales, an age-based eligibility rule for the older shingles vaccine created two nearly comparable groups on either side of a birthday cutoff. Subsequent analyses reported roughly a 20 percent lower risk of dementia among those eligible for vaccination, and related studies in other populations have produced encouraging signals. The biology is plausible, but the mechanism and the size of any causal effect remain uncertain. Shingrix is already recommended as it substantially reduces shingles and postherpetic neuralgia. The possible dementia benefit is an intriguing additional reason to be vaccinated.

Further Reading

- Preserving dignity: [Dementia Reimagined](#). Tia Powell.
- Family and caregiver: [The 36-Hour Day](#). Nancy L. Mace & Peter V. Rabins.
- Caregiver's grief: [Loving Someone Who Has Dementia](#). Pauline Boss.
- Patient's experience: [What I Wish People Knew About Dementia](#). Wendy Mitchell.
- Multiple resources: [Alzheimer's Association](#)
- [World Alzheimer Report 2023–Reducing Dementia Risk: Never too early, never too late](#)

BRAIN HEALTH — ONE-PAGE CHEAT SHEET

Protect the brain by protecting the body, senses, relationships, and capacity to keep learning.

The 2024 Lancet Commission identified 14 potentially modifiable dementia risk factors across life and estimated that addressing them could prevent or delay about 45% of dementia cases at the population level.

♥ MOVE

Aerobic activity + strength training. Sit less.

• KNOW YOUR NUMBERS

Treat high blood pressure, diabetes, obesity, and high LDL cholesterol.

♦ EAT WELL

Mediterranean-style: plants, legumes, whole grains, nuts, olive oil, fish.

▲ CHALLENGE THE BRAIN

Learn, read, create, solve problems; build cognitive reserve across life.

⬇️ PROTECT YOUR HEAD

Prevent falls and traumatic brain injury; use seat belts and appropriate helmets.

☾ SLEEP

Protect restorative sleep; evaluate suspected sleep apnea.

• HEAR + SEE

Correct hearing and vision loss; use prescribed aids consistently.

↔️ CONNECT

Maintain friendships, family ties, community and meaningful contact.

✖️ AVOID TOXINS

Don't smoke. Avoid heavy alcohol use. Minimize air-pollution exposure when feasible.

☺️ CARE FOR MOOD

Recognize and treat depression; mental and cognitive health are connected.

THE 14 MODIFIABLE RISK FACTORS

EARLIER LIFE

Less education / fewer opportunities for cognitive enrichment

MIDLIFE

Hearing loss • high LDL • hypertension • smoking • obesity • depression • physical inactivity • diabetes • excessive alcohol • traumatic brain injury

LATER LIFE

Social isolation • air pollution • untreated vision loss

YOUR QUICK BRAIN-HEALTH CHECK

☐ BP controlled

☐ LDL/glucose reviewed

☐ Exercise: aerobic + strength

☐ Hearing & vision addressed

☐ Sleep is restorative

☐ No smoking / no heavy alcohol

☐ Mood addressed

☐ Socially connected

☐ Still learning & challenging yourself

Seek evaluation when cognitive changes interfere with function rather than waiting for a crisis. The goal is not to eliminate risk—an impossible promise—but to shift the trajectory, delay disability, and preserve independence for as long as biology and luck permit.

Vaccinate if appropriate.

THE TAKE HOME:

Think in decades, not days. Move • sleep • learn • connect • hear • see • protect your head • and aggressively protect vascular health.

Risk reduction is not guaranteed. Age and genetics still matter. We can develop dementia despite doing everything “right.”

LOOKING AHEAD

What the next decade may bring—and what will remain true

Could the Brain Be About to Have Its "Ozempic Moment"?

One of the biggest medical stories of the past decade has been the remarkable success of GLP-1 medications such as Ozempic, Wegovy, Mounjaro, etc. Originally developed to treat diabetes, these drugs have since proven beneficial for obesity, heart disease, kidney disease,

sleep apnea, and several other conditions. They changed the way we think about treating disease by targeting one of the body's master regulatory systems rather than a single symptom.

Recently, the FDA approved the first orexin agonist, **oveporexton**, for the treatment of narcolepsy type 1, a disorder characterized by overwhelming daytime sleepiness and sudden episodes of muscle weakness known as cataplexy. Unlike traditional stimulants, which broadly increase dopamine or norepinephrine and may cause anxiety, elevated blood pressure, or dependence, orexin agonists aim to restore the brain's own wakefulness system, producing a more natural state of alertness.

What makes this development especially intriguing is its potential beyond narcolepsy. Because orexins influence attention, motivation, mood, and reward, researchers are exploring whether these drugs might eventually help treat conditions such as excessive daytime sleepiness, ADHD, depression, addiction, and even some forms of sleep apnea. These possibilities remain experimental, and larger clinical trials are needed before any conclusions can be drawn. The eventual therapeutic target may be circadian “orexin” optimization rather than maximal orexin activation, with possible benefits for cognitive restoration, neuronal recovery, possibly delaying onset of conditions such as Parkinson's and Alzheimer's Disease. Plausibly, the best “brain-aging” drug may involve a combination of Orexin OX2R activation during daytime and a nighttime Orexin suppression!

But if the history of GLP-1 medications teaches us anything, it is that targeting a fundamental biological control system can sometimes produce benefits far beyond what anyone originally imagined **(and another reason why having political appointees decide how research dollars are allocated is absurd and only concocted by the Trump “administration” and his sycophants)**. Whether orexin therapies will follow the same path remains uncertain, but they represent one of the most exciting new frontiers in neuroscience and another reminder that some of the greatest advances in medicine come not from treating individual symptoms, but from understanding—and restoring—the body's own remarkable regulatory systems.

The brain works as an integrated network, and future therapies are increasingly aiming to restore those networks rather than simply masking symptoms.

Brain medicine is entering an era of earlier detection and biological classification. Blood biomarkers will become more accurate and more available. Tau imaging may help stage disease and select therapies. Artificial intelligence will identify subtle patterns in speech, gait, retinal images, electrophysiology, and MRI. These tools may shorten diagnosis, but algorithms will also amplify bias and incidental findings unless used carefully.

The future will probably include better risk stratification, more convenient biomarkers, therapies that target tau, inflammation and synaptic resilience, and more precise selection of patients for anti-amyloid treatment and new biologic pathways. Yet the foundations will remain remarkably familiar: education, movement, sleep, hearing, vision, cardiovascular health, metabolic stability, purpose, and human connection.

There is no magic pill. Increasing longevity takes hard daily work.

SECTION II • DEBUNKING MEDICAL MYTHS: Q & A

Eight years of Medical Bits — question the mantra, then examine the evidence

Q: Do I need an annual pneumonia vaccine?

A: Myth. Pneumococcal vaccination is an annual vaccine. NO. Current CDC guidance recommends PCV15, PCV20, or PCV21 for adults aged 50 and older who have not received a pneumococcal conjugate vaccine or whose history is unknown. PCV20 or PCV21 usually completes the series; if PCV15 is used, one PPSV23 dose is generally given later. Prior vaccines and certain risk conditions change the schedule. Discuss with your doctor.

Q: Is all fiber equally beneficial?

A: Myth. Whole foods naturally rich in fiber—vegetables, legumes, fruit, nuts, and intact grains—bring micronutrients and a complex food matrix. A processed food does not become healthy merely because isolated fiber was added to it. Some supplemental fibers do have specific evidence and clinical uses, but “contains fiber” is not a magic statement.

Q: Do upper-respiratory infections usually need antibiotics?

A: Myth. Most uncomplicated upper-respiratory infections are viral. Antibiotics do not shorten viral illness and can cause adverse effects, Clostridium difficile infection, and antimicrobial resistance. Bacterial sinusitis, streptococcal pharyngitis, pneumonia, and selected complications are different questions and require evaluation.

Q: Do we use only ten percent of our brain?

A: Myth. Brain imaging and neurologic disease show that essentially every region has a role, although not every neuron fires maximally at the same moment. The myth survives because it flatters us with the idea of enormous dormant capacity. The real opportunity is more interesting: networks can become more efficient and flexible through learning, movement, recovery, and practice.

Q: Should we all take dietary supplements?

A: Myth. The supplement market contains roughly 100,000 products and is worth more than \$60 billion annually. Supplements are regulated differently from drugs, and most have not demonstrated clinically meaningful benefit in randomized trials for preventing memory loss, low energy, heart disease, aging, neurodegenerative disease, etc.

“Natural” does not mean effective, pure, or risk-free.

Q: When may supplements be beneficial?

A: When there is a defined indication. Examples include a documented vitamin or mineral deficiency; vitamin B12 for many vegans and for selected patients after gastric or terminal-ileal surgery; fat-soluble vitamins for certain malabsorption syndromes, cystic fibrosis, inflammatory bowel disease, or bariatric surgery; folic acid and prenatal vitamins with iron in pregnancy; and AREDS2 formulations for appropriate stages of age-related macular degeneration. Multivitamin trials have suggested a possible small cognitive benefit in some older adults, but the finding requires perspective and is not Alzheimer’s treatment.

Q: Does a biologically plausible mechanism prove that a supplement works?

A: No. A molecule can influence a pathway in yeast, nematodes, flies, mice, or a laboratory dish and still fail to improve human health. Mechanism is the beginning of a clinical question, not the answer.

Q: Do crossword puzzles prevent dementia?

A: Not by themselves. Puzzles can be enjoyable and cognitively engaging, but repeated practice mainly improves the practiced task. A broader life of learning, movement, sensory health, social participation and vascular risk control is more beneficial.

Q: Does one glass of wine protect the brain?

A: The older observational story has weakened. When confounding issues are addressed, the apparent benefit shrinks. Wine may be an enjoyable social ritual, but no amount is healthy.

Q: Does a family history make dementia inevitable?

A: No. Genes influence risk, but they do not write the entire future. Age, vascular health, education, sensory function, lifestyle, and chance all matter. Even APO-E4 is a risk factor, not a final verdict. Keep it simple. When benefit is not proven, restraint is the most scientific and effective choice (in all aspects of life).

RESOURCES ON THE WEB

Perspective, movement, balance, mindfulness, and practical health

We have recommended these resources in prior issues of Medical Bits; the hyperlinks are active in Word and Google Docs. The first group will make you smile—or at least remind you of our improbable and yet, magnificent existence.

PERSPECTIVE AND THE UNIVERSE

- [A time-lapse of the entire Universe \(10 minutes\)](#)
- [The massive expanse of our Universe \(6 minutes\)](#) and our magnificent insignificance.
- [The relative sizes of the Universe](#) will remind you of our improbable, yet, amazing existence
- [These APOD Videos](#) will make you laugh!
- [Nothing Lasts Forever—Carl Sagan](#)

MOVE

- [The Scientific 7-Minute Workout](#)
- [Get the Seven workout app](#)
- [An 11-minute body-weight workout](#)
- [A 9-minute strength/core routine](#)
- [35 simple health tips](#)
- [Can you pass the 10-second balance test?](#)
- [Pelvic-floor exercises](#)

MINDFULNESS

- [Start exploring mindfulness](#)

Remember: movement does not have to be elaborate to matter. Seven minutes is better than zero; eleven minutes is better than seven; and a daily walk becomes more valuable when combined with strength, balance, and occasional intensity. Mindfulness is not a substitute for treatment, but regular practice can improve attention, stress, blood pressure, pain tolerance, and well-being becomes more valuable when combined with strength, balance, and occasional intensity.

Exercise is candy for our muscles. They need intermittent daily activity to remain metabolically healthy.

SECTION III • OFFICE UPDATES

Office news, coverage, and reminders

- We continue to provide Concierge General Medicine and Consultative Pulmonary Medicine at

5454 Wisconsin Avenue, Suite 1710, Chevy Chase, Maryland 20815.

- Parking access is at the back of the building, and free valet parking is available. Ask for validation at the office (through the Gym, on the 18th floor).
 - Medication refills and clerical support:
 - Lexi Manning: Alexandra.Manning@premier-medicine.com
 - Olivia Dragovits: ODragovits@premier-medicine.com
 - Appointments: Ashley Alvarez AAlvarez@premier-medicine.com or 301-637-5555.
 - Insurance and billing: Brenda Perez BPerez@premier-medicine.com
 - Sadly, our wonderful Nurse Practitioner, Irene Rieder, is leaving on October 1. She has decided to dedicate more of her energy to building her family and new career pursuits. Her email is Irene.Burgos@premier-medicine.com. We are actively recruiting but know that she will not be easy to replace.
 - Dr. Picone: cpicone@premier-medicine.com
 - For urgent matters, my cell phone is 301-943-5189
 - If you are hospitalized, please ask the admitting physician to notify me.
 - Email remains the preferred method of communication because it allows documentation and delegation. Please, keep messages succinct and use bullet points.
 - I will be away on vacation September 7 through September 19, 2026.
- My Nurse Practitioner and my colleagues, Drs. Vassallo, Talesnick, Fishman, and Shapiro, will cover in my absence—but I am never too far from email or phone, as you well know.

You know that the only certainty in life is death—and the only fountains of youth proven by science and experience are love, exercise, laughter, humor, and a positive attitude.

Have a healthy season—and continue to learn as if you intend to live forever.

Carlos E. Picone, MD

1 FROM THE BIG BANG TO THE HUMAN BRAIN

A 13.7 BILLION-YEAR JOURNEY



2 THE SEVEN PILLARS OF BRAIN HEALTH

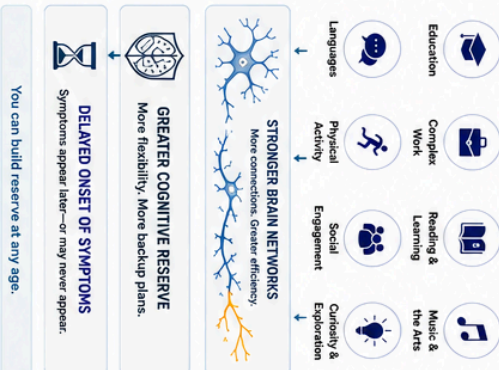
Small daily choices. Big long-term impact.



These pillars work together to support brain cells, strengthen connections, and preserve cognitive function.

3 COGNITIVE RESERVE: YOUR BRAIN'S SAVINGS ACCOUNT

Built over a lifetime. Borrowed when needed.



4 SLEEP: YOUR BRAIN'S NIGHT SHIFT

Restore. Clear. Consolidate.



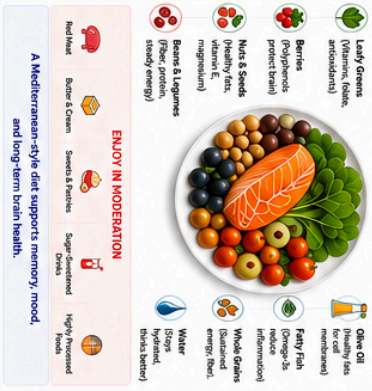
5 EXERCISE CHANGES YOUR BRAIN

Move more. Think better.



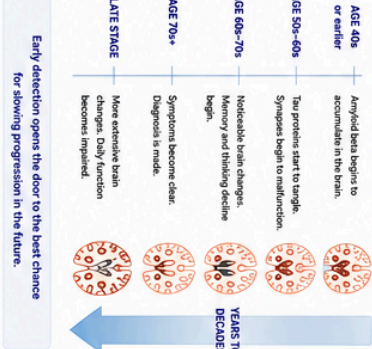
6 THE MEDITERRANEAN PLATE FOR BRAIN HEALTH

Eat to think, not just to live.



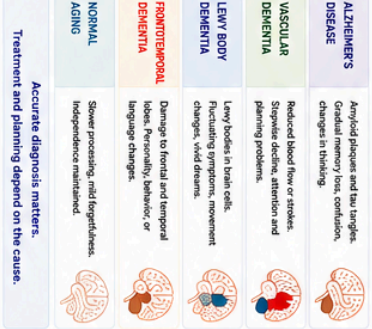
7 ALZHEIMER'S DISEASE: A LONG SILENT TIMELINE

Changes begin decades before symptoms.



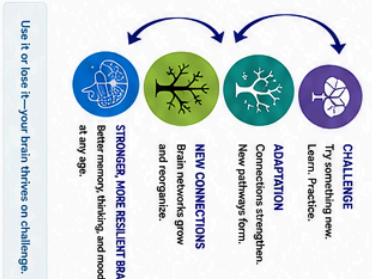
8 DIFFERENT DEMENTIAS: DIFFERENT CAUSES

Different diseases. Different patterns.



9 NEUROPLASTICITY: THE BRAIN'S SUPERPOWER

The brain can change throughout life.



10 MEDICAL MYTHS vs. FACTS

Question claims. Look for evidence.



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