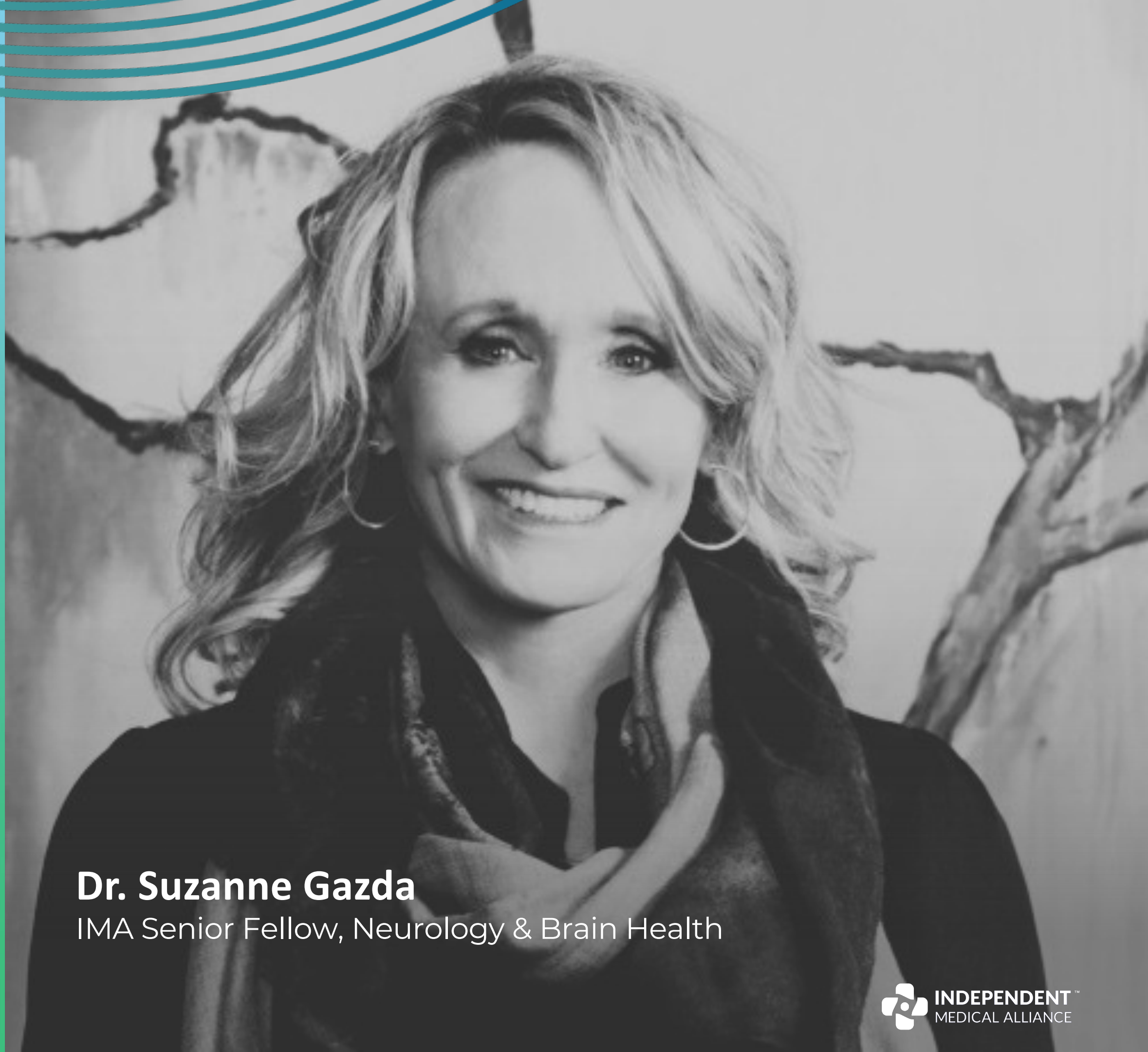




**EARLY DEMENTIA – DIAGNOSIS
AND REVIEW OF EVIDENCE-
BASED AND EMERGING
TREATMENT APPROACHES**



Dr. Suzanne Gazda
IMA Senior Fellow, Neurology & Brain Health



**Early Dementia: Diagnosis and Review of
Evidence-Based And Emerging Treatment Approaches
with
Suzanne K. Gazda MD**

A Global Crisis of Cognitive Decline A Dementia Storm on the Horizon

- Cognitive strength is our most valued asset. And yet, we are seeing an unprecedented decline in cognitive health
- Cognitive impairment is a growing global health crisis with profound implications for individuals, families, and society at large
- Over 7 million Americans are living with Alzheimer's disease, and the numbers are rapidly rising
- It is estimated that dementia will afflict approximately 50 percent of the next generation of senior citizens (that would be every other person between the ages of 40 and 60 reading this article right now).

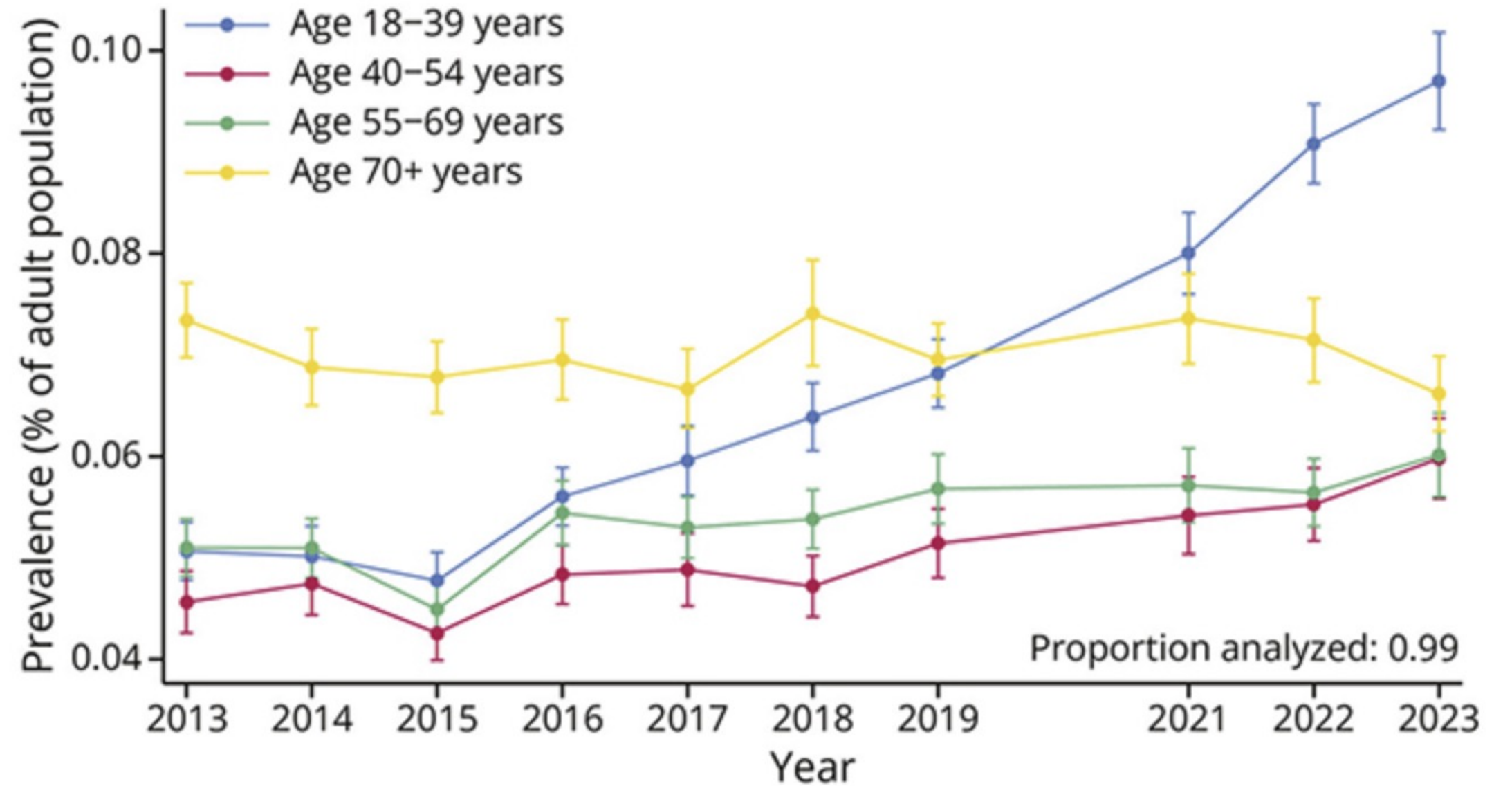
Risk and future burden of dementia in the United States NIH and the Alzheimer's Association

Cognitive Decline is Happening in Younger Populations

- A study analyzing more than 4.5 million U.S. adults has uncovered a striking trend: self-reported cognitive disability—difficulty concentrating, remembering, or making decisions—has surged by 40% over the past decade (Wong K 2025)
- Researchers found the percentage of overall adults reporting cognitive disability increased from 5.3% in 2013 to 7.4% in 2023, with **young adults** (ages 18 to 39) seeing the biggest rise. (Herrera E)



Memory Problems Surging in Adults Under 40 (Herrera E)

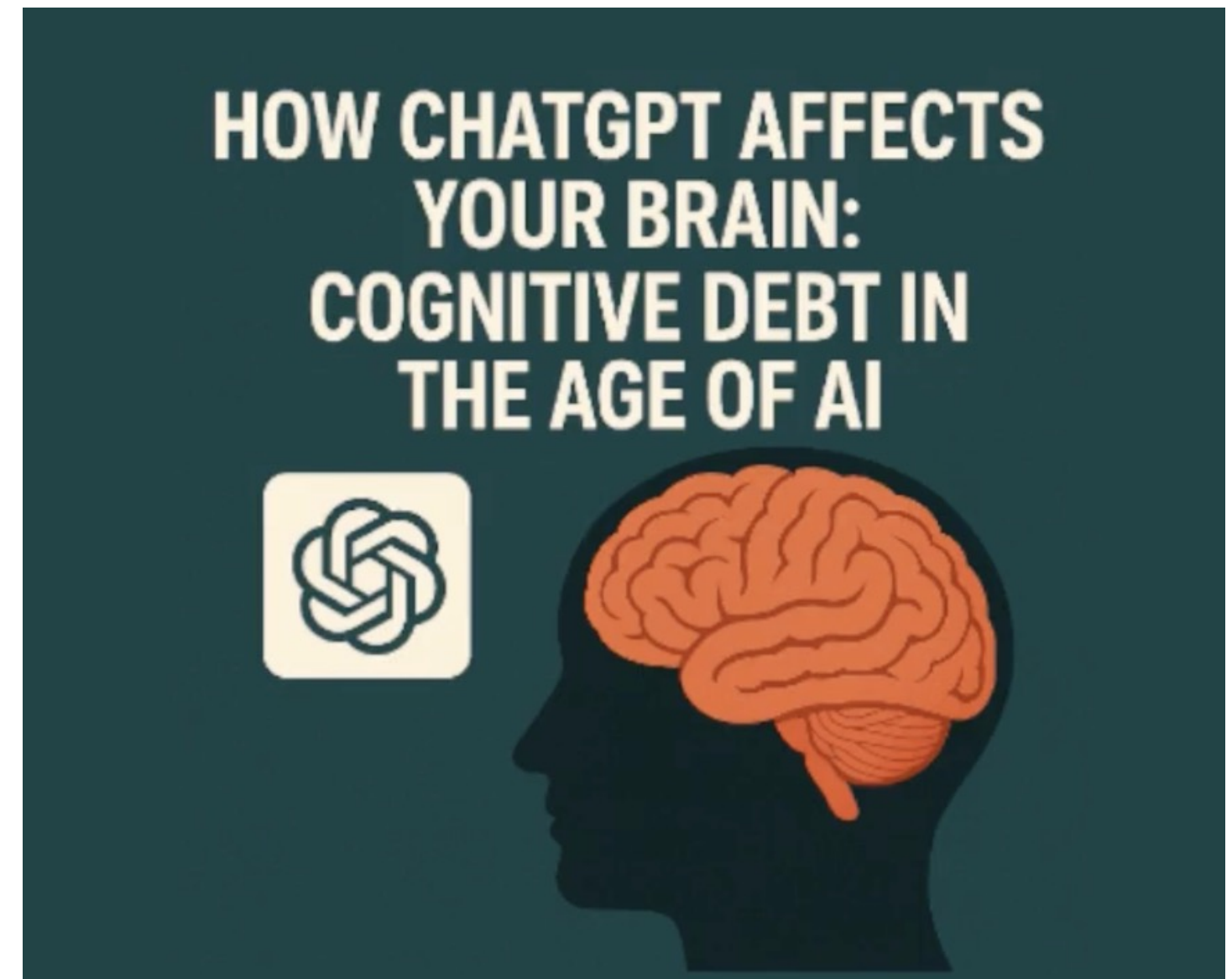


Self-reported cognitive disability rates rose in all age groups except those aged 70 and over. (Wong et al., *Neurol.*, 2025)

Is there a Hidden Cost to AI on Brain Health ?

- 54 subjects—18 -39y/o were divided into three groups and asked them to write several SAT essays using OpenAI's ChatGPT, Google's search engine, and nothing at all, respectively. Researchers used an EEG to record the writers' brain activity across 32 regions (study done at MIT)
- ChatGPT users had the lowest brain engagement and “consistently underperformed at neural, linguistic, and behavioral levels (Kosmyna N 2026)
- We lose skills of analyzing neural connectedness and critical thinking skills

Zammit AR 2026 found lifelong cognitive enrichment was an important measure to maintaining brain health



Cognitive Decline Post 2020

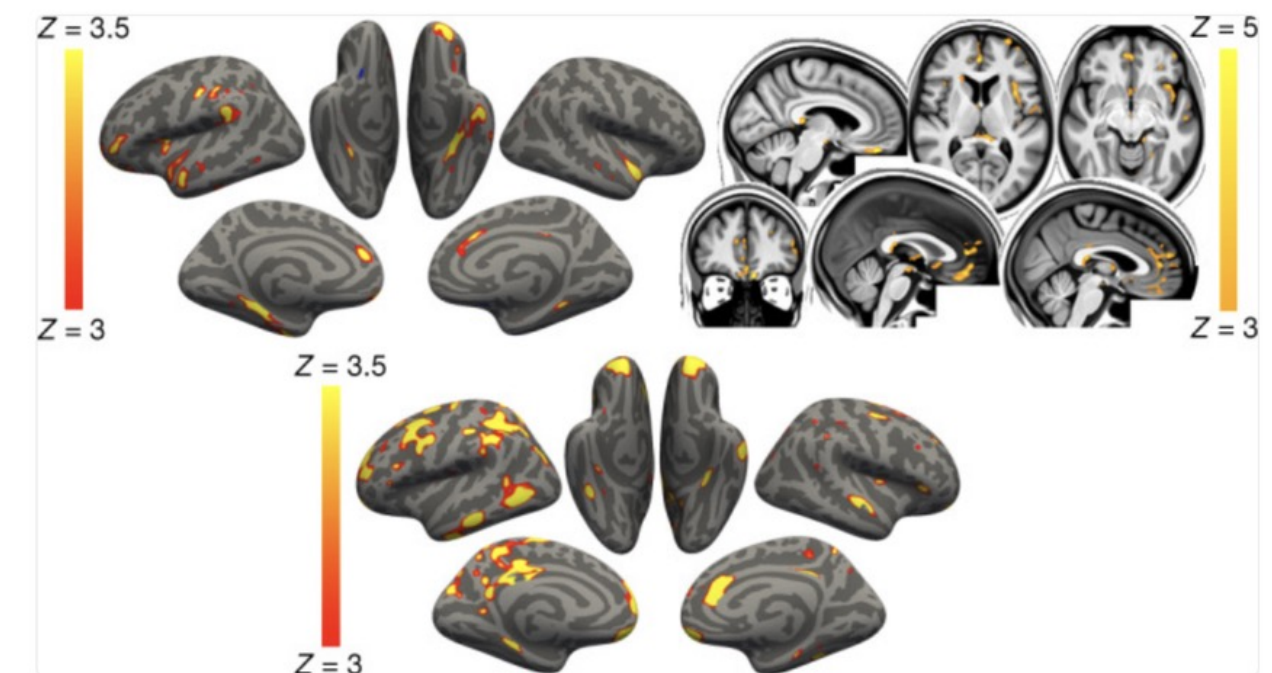
- In the U.S., a significant rise in cognitive decline, often referred to as "brain fog," has been documented following COVID-19 infections. (Becker J)
- A JAMA Network Open survey found that 57% of American adults with post-COVID-19 conditions reported cognitive symptoms daily.(Loque J)
- ADOD burden grew most rapidly in 2019–2021, suggesting COVID-19 accelerated neurodegeneration (Gi J 2026)
- Having recovered from COVID is now linked to a risk of neurodegenerative disease (Javet A) (Bedran D) (Zhao J) (Bonhenry D) (Zhao Y)



Even Mild Covid Can Lead to an Overall Reduction in the Size of the Brain Equivalent to 10 years of Aging (UK Biobank 2021)

- SARS-CoV-2 infection was associated with subtle but significant changes in brain structure and function post Covid (Douaud G 2021)
- Reduced grey matter volume in the orbitofrontal cortex, para-hippocampal gyrus, and the olfactory cortex (smell)
- Altered functional connectivity between the hippocampus and orbitofrontal cortex, even in mild infections
- Fallout: Memory, executive function, visuo-spatial processing, decision making, emotional regulation

Fig. 2. Vertex-wise and voxel-wise longitudinal group differences in grey matter thickness and changes.



PRESS RELEASE

COVID-19 Associated with Long-Term Cognitive Dysfunction, Acceleration of Alzheimer's Symptoms

Press release from
Alzheimer's Association
International Conference
(AAIC®) 2021

- Cumulative data points to disturbing trends showing COVID-19 infections leading to lasting cognitive impairment and even Alzheimer's symptoms
- Alzheimer's Association International Conference (AAIC) report the presence of brain fog was proposed as a high risk of having AD

[Home](#) > [Journal of Neurology](#) > [Article](#)

Need for awareness and surveillance of long-term post-COVID neurodegenerative disorders. A position paper from the neuroCOVID-19 task force of the European Academy of Neurology

Volume 272, article number 380, (2025) [Cite this article](#)

Results

Autopsy studies revealed diverse changes in the brain, including loss of vascular integrity, microthromboses, gliosis, demyelination, and neuronal- and glial injury and cell death, in both unvaccinated and vaccinated individuals irrespective of the severity of COVID-19. Recent data suggest that microglia play an important role in sustained COVID-19-related inflammation, which contributes to the etiology initiating a neurodegenerative cascade, to the worsening of pre-existing neurodegenerative disease or to the acceleration of neurodegenerative processes. Histopathological data have been supported by neuroimaging, and epidemiological studies also suggested a higher risk for neurodegenerative diseases after COVID-19.



Memory and concentration problems are common in long COVID and must not be ignored, say scientists

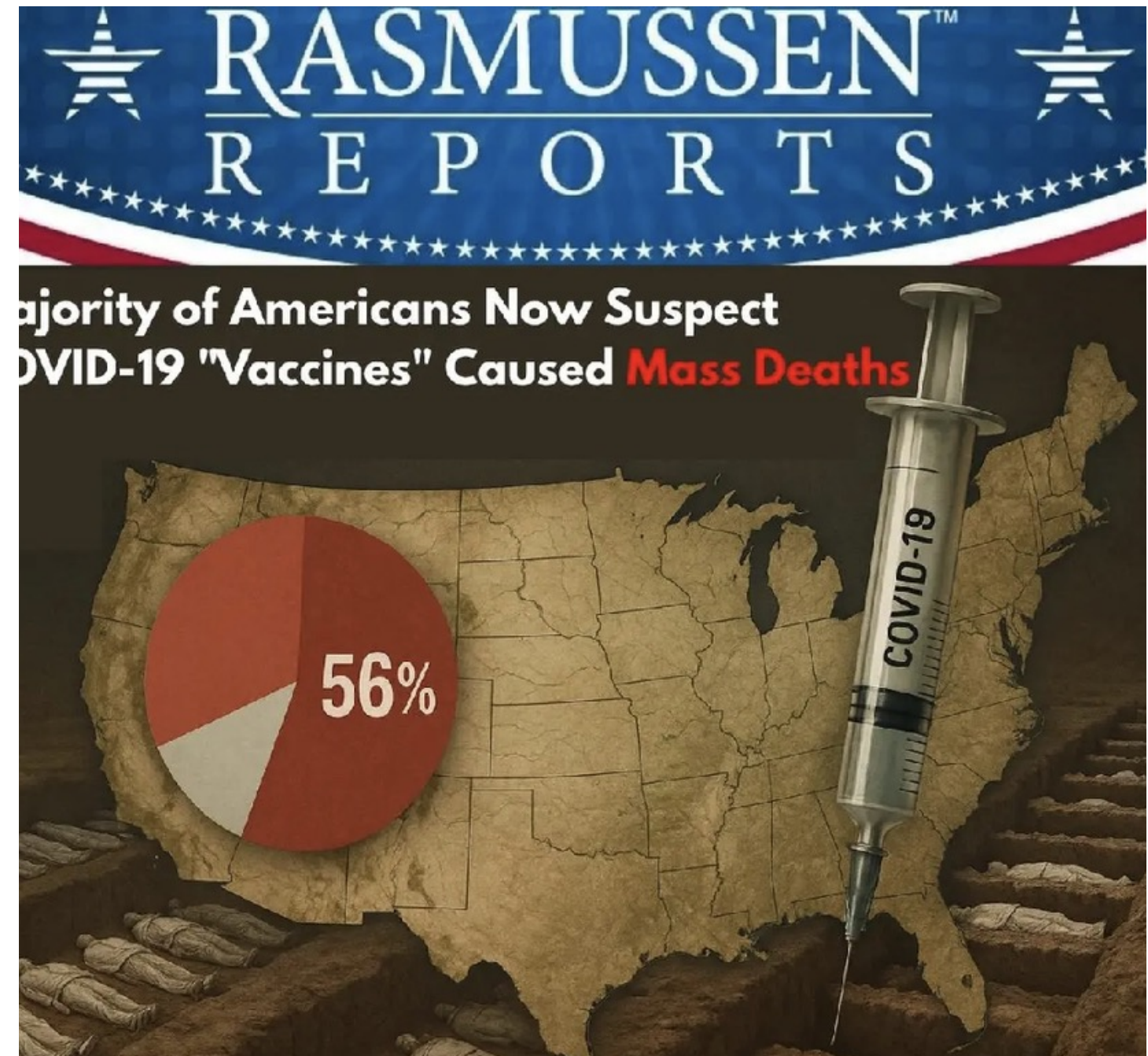
What is Causing the Rise of ‘Forgetfulness’?

PASC and PACVS: A Global Crisis

- Long COVID and PACVS represents a profound, enduring public health crisis with multi-symptoms and multi-organ involvement
- These are one of the most complex, widespread, and under-addressed biological crises of our era with no resolution in sight
- The associated cognitive decline needs our attention

Untold Unspoken Harm

- 9%–34% of COVID vaccine recipients developed side effects with 7-13% developed serious side effects
- 7.5-22% know someone with a severe vaccine injury
- 24-28% know someone who they believe died from the vaccine
- **46-55% believe the COVID vaccines have killed a significant number of people** (Rasmussen Report 2025)
- 63 million had side effects from the COVID-19 vaccine, including more than 17 million who experienced major side effects. (based on US adult population of 258 million)



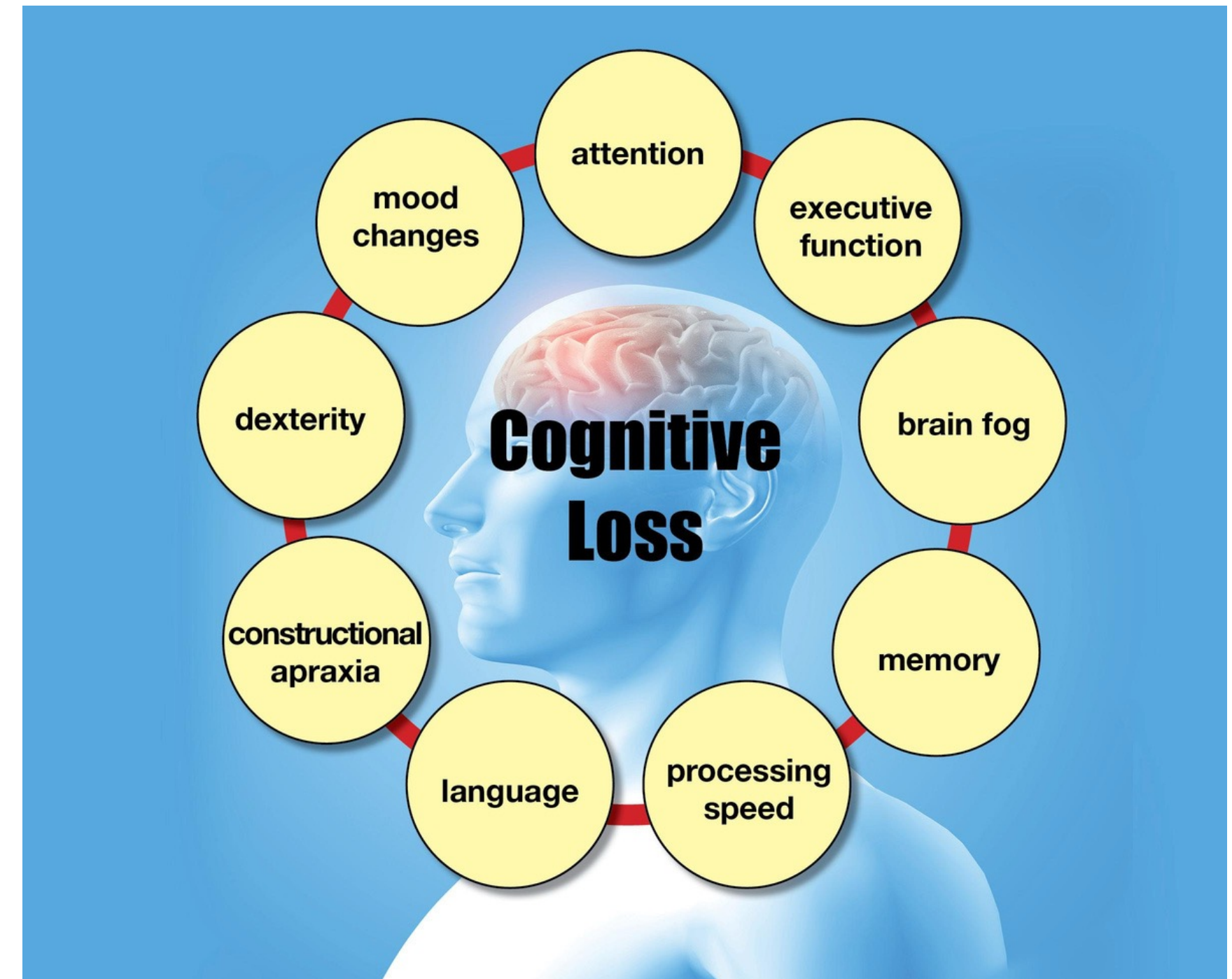
Millions Are Suffering with Brain Fog

- Cognitive Decline is very common in Long Covid and with PVI (Jimenez M)(Van der Feltz-Cornelis C)(Davis H) (React 19 data)
- The onset of cognitive decline can be very rapid post vaccine (Chaurasia A)
- Millions of Americans with mild cognitive impairment are undiagnosed (Liu Y 2023)
- Estimates 86% ((Jimenz M 2026) vs 20-50% (Feltz-Cornelis C 2024)
- Long COVID is an independent risk factor for dementia (Frontera J)



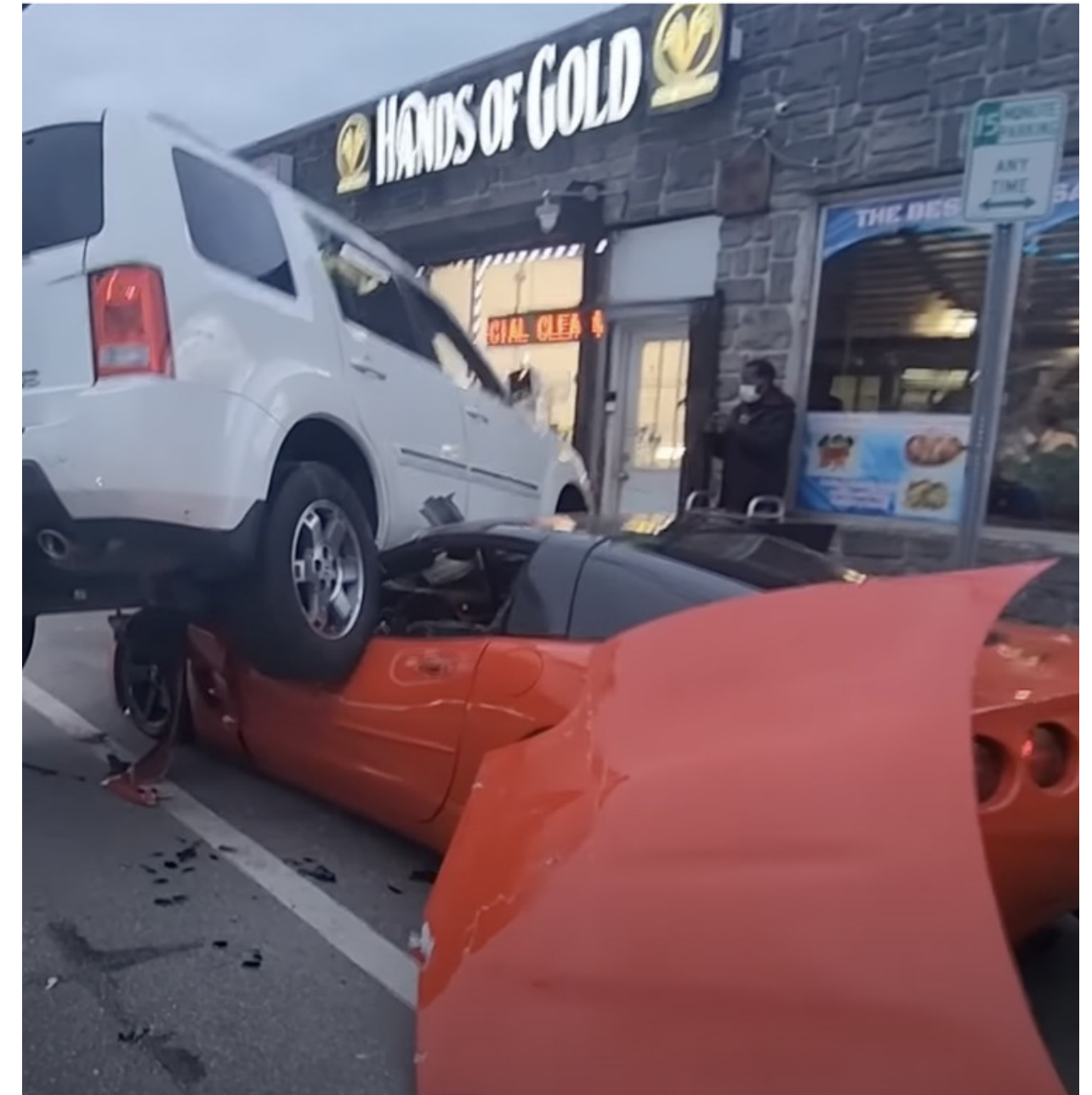
Brain Fog and Cognitive Loss in Long COVID

- Many cognitive domains can be affected
- Memory and attention deficits can last more than 18 months after infection, even in non-hospitalized patients (Serafim A)



The Reckless Driving Surge or Cognitive Impairment post 2020 ?

- Marked difficulty with constructional skills with visuospatial impairment and constructional apraxia are found post COVID (average age 38) (De Paula J)
- PASC cognitive impairments are at the same magnitude as intoxication at the drink driving limit or 10 years of cognitive aging (Holdsworth DA 2022)
- Driving styles have not fully returned to pre-pandemic levels (Kummetha V 2026)
- The lingering effects of COVID-19 on neurological function may be a risk factor for behavior leading to road deaths (Robertson L 2025)



Is Long COVID The New Alzheimer's?

- A large study of over 2,30,000 patients found that the risk of dementia was significantly higher in the post COVID patients than controls (mean age 42.5) Taquet 2022
- People who got COVID-19 were twice as likely to receive a diagnosis of Alzheimer's disease in the 12 months after infection (Xu E 2022)
- Women 65 and older were more prone to developing Alzheimer's disease one year post infection. 85 year or older at highest risk (Wang 2022)
- Long COVID nearly quadruples the long-term risk of Alzheimer's-related mild cognitive impairment, turning a viral illness into a potent accelerator of neurodegeneration (Frontera J 2026)

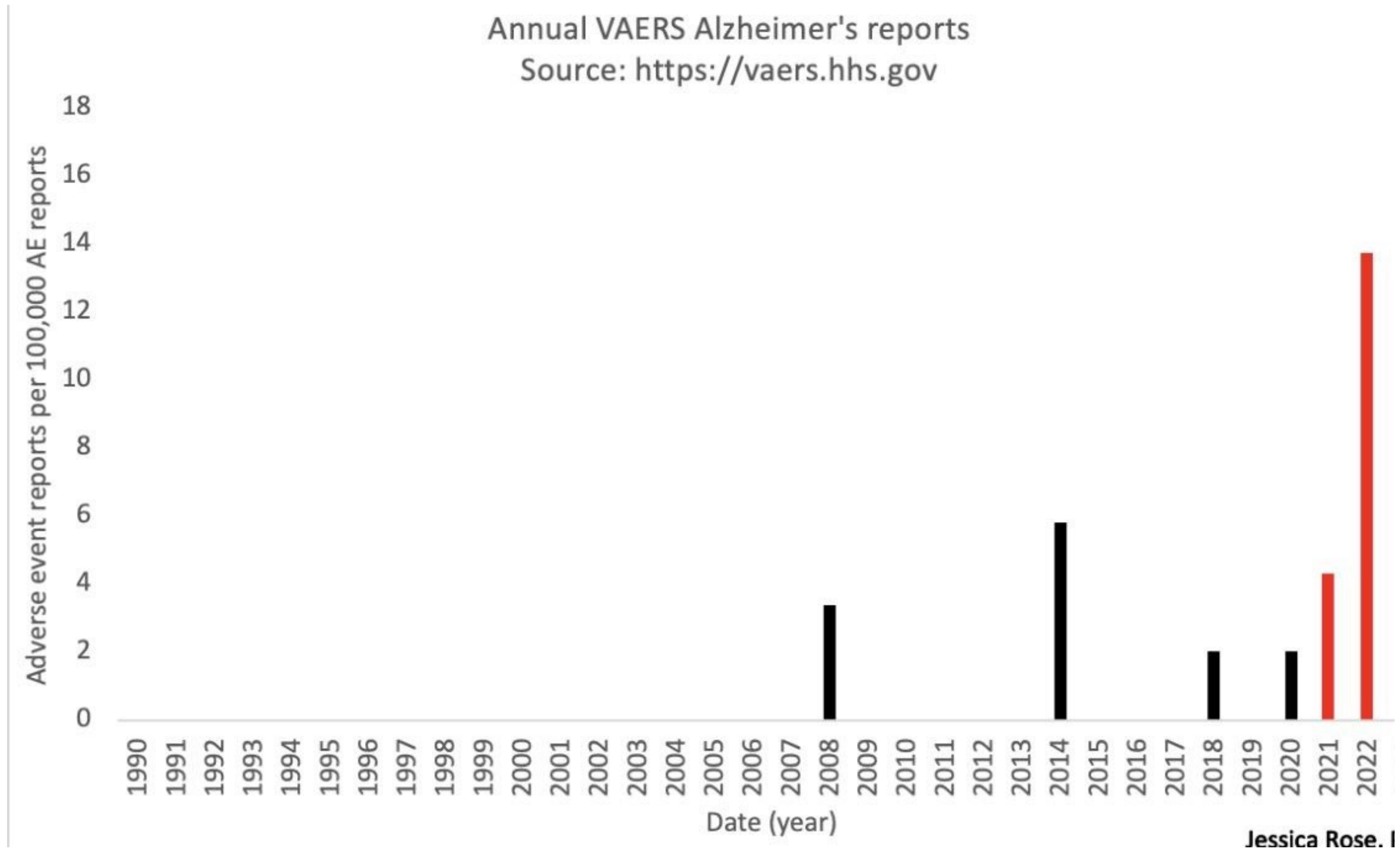
NEWS

Alzheimer's and Long COVID could they be related?

by: [Brooklynn Norris](#), [Stuart Price](#)

Posted: Aug 13, 2022 / 02:23 PM CDT

Updated: Aug 13, 2022 / 02:23 PM CDT



SARS COV 2 is a Risk Factor for ADOD

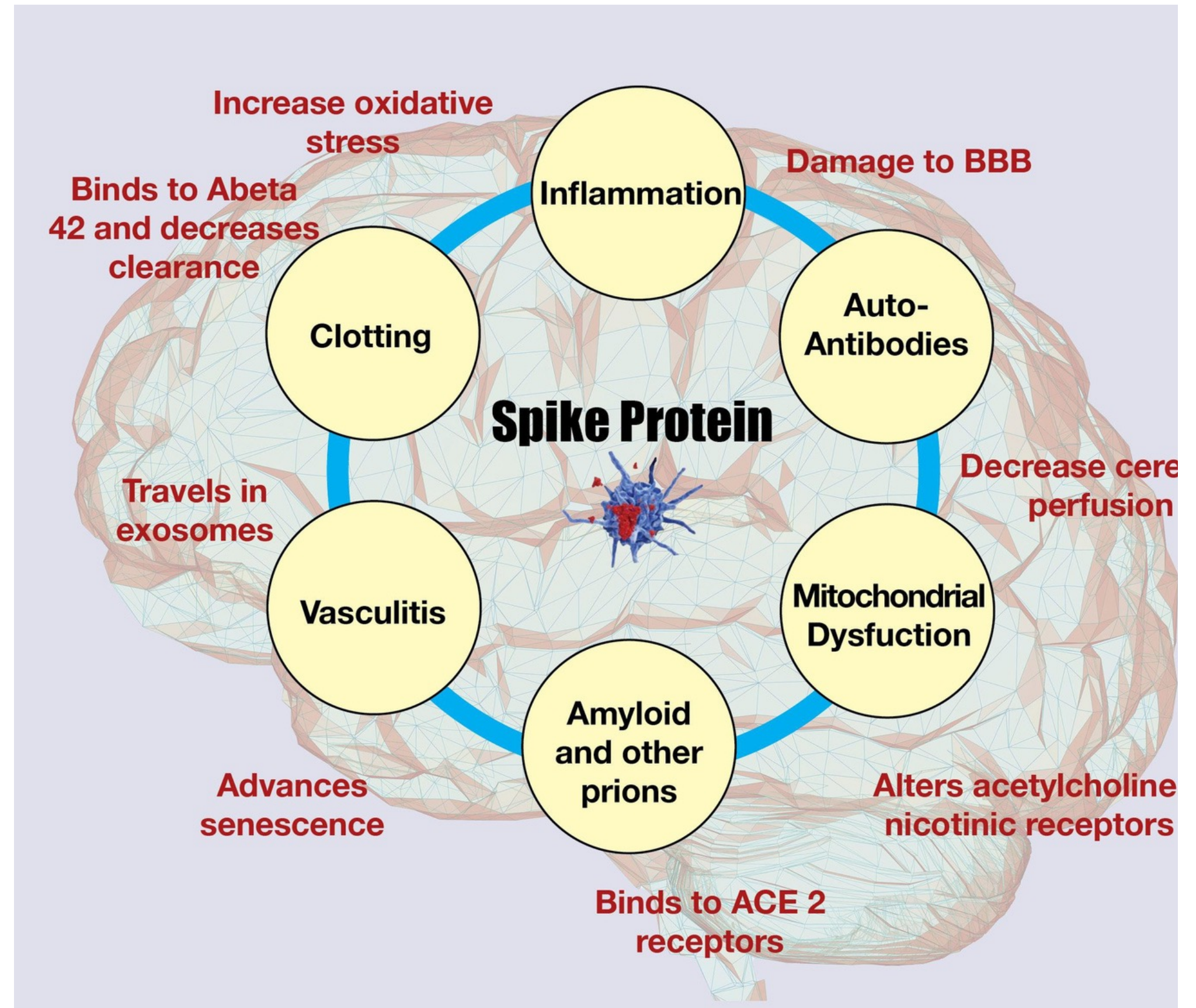
- A February 2026 Alzheimer's & Dementia study reported Long COVID patients had 10% larger choroid plexus volume, correlating with pTau217 and glial fibrillary acidic protein levels tied to AD progression (Pang A 2026)
- Mild/moderate COVID-19 was linked to blood changes in amyloid-beta proteins, akin to APOE4 risk, with greater effects in older or hospitalized patients (Duff E)
- Elevated biomarkers post covid indicate a process that may not stop over time partly due to chronic astrocyte activation and high levels of inflammatory cytokines (Ruchisrisarod C) (Yang X) (Brandestetter A) (Wang X) (Bohhenry D 2024) and more

Spike (S) protein persistence or immune response to viral COVID infection or vaccination has emerged as a key factor in PASC and PACVS

- Spike protein detected in blood plasma up to 709 days after vaccination (Bhattacharjee et al./Yale LISTEN cohort)
- S1 has been found in cerebral arteries (Ota A), in monocytes (Patterson B), in brain, heart, and vasculature (Morz), in meninges and frontal cortex (Rong Z) thrombi (DeMichelle M), skin (Yamanoto M) heart (Yonger L) serum (Brognna C)
- S1 recently found on the fetal side of placenta (Bartman C 2026)

“Spike Protein” or immune response to viral COVID infection or vaccination is Fueling Neurodegeneration

- Spike protein promotes the formation of amyloid and other prions, drives ROS, damages mitochondria and the BBB, fuels neuroinflammation, mast cell activation, fuses neurons and glia, blocks autophagy, inhibits the nicotinic Ach receptors, and more



Accelerated Aging post COVID

- SARS COV 2 can have a significant impact on brain health including accelerated brain aging that in some cases does not return to pre-Covid levels (Mohammad-Nejad AR 2025)(Tang M 2025)(Bruno R 2025)
- SARS-CoV-2 spike protein **or immune response to viral COVID infection or vaccination** triggers cellular senescence in human astrocytes. Senescent astrocytes play critical roles in the development of many severe brain diseases (Hassler W 2025)



(master1305 | iStock)

How One Crisis Worsens Another

- Beckman D et al found a substantial increase in **immune response to viral COVID infection or vaccination** post COVID whenever amyloid plaques are present (primate model) suggesting that SI is fueling prion formation
- Memory centers particularly vulnerable (entorhinal cortex and the hippocampus)
- *“I believe we are going to see, in a few decades, an epidemic of early demented people, especially the ones that had COVID-19 several times. I also believe that older people who accumulate amyloid plaques in normal aging will shift to dementia and Alzheimer's much faster than the normal progression we often observe”.*



Can We Prevent Alzheimer's Disease Within a Decade?

“Alzheimer's should be a rare Disease”

Dr. Dale Bredeesen

A Smoldering Brain

- One of the cardinal features of Alzheimer's disease is the accumulation of extracellular amyloid plaques and neurofibrillary tau tangles. But there are other more complex network of changes, including myelin degeneration, iron dysregulation, volume loss, alteration of the BBB, high levels of neuroinflammation and disrupted communication between neurons and glial cells.
- A study published in the journal Neurology, showed this devastating disease may begin almost 20 years earlier (Rajan K)
- Early intervention during this stage may prevent or prolong the time until symptoms start.

What Causes Alzheimer's Disease? Looking Up Stream

- Advancing age
- Vascular disease
- Inflammation (leaky gut, poor oral hygiene, poor diet)
- Poor sleep
- Lack of exercise
- Infections (Virus', Lyme, P. gingivalis, syphilis)
- Exposure to toxins (glyphosate, air pollution, heavy metals, chemicals, plastic nanoparticles, spike protein or **immune response to viral COVID infection or vaccination**)
- Loss of trophic support (hormones and key nutrients like B12, zinc, Vitamin D)
- Stress
- Trauma
- Cellular deficiencies (limited mitochondrial function, reduced Sirt1)
- Genetics

Toxic Exposure and Risk Of Alzheimer's

- First responders at the WTC have a higher risk of early onset dementia (Clouston A)
- Mounting evidence links a variety of neurological problems to dirty air that is particularly exacerbated in urban cities throughout the world. (Pozzer A) (Kim B) (Deng Y)
- Older women living in highly polluted areas have double the risk of dementia (Kim B) (Deng Y 2026)
- Even living in dirty air for one year showed advanced pathology (amyloid and tau) (Deng Y 2026)



Levels of microplastics in human brains may be rapidly rising, study suggests (Nihart A)

- Microplastics are broken down from plastic waste and have polluted the entire planet, from the summit of Mount Everest to the deepest oceans
- A study published in Nature 2025 that found levels of microplastics in the brain are at much higher amounts than in other organs, such as the liver and kidneys
- Those diagnosed with dementia have up to 10 times more microplastics present in their brains than those without the condition



Microplastics are permeating our world. Our brains are no exception.
NUGROHO RIDHO/MOMENT/GETTY IMAGES PLUS

Chemicals in Countless Household Items Harming the Brain

- Some home chemicals specifically affect the brain's oligodendrocytes, a specialized cell type that generates the protective insulation around nerve cells (Cohen E)
- Quaternary ammonium compounds and flame retardants are present in many personal-care products and disinfectants (Du H)
- Parabens often in combination with phenols and phthalates are linked to lower cognitive scores and behavioral issues. These can disrupt normal neural signaling. (Gonzalez-Palacio P)



Common household chemicals pose new threat to brain health

Research shows chemicals in countless household items harm specialized cells in the brain

Once Upon a Time: The Amyloid Hypothesis

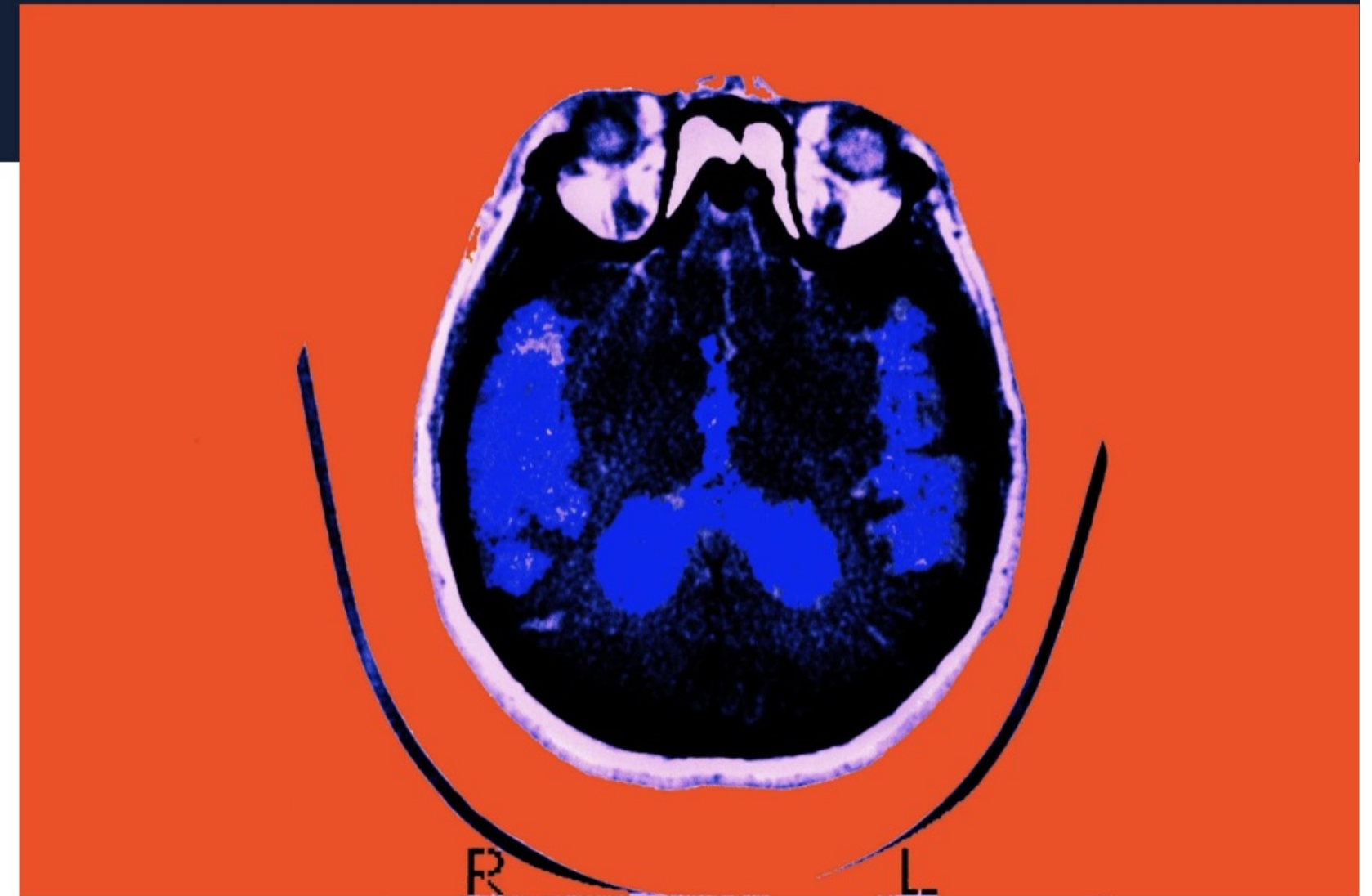
“Alzheimer’s snips away at the threads, a slow unraveling, a steady retreat; as a witness all you can do is watch, cry, and whisper a soft stream of goodbyes.” Patti Davis

The Amyloid Hypothesis

- A paper cited nearly 2500 times, published in 2006 was the “landmark AD research” paper claiming to have found amyloid as the cause of AD
- This theory fueled the race to find the ‘anti-amyloid drug’
- Fabrication of data and image manipulation led to a recent retraction (Science 2022)

Allegations of fabricated research undermine key Alzheimer’s theory

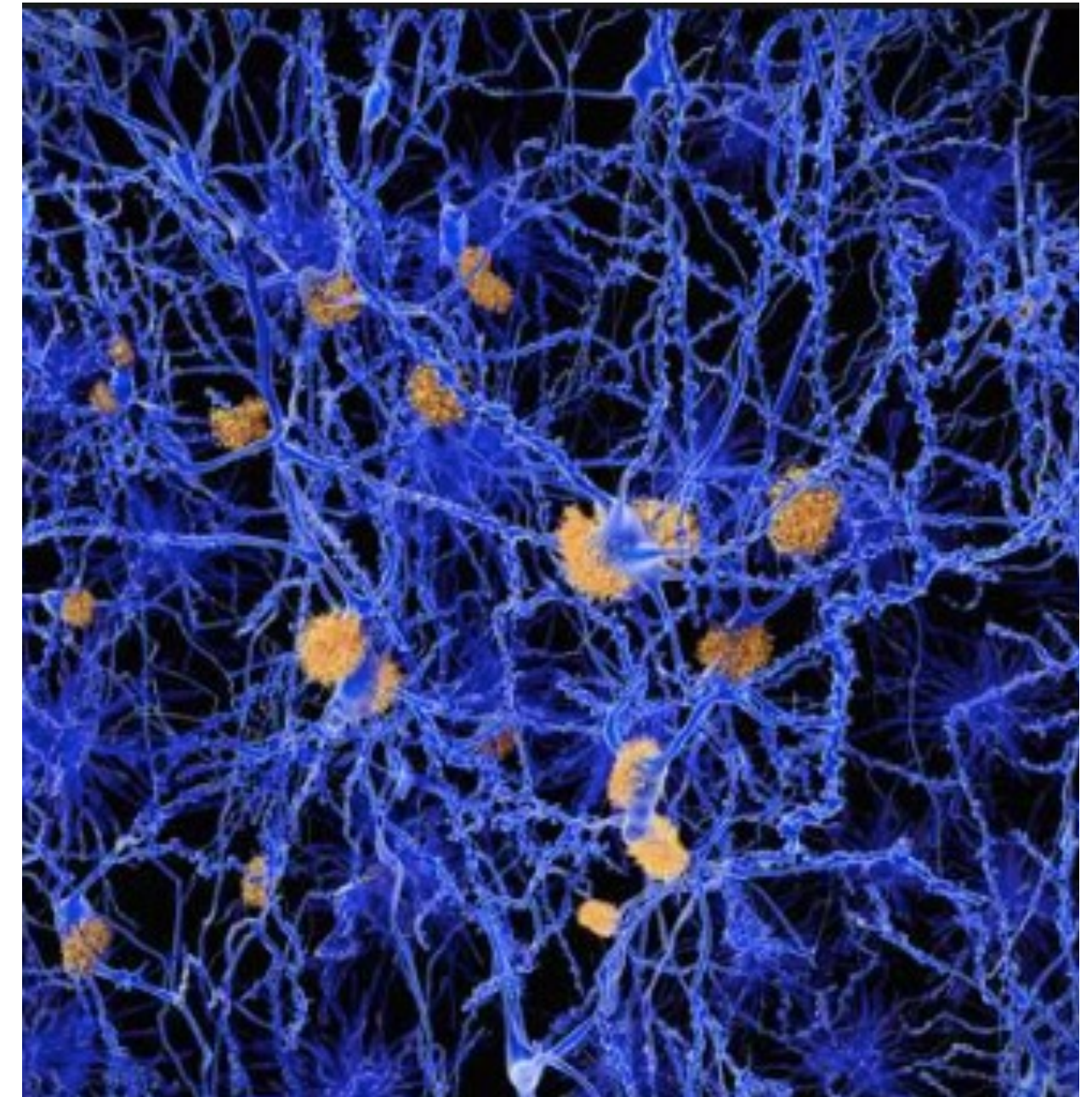
A six-month investigation by Science magazine uncovered evidence that images in the much-cited study, published 16 years ago in the journal Nature, may have been doctored.



— A scan of the brain of a patient affected by Alzheimer's disease in an axial section.
BSIP / UIG via Getty Images

Is Amyloid Friend or Foe?

- Amyloid-beta is a protein fragment that has long been implicated in the pathogenesis of Alzheimer's disease. It is a known neurotoxin that destroys nerve synapses and then clumps into plaques that lead to nerve cell death
- Research suggests amyloid-beta can play a protective role against fungal, bacterial, viral infections and heavy metals.
- Brain amyloid is not synonymous with dementia
- Studies have shown that large amounts of amyloid plaques were present in healthy, non-demented individuals (Mormino EJ)



Heaven Help Us

- In 1986 Dr. David Snowdon, one of the world's leading experts on Alzheimer's disease, embarked on a revolutionary scientific study that would forever change the way we view aging—and ultimately living.
- The “Nun Study” involved 678 Catholic sisters
- A third of the brains tested at autopsy showed signs of Alzheimer's but cognitive tests had revealed that the brain owners had shown no symptoms
- Brain amyloid PET scans of cognitively unimpaired people often look the same as the PET scans of people with AD (Snowden D 2003)

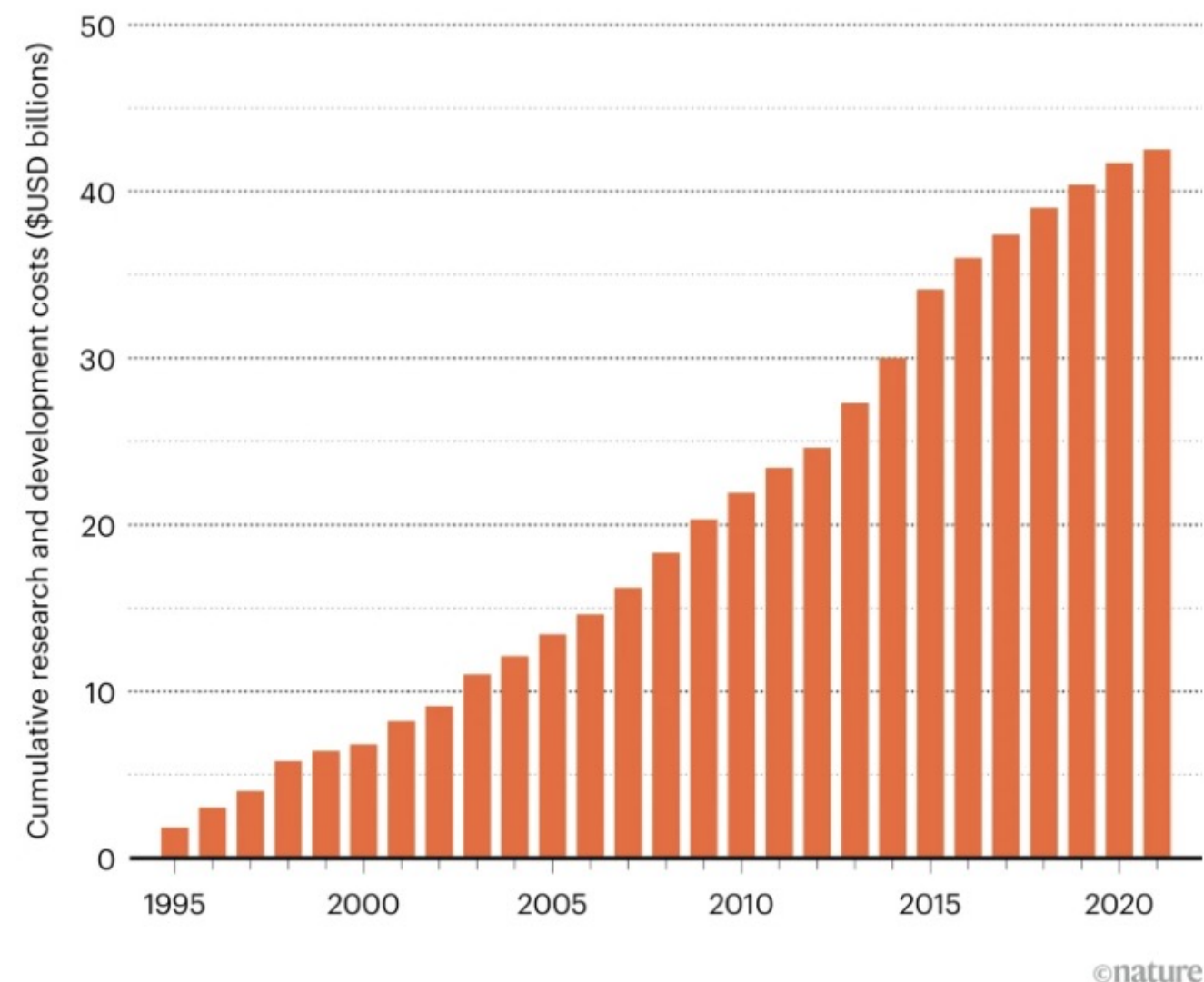


99% of Clinical Trials for Alzheimer's Have Failed in a 1.1 Trillion Dollar Market for Big Pharma

- Alzheimer's disease therapeutics has been, arguably, the field of greatest failure of biomedical therapeutics with 43.5 billion spent since 1995
- Reducing A β peptide production and amyloid formation in the brain did not slow cognitive decline or improve daily life in AD patients
- Over 20 anti-amyloid drugs targeting the A-beta cascade have been tried in clinical trials (Kim C) (Lanhuisa E)

MOUNTING COSTS

Between 1995 and 2021, pharmaceutical companies spent an estimated US\$42.5 billion on Alzheimer's disease research and development, and with few new drugs to show for it. Of the 235 drug candidates that were pursued over this period, 112 remain in development, 6 have reached commercialization and 117 have been discontinued, equating to a 95% failure rate by 2021.



Source: Institute for Health Metrics and Evaluation; Cummings, J. L., Goldman, D. P., Simmons-Stern, N. R., Ponton, E. *Alzheimers Dement.* **18**, 469–477 (2022)

Anti-Amyloid Therapies

- Anti-amyloid therapies, such as lecanemab, donanemab, and gantenerumab, have demonstrated real-world and trial-based efficacy in slowing Alzheimer's disease (AD) progression by 20-35% in early stages, reducing amyloid plaques, and delaying symptom onset, though benefits are modest, stage-dependent, and accompanied by safety risks like ARIA
- The mAbs reduce A β plaques by activating microglia, leading to phagocytosis and degradation of fibrillar A β via the endosomal-lysosomal pathway. Each approved mAb targets distinct A β species (Chundra U 2025)

The Search for the Holy Grail: The Anti-Amyloid Drug

- **Aduhelm:** The FDA approved Aduhelm based solely on how it impacted biomarkers (beta-amyloid plaques), clinical trial results were mixed, but the FDA approved it anyway. Pulled in 2024 by Biogen to work on other products



Lecanemab

- Approved in Jan 2023
- Slowed clinical decline by 27% (only .45 points on an 18-point rating scale) and lowered amyloid on PET
- Dose: IV every 2 weeks. Frequent MRIs needed
- High risks (17.3% had ARIA) 3 deaths
- \$82,500/patient per year with 12.9 billion in sales by 2028
- Long term showed sustained mild-moderate benefit for mild to moderate AD (Van Dyk 2025)

For Alzheimer's patients, a new era of treatment brings hope and risk



By [Laurie McGinley](#)

Updated July 7, 2023 at 4:00 p.m. EDT | Published July 3, 2023 at 6:00 a.m. EDT



Donanemab (Kisunla) approved July 2024

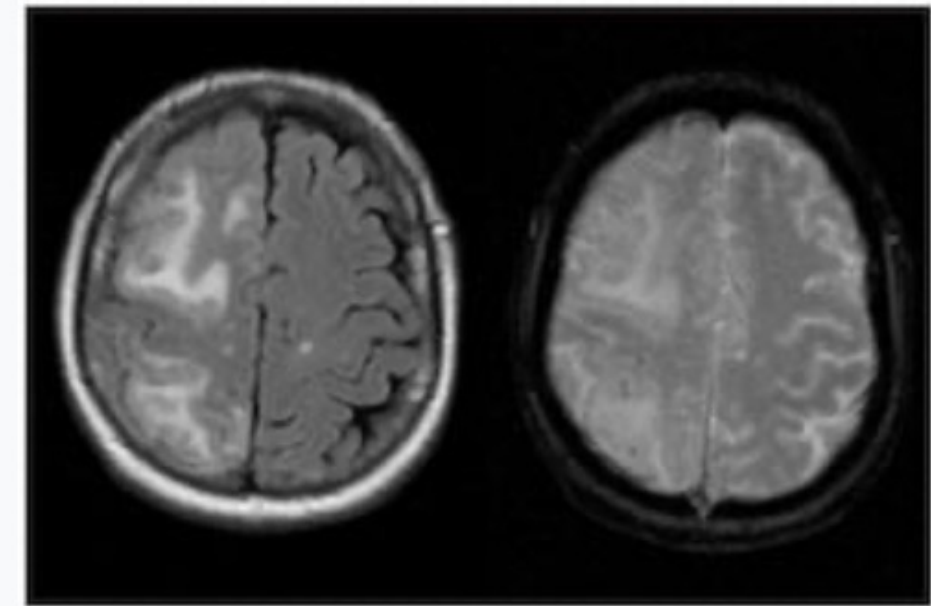
- The drug slowed cognitive decline by 35 percent compared with placebo, and helped people maintain their daily activities. (Sims J)
- In a late-stage study of donanemab published in JAMA, 37 percent of people on the drug experienced some type of ARIA with bleeding or swelling in the brain
- There were 3 deaths
- 17% of people were able to stop treatment after six months, 47% at 12 months and 69% at 18 months.
- Dosed every 4 weeks

Baseline and Frequent MRI monitoring is needed

- Routine safety MRIs: before the 5th, 7th, and 14th infusions (≈ weeks 8, 12, and 26)
- Amyloid-related imaging abnormalities (ARIA) are MRI-detected brain signal changes seen in Alzheimer's patients treated with anti-amyloid monoclonal antibodies (Ramanan V)
- ARIA happens because removing amyloid from the brain—especially from blood vessel walls—temporarily makes those vessels “leaky” and fragile.
- APOE 4 are highest risk
- Most are mild but some can be very severe including death

Amyloid-related imaging abnormalities

Other names ARIA



Two MRI scans demonstrating the difference between ARIA-E (left) and ARIA-H in the **parietal** region (right)

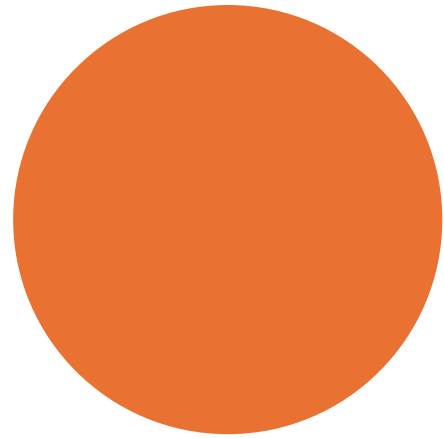
What if You Knew Alzheimer's Was Coming for You?

Simple blood tests may soon be able to deliver alarming news about your cognitive health.

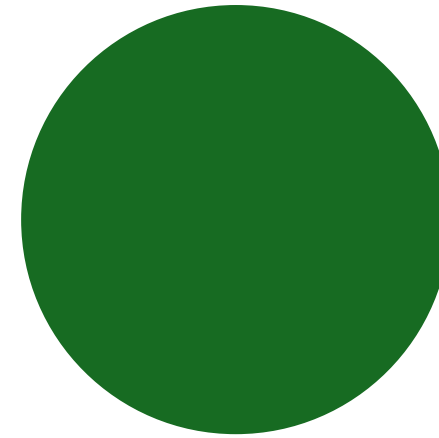
Diagnosing Alzheimer's Disease

- Until recently, the question of whether amyloid plaque—a hallmark of Alzheimer disease—was present in the brains of individuals with cognitive impairment could be settled only after their death.
- PET scans are costly (3000-6000 dollars) and are rarely covered by insurance
- A spinal tap is an invasive procedure
- Blood-based biomarkers are especially promising because they are far less invasive and potentially more accessible than brain imaging or spinal fluid tests

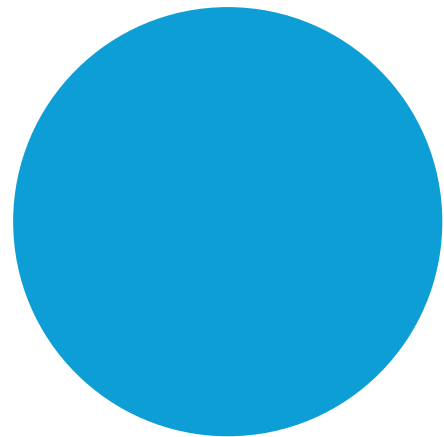
Key Biomarkers



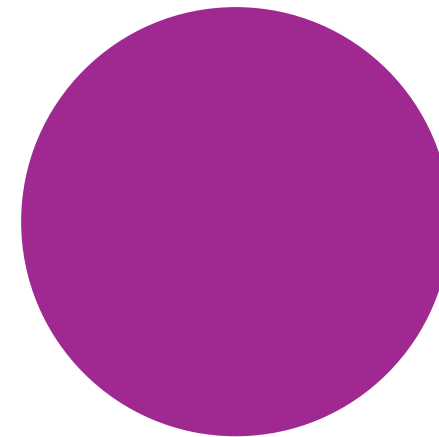
p-Tau 217 is positive in the pre-symptomatic state. It is 95% accurate in predicting amyloid pathology.



NfL (neurofilament light) one of the first biomarkers to be abnormal and suggest neuronal damage from any cause



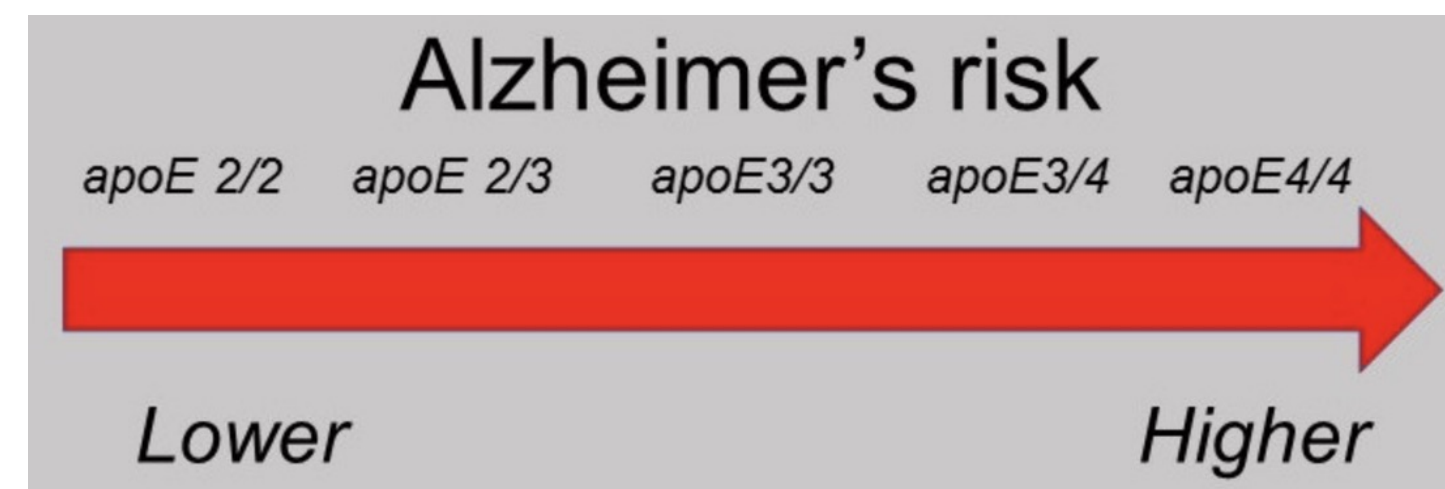
GFAP (glial fibrillary acidic protein) a biomarker of ongoing inflammation and repair in the brain.



It is best to have patients fast from the night before to do all these brain Biomarkers

APOE 4: Harder to Clear Amyloid

- ApoE4 increases the risk by 20% vs those with two copies a 60 percent lifetime risk. (Sienski G)
- ApoE4 may drive harmful immune and vascular-related protein changes that make the brain more vulnerable to AD
- About 25% of people carry one copy of APOE4 & 2 3% carry two copies
- APOE ϵ 4 carrier status is associated with early cognitive decline in cognitively healthy adults, emphasizing the importance of midlife risk awareness and lifestyle interventions (Chung Y 2025)



p-Tau 217

- p-Tau 217 is a valuable biomarker for detecting early-stage AD for it is a specific form of phosphorylated tau that is particularly elevated in the early stages of AD. It is 95% accurate in predicting amyloid pathology.
- Can go up with kidney dysfunction or with weight loss
- When it turns positive, AD is around 2-6 years around the corner
- NeuroCode , Brain Scan , Lab Corp, Quest
- Test every 5 years

Blood Test Predicts Dementia in Women as Many as 25 Years Before Symptoms Begin

- P-Tau217 was strongly linked to **future** dementia risk across decades of follow-up in a large, diverse cohort of U.S. women (65-70 y/o) in the Women's Health Initiative Memory Study (25yr f/u) (Shdyab A 2026)
- All were cognitively unimpaired at the beginning
- Higher risk in APOE 4



UC San Diego study suggests we may be able to identify women at elevated risk for dementia decades before symptoms emerge. Courtesy of iStock/Vanessa Nunes

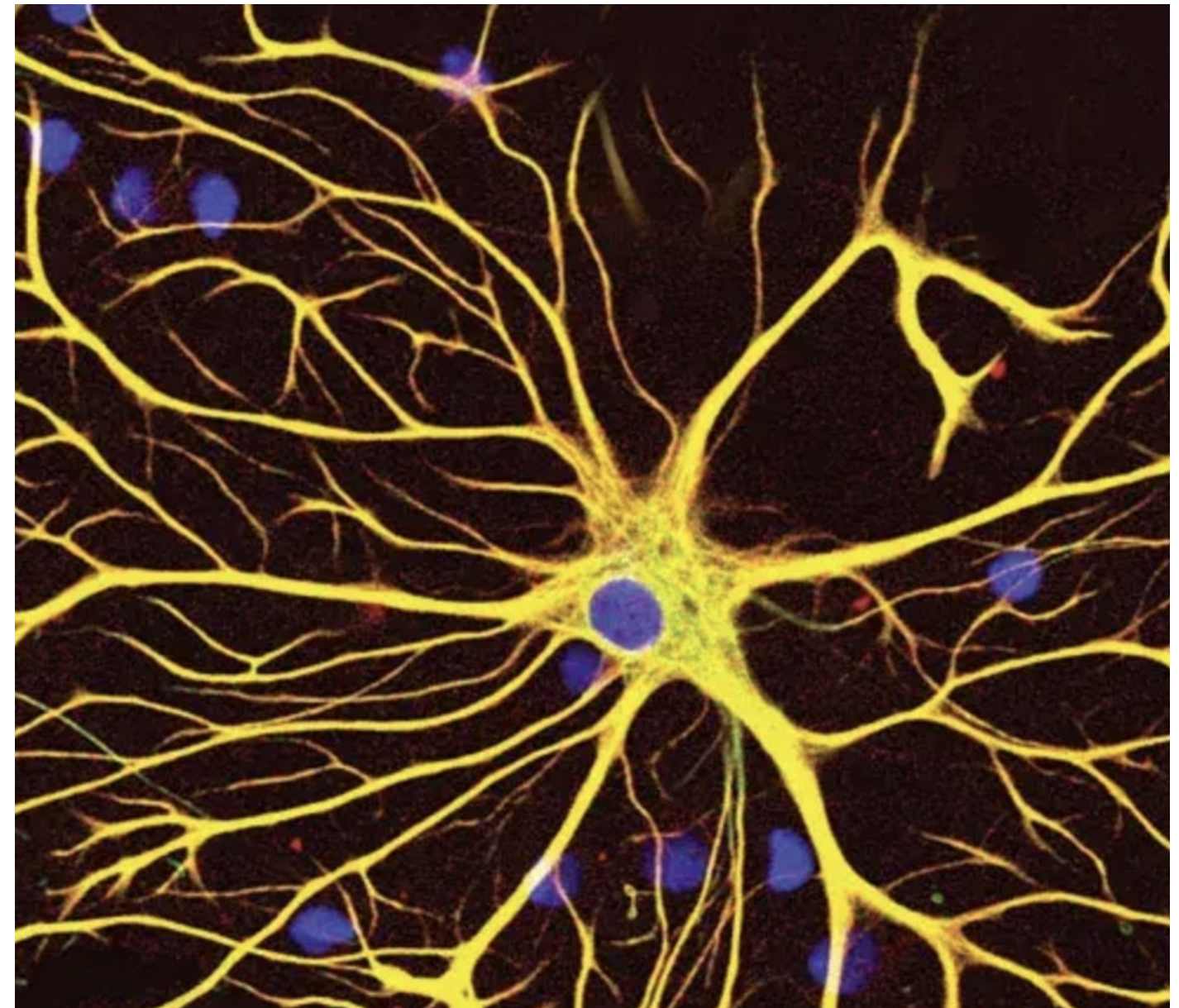
Recent New FDA approved Biomarker to Detect Brain Amyloid Plaques

- **Lumipulse G pTau217/ β -Amyloid 1-42 Plasma Ratio** is indicated for use in adults 50 years or older—who are exhibiting signs and symptoms of Alzheimer disease. (Ashton N)
- This ratio enhances diagnostic accuracy for Alzheimer's disease, improving sensitivity and specificity compared to using either biomarker alone (Rissman RA)
- It is as good as PET and CSF markers
- Never use as a stand alone diagnostic marker



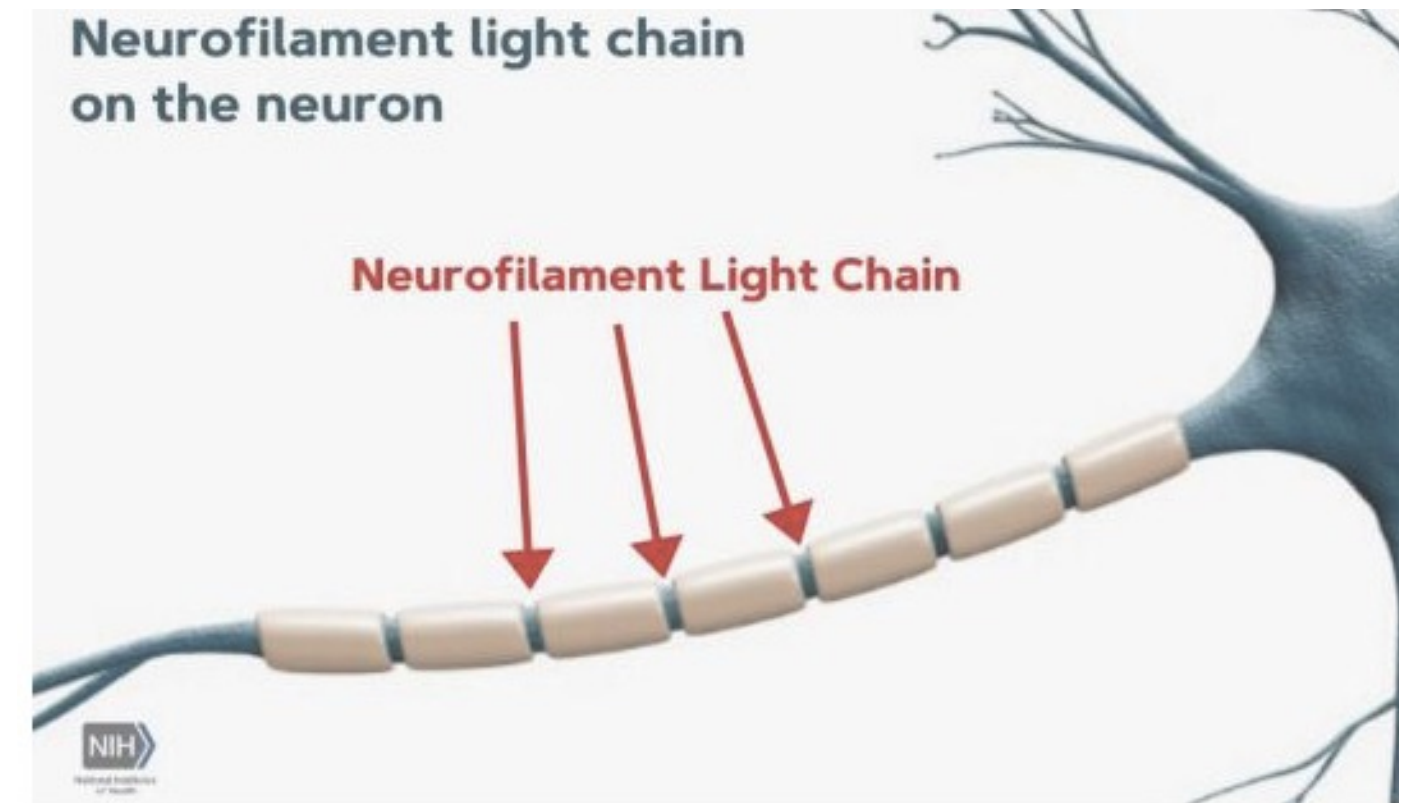
Glial fibrillar acid protein (GFAP)

- GFAP is an intermediate filament protein uniquely found in astrocytes in the CNS and is a biomarker of ongoing inflammation and repair in the brain.
(Wang X)
- More of an all-cause marker
- GFAP has been found to be elevated in CJD and other prion disease
- Elevation of GFAP in repetitive TBI is correlated with volumetric losses in the brain and cognitive changes



Neurofilament Light Chain (nFl) A Marker of Axonal Damage

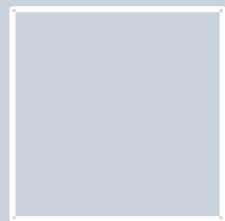
- High plasma NfL levels correlate with poor cognition and brain atrophy, with higher values among subjects with MCI associated with more rapid brain atrophy.
- NfL levels are used to monitor clinical stability in multiple sclerosis.
- NfL breaks away from the axon post TBI and can be a diagnostic marker post concussion (Hussain I)
- Sometimes elevated in Long COVID (Gutman E)



Conclusions



If someone is asymptomatic and has a positive test, this may suggest they will likely develop Alzheimer's disease in the ensuing years



These markers can also be used to monitor clinical response to our treatment protocols



AD biomarkers offer exciting potential for improving diagnosis, monitoring, and research, it's crucial to be mindful of their limitations and use them cautiously and ethically, always in conjunction with a thorough clinical assessments (Lu Y)

Lifestyle Interventions Really Do Work

The FINGER and POINTER Studies

- These studies looked at whether changes in diet, exercise, social engagement, and health monitoring reduced the risk of cognitive decline in older adults
- In the POINTER a self guided and a structured group focused on lifestyle interventions (1) physical activity/exercise; (2) nutrition; (3) cognitive and social engagement; and (4) knowing your numbers (blood pressure, blood sugar, weight)
- Both groups improved but the structured group did much better with slowing the cognitive aging clock by one to two years. (Ornish D 2024)

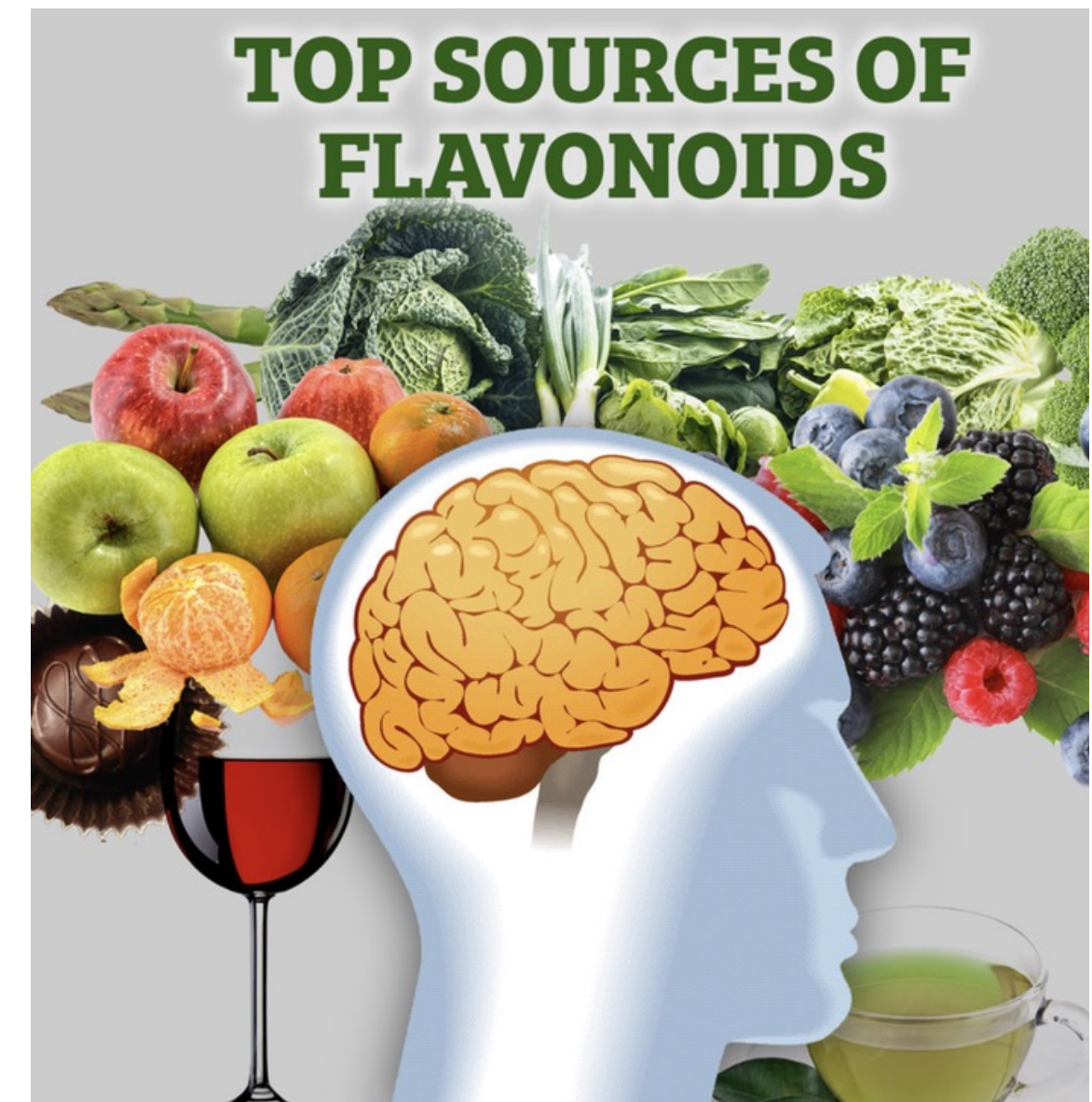
The Bredesen Protocol

- Alzheimer's is the result of a defense mechanism when energy is too low, inflammation too high, or if toxins too heavy your brain switches from building connections to protecting itself
- The core philosophy of the protocol is that Alzheimer's is not a single disease but a result of numerous contributing factors
- Lifestyle changes include a keto-flex diet, rigorous exercise, stress management, improved sleep hygiene, and brain training activities, targeted supplements and detoxification.



Food Is Medicine

- Higher consumption of ultra-processed foods was associated with a higher rate of global and executive function decline after a follow-up of eight years. (Goncalvas N)
- Ultra-processed food, is called “thoughtless food” because of its potentially negative effect on our thinking abilities.
- Other studies have concluded higher flavonoid use can reduce the risk of dementia
- The Western Diet has been associated with cognitive decline even if a healthy diet is introduced in adulthood (Hayes A)



The Mediterranean Diet

- The Mediterranean diet emphasizes high intake of vegetables, fruits, whole grains, legumes, fish, nuts, seeds, olive oil, and moderate amounts of dairy, eggs, poultry, and wine, while limiting red meat, processed foods, sweets, and refined grains.
- This eating pattern supports brain health by reducing inflammation, oxidative stress, improving blood sugar control, preserving white matter integrity, enhancing cognitive functions like memory and executive function, and slowing brain shrinkage



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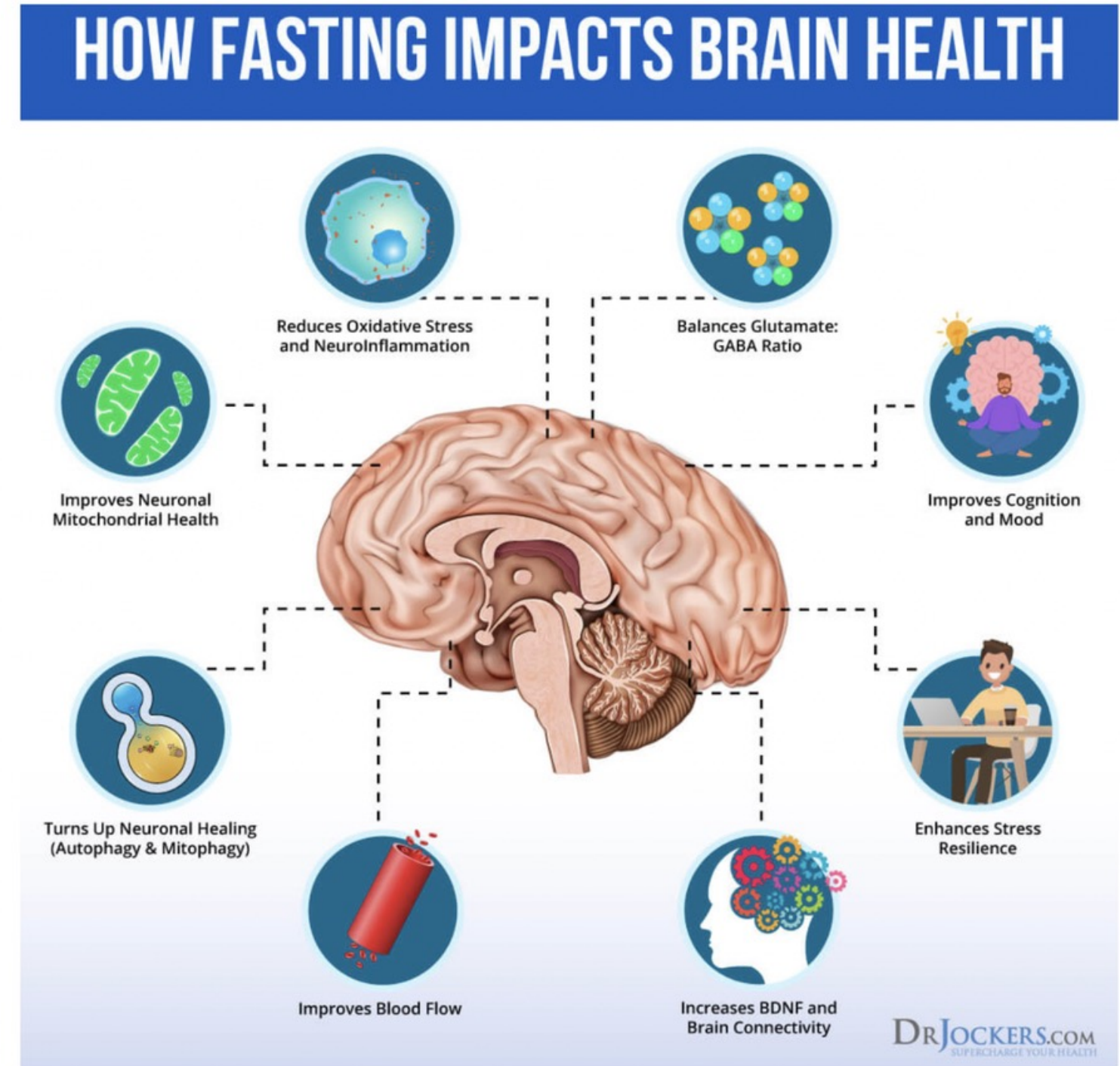
The Mind Diet



- The MIND diet is a brain-healthy eating plan that combines the Mediterranean diet and the DASH (Dietary Approaches to Stop Hypertension) diet, with a specific focus on foods that may protect cognitive function
- Brain autopsies from 600 older adults (avg age 91) found that people who had reported sticking to a Mediterranean or MIND diet showed less evidence of Alzheimer's pathologies, including tau tangles and amyloid plaques. (Agarwe P 2023)

Intermittent Fasting

- IF becomes a lifestyle/most 12 hours (ApoE 4 need 14-16 hours)
- Fasting reduces inflammation, increases BDNF, improves insulin sensitivity, metabolic and vascular health and upregulates autophagy
- In a study, published **Cell Metabolism**, mice that were fed on a time-restricted schedule showed improvements in memory and reduced accumulation of amyloid proteins in the brain. (Whitaker D 2023)



Key Strategies for Optimizing Brain Health

- Diet (Non-GMO/Organic)
Mediterranean or Mind Diet
- Enhance autophagy with
intermittent fasting
- Exercise and resistance training
- Sleep
- Keep homocysteine in check
- Optimize metabolic help
- Gut Health
- Reduce stress (meditation, quality
sleep, counseling, address trauma)
- Engage in brain training and
stimulation
- Limit alcohol
- Reduce toxic load

Sleep Disturbances Forecast Poor Brain Health

- Quality deep sleep is essential for brain health
- Deep sleep helps clear beta-amyloid and other waste products from the brain (Winer J)
- During waking hours, our brains are primed to make or “encode” new memories. But during sleep, our brains switch to consolidation mode.
- Disrupted sleep is connected with dementia and its progression (Mander B)

Exercise Risk Reduction Through Multiple Ways

- Increases blood flow and oxygen to the brain
- Raises BDNF which helps new neurons grow and existing ones survive, particularly in the hippocampus, a key area for memory and learning
- Strengthens connections between neurons
- Supports vascular and metabolic health
- Helps clear amyloid and tau by stimulating the glymphatic system
(Thomas R)

Exercise is the Fountain of Youth for the Brain

- Epidemiological data show that exercise reduces AD risk by 45%, while a sedentary lifestyle increases it by 53%. (Marino F)
- The 2024 Lancet Commission estimates that up to 45% of dementia cases could be prevented by modifying 14 risk factors during different times in the life course, one of which is midlife physical activity (Livingston G)



Get Moving for Optimal Brain Health

- Aim for at least 150 minutes of moderate-intensity aerobic exercise or 75 minutes of vigorous-intensity aerobic exercise per week according to the CDC and Harvard Health.
- Two or more resistance training sessions per week can improve memory, cognition, and reduce brain atrophy, especially for older adults.
- For vigorous training, HIIT, a type of interval training that alternates between short bursts of high intensity movements with lower intensity ones.



Walking Brain Benefits

- Alzheimer's-related decline was delayed by an estimated 3 years on average for those who walked 3,000-5,000 steps per day, and by 7 years in people who walked 5,000-7,500 steps per day
- Just 40 minutes of walking, three times week has been shown to increase the size of the hippocampus , a brain region vital for memory. (Wai-Yeng Wendy Yau 2025)
- Ok to try short 5 min burst if sedentary

Scientists Find That Just 5 Minutes of Brisk Walking Boosts Brain Health— Here's How

Another reason to get out and smell the roses!

BY [KAYLA BLANTON](#) PUBLISHED: JUN 21, 2025



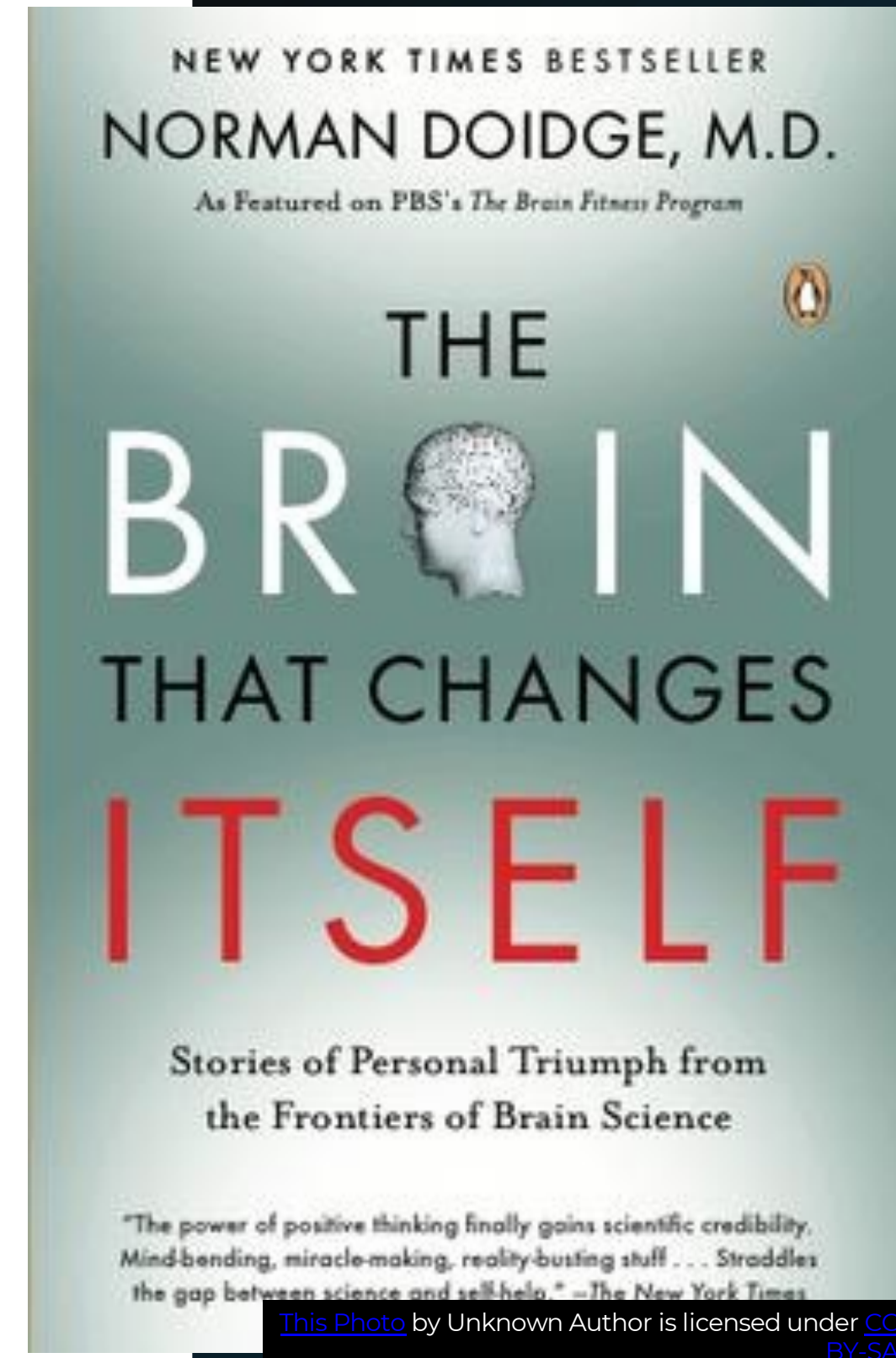
Neuroplasticity is key for healthy brains

- Crossword puzzles alone are not enough to counteract an unhealthy lifestyle.
- There are now more than 300 published studies on BrainHQ—including a new groundbreaking study showing BrainHQ turned back the clock on the biochemical aging of the brain by about 10 years.
- BrainHQ increases acetylcholine (Coe N 2026)
(Attara M 2025)
- Lifelong cognitive enrichment should be all of our goals (Zammit AR)



Neuroplasticity is One of the True Wonders of the Mind

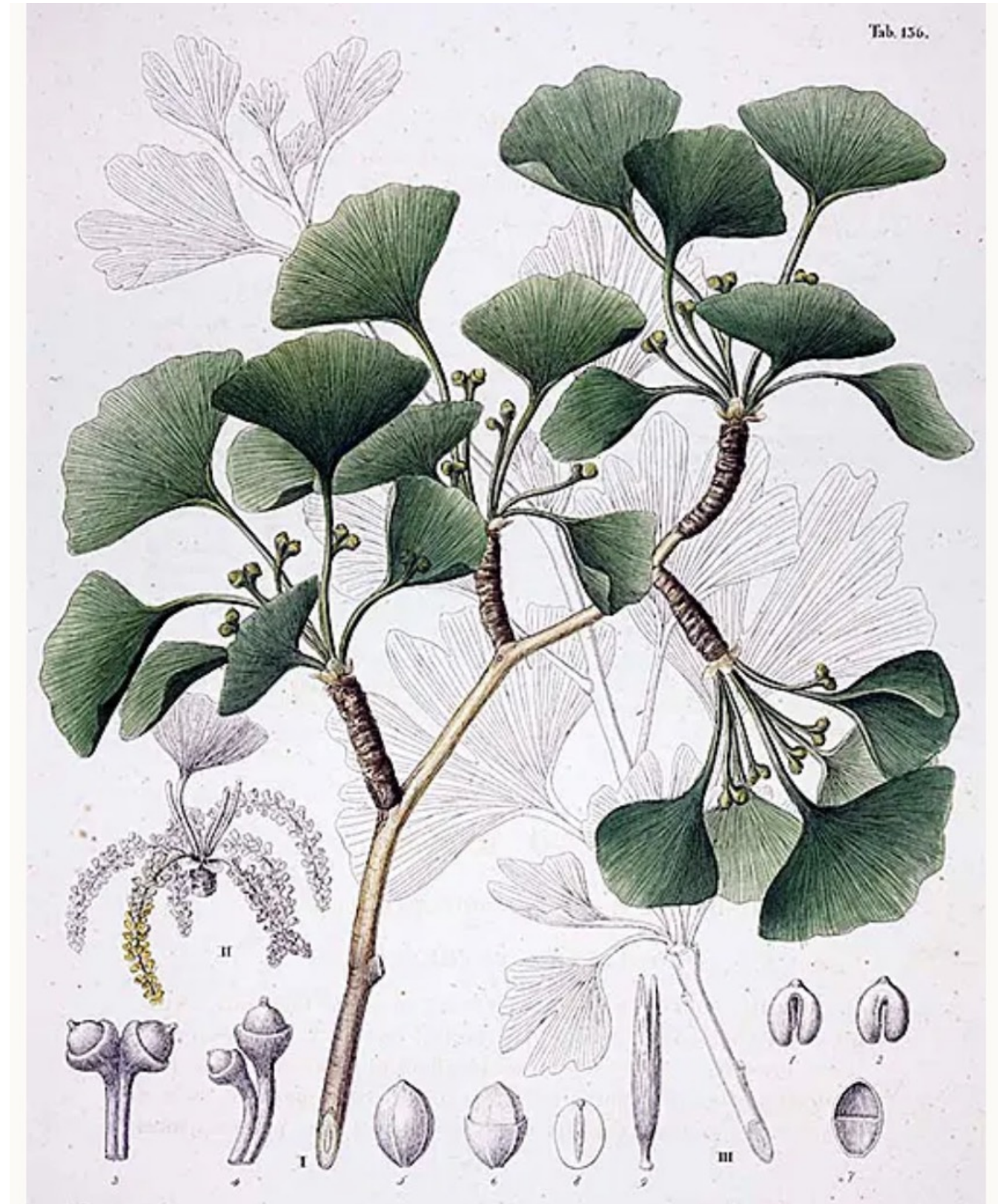
- Learn something new every day
- Watch documentaries that are challenging.
- Ask people questions. Learn about their lives.
- Join a book club
- Listen to music
- Learn a new recipe or a new language ,a dance, learn to paint or draw
- Take a class. Be a life-long learner
- Stay social
- Photo-biomodulation
- Meditation



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The Life Story of The Oldest Tree on Earth

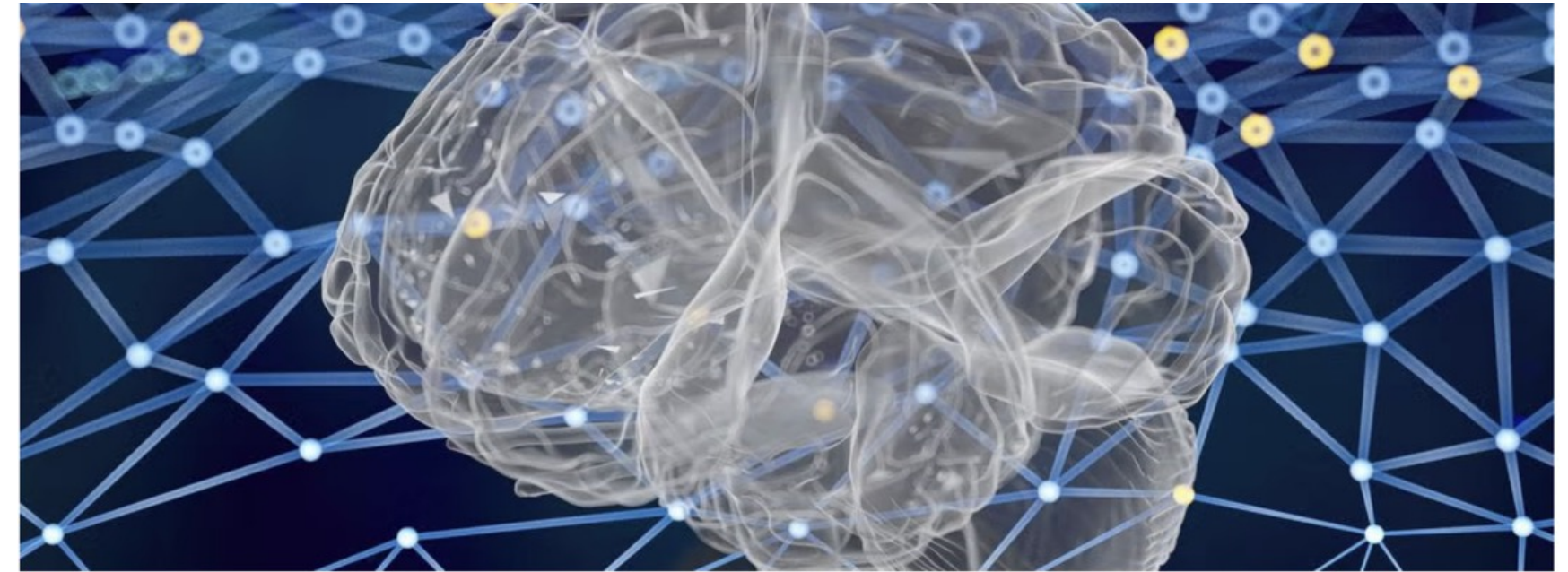
- Ginkgo trees were planted around Buddhist temples and in palace/temple gardens and were valued as a sacred or auspicious tree symbolizing longevity, resilience, and peace.
- **Ginkgo Biloba:** Shown to have antioxidant, antiamyloid properties and increases CBF and acetylcholine release
- When combined with Aricept, reduced amyloid oligomer formation and improved cognitive function was seen (Yang Y 2025)
- May help with ADHD



An early Western botanical illustration of *Ginkgo biloba*, published in Europe in 1835.

Shining Stars

- **P7C3-A20** RX restores nicotinamide adenine dinucleotide (NAD+) homeostasis, reverses tau phosphorylation, blood-brain barrier deterioration, oxidative stress, DNA damage, and neuroinflammation and enhances hippocampal neurogenesis and synaptic plasticity resulting in full cognitive recovery and reduction of plasma levels of the clinical AD biomarker p-tau217(mice) (Chaubey K 2026)
- It works by restoring NAD biosynthesis to enhance neuronal survival and repair



New study shows Alzheimer's disease can be reversed to achieve full neurological recovery—not just prevented or slowed—in animal models

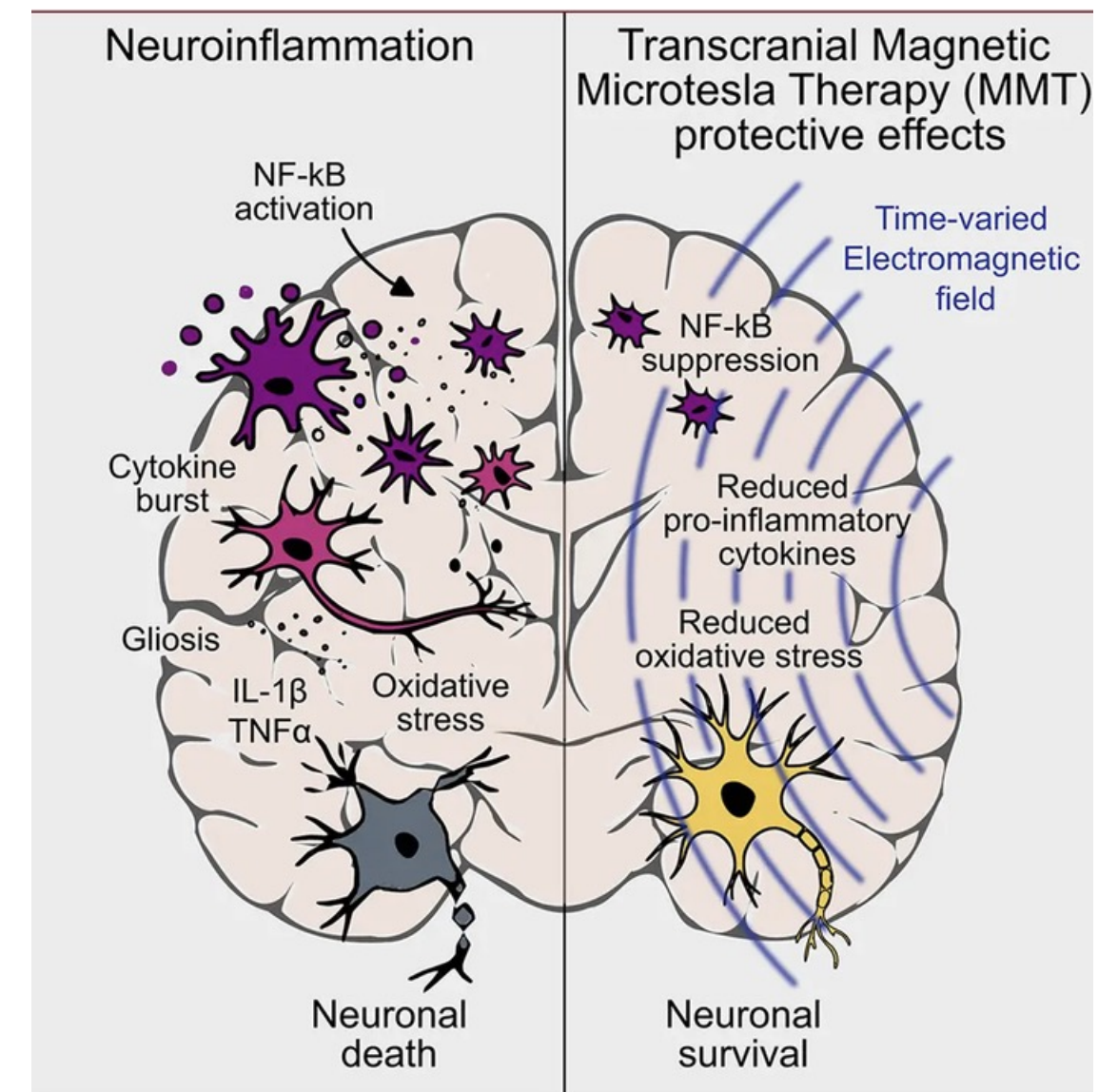
Sildenafil

- Promotes growth of nerve extensions (neurites) and reduces abnormal tau protein & amyloid beta, lowers neuroinflammation and improves blood flow and oxygen supply to the brain.(Fang J)
- Emerging evidence suggests it is neuroprotective (Webb A)
- A 2024 study by Gohel D et al confirmed a ~60% AD risk reduction (2024)



New magnetic therapy could transform treatment of neuroinflammation—a hidden driver of brain disease

- In neurodegenerative diseases like Alzheimer's, Parkinson's, and multiple sclerosis, as well as some psychiatric disorders, chronic or excessive neuroinflammation is a major driver of damage and decline
- MMT **Microtesla Magnetic Therapy**
- Animal data shows sustained and safe suppression of neuroinflammation and neuroprotection (Nguyen N 2026)
- Putrino labs just completed a very positive study in Long Covid cognitive decline (paper submitted for peer review)



Shingles vaccine?

- Reduced dementia risk by about 20% over 7 years (Xie M 2025)
 - Clinical and subclinical reactivations of the neurotropic herpesvirus that causes chickenpox and shingles may constitute a chronic immune stressor that drives inflammatory pathways in both the peripheral and central nervous system, interfering with neuroimmune homeostasis in older age (Eyting M2025)
 - Oral antivirals for herpes have failed trials
- Photo credit Stanford Medicine April 2025



Post Marketing Side Effects of Shingles Shot

- Guillain-Barre Syndrome, Chronic Inflammatory Demyelinating Polyneuropathy
- Vision problems, including blindness, eye infections, and retinal damage
- Stroke
- CHF /MI
- Vasculitis
- Shingles
- Bells Palsy
- Pneumonia
- Death

HOME > A NEW ALZHEIMER'S TREATMENT THAT BRINGS BACK "LOST" MEMORIES

A New Alzheimer's Treatment That Brings Back "Lost" Memories

HEALTH

Phenylbutyrate (PBA)

A molecule with
remarkable
potential

- PBA injection into mice with Alzheimer's disease. proved effective even at an advanced stage of the illness with a striking restoration of memory (Morgan G 2026)
- PBA's mechanism of action focuses on 'proteotoxicity' and acts as a 'chemical chaperone' in the brain, preventing the buildup of misfolded proteins
- Also, PBA inhibits histone deacetylases and may influence gene expression in a way that enhances neuronal function and promotes the removal of toxic proteins.
- It has potent anti-inflammatory effects

Levetiracetam may stop Alzheimer's before it starts but start early

- Northwestern University researchers recently found that levetiracetam prevented the formation of toxic amyloid beta peptides (Rao N 2026)
- High doses may cause harm (e.g., apoptosis in models), so low doses are preferred clinically ie 250 mg / day
- Low-dose levetiracetam (Keppra) may help brain fog by normalizing hippocampal overactivity and improving memory performance (Vossel K)
- Keppra dampens excess firing (“network hyperexcitability”) and lowers neuroinflammation



Plasmalogens

- Plasmalogens support neuronal membrane structure, synaptic transmission, myelination, neurogenesis, and antioxidant defense while inhibiting neuroinflammation, with deficiencies linked to cognitive decline and Alzheimer's disease. (Su X)
- Low plasmalogens may serve as an early biomarker for AD risk; repleting them (via supplements or diet) is proposed as a preventive strategy, potentially before symptoms appear (King M)

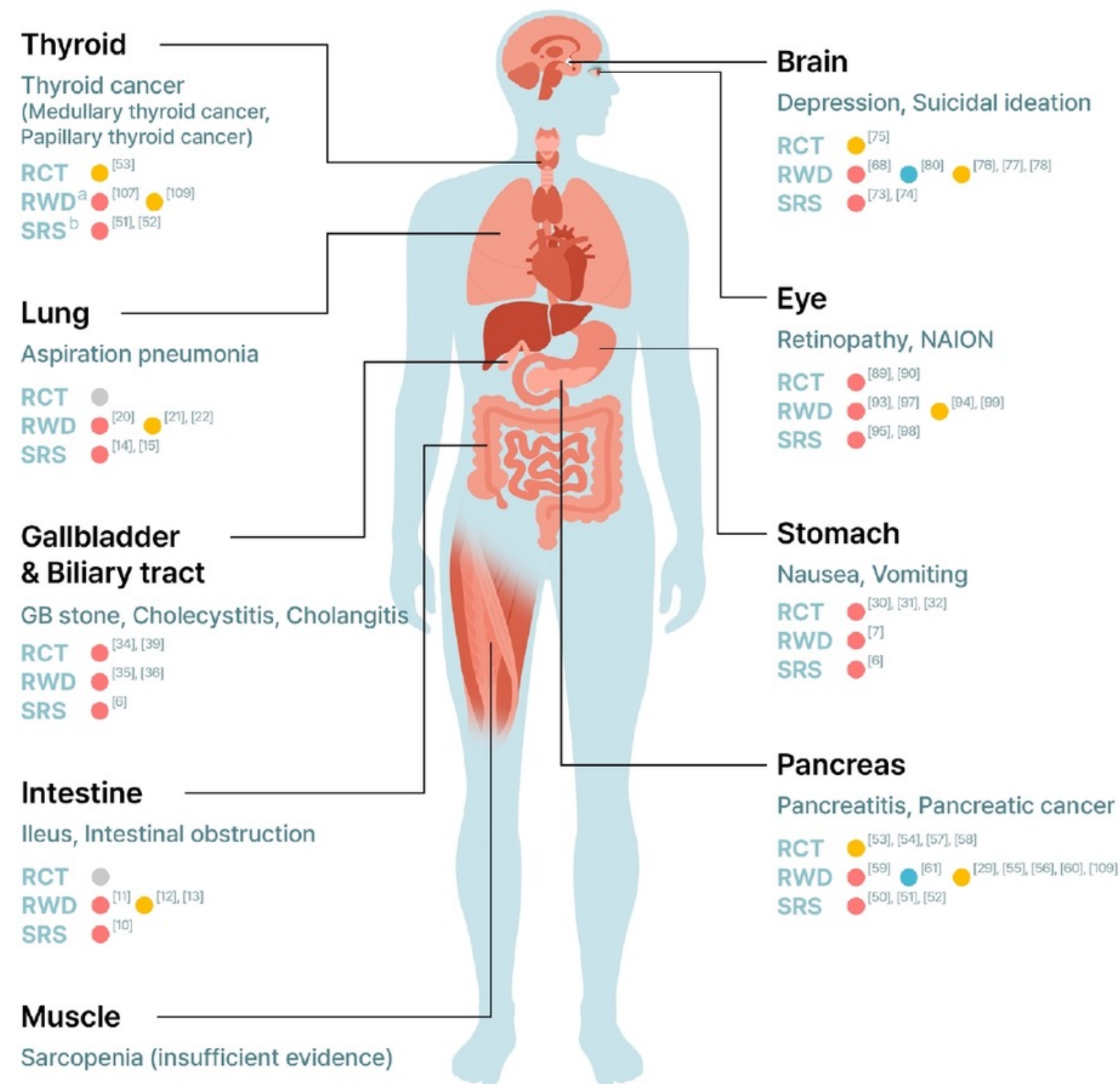


GLP1 Agonist for Dementia

- GLP-1RAs reduce neuroinflammation, amyloid- β and tau aggregation, pro-inflammatory cytokines, and microglial activation while promoting neuronal proliferation & synaptic resilience (Fessel J)
- A large observational study using HER's from over 60,000 adults with type 2 diabetes and obesity found that GLP-1 receptor agonists like semaglutide and tirzepatide were associated with a 37% lower 7-year risk of dementia (Livingston G)
- **Dulaglutide** may have superior brain penetration (61.8%) and showed cognitive benefits in a large randomized trial of 8,828 participants. GLP-1RAs could address dysfunction in all major brain cell types (neurons, astrocytes, microglia, etc.) to potentially cure AD. (Edison P 2026)

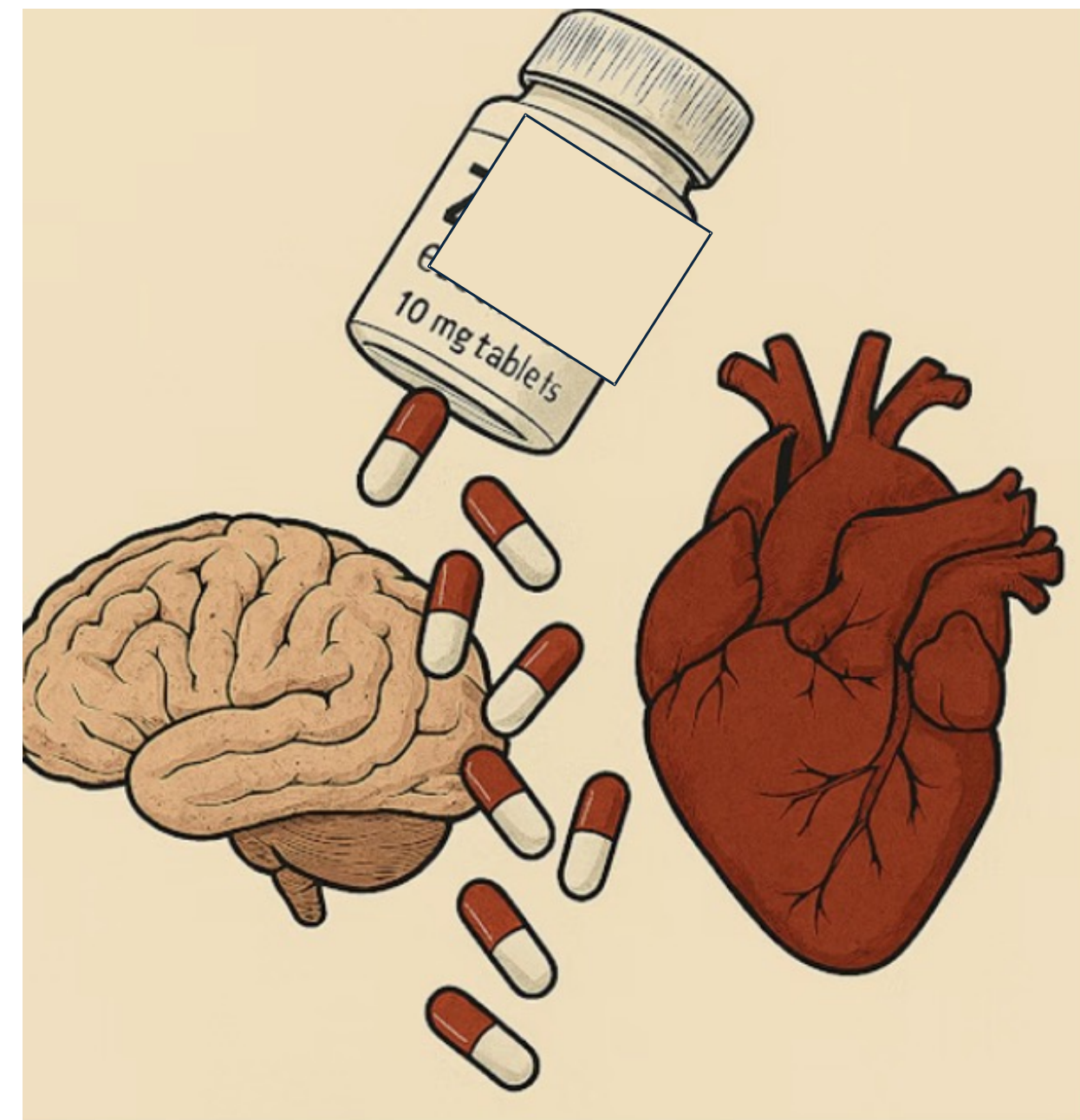
GLP1A Risks

- A study published in Nature showed showed a significant association between GLP-1 RA treatment and an 98% increased risk of any psychiatric disorders. Notably, patients on GLP-1 RAs exhibited a 195% higher risk of major depression, a 108% increased risk for anxiety, and a 106% elevated risk for suicidal behavior. (Cornelius E) (Kim I)



Hiding in Plain Sight: FDA-Approved Cholesterol Drug Ezetimibe as a Treatment for Alzheimer's

- An FDA-approved cholesterol-lowering medication may significantly reduce the risk of Alzheimer's disease and related dementias.(Gorbunova V)
- It could lower this risk by over sevenfold by disrupting harmful protein aggregates in the brain, reducing oxidative stress, and promoting autophagy
- A study by **Ganne et al.** reported a sevenfold reduction in ADRD risk among ezetimibe users (nearly 1 million controls matched for age, gender, and risk factors like hypertension)



Aloe polymannose multnutrient complex (APMC)

- Aloe vera was discovered over 6000 years ago. It was called the Plant of Immortality
- A 12-month open-label pilot study found that patients consuming an APMC supplement daily showed statistically and clinically significant improvements in their cognitive function scores (ADAS-cog) after nine and 12 months.(Martin A)
- Also, rebalancing of the Th1/Th2 immune ratio and significant decreases in certain inflammatory markers
- Increases BDNF



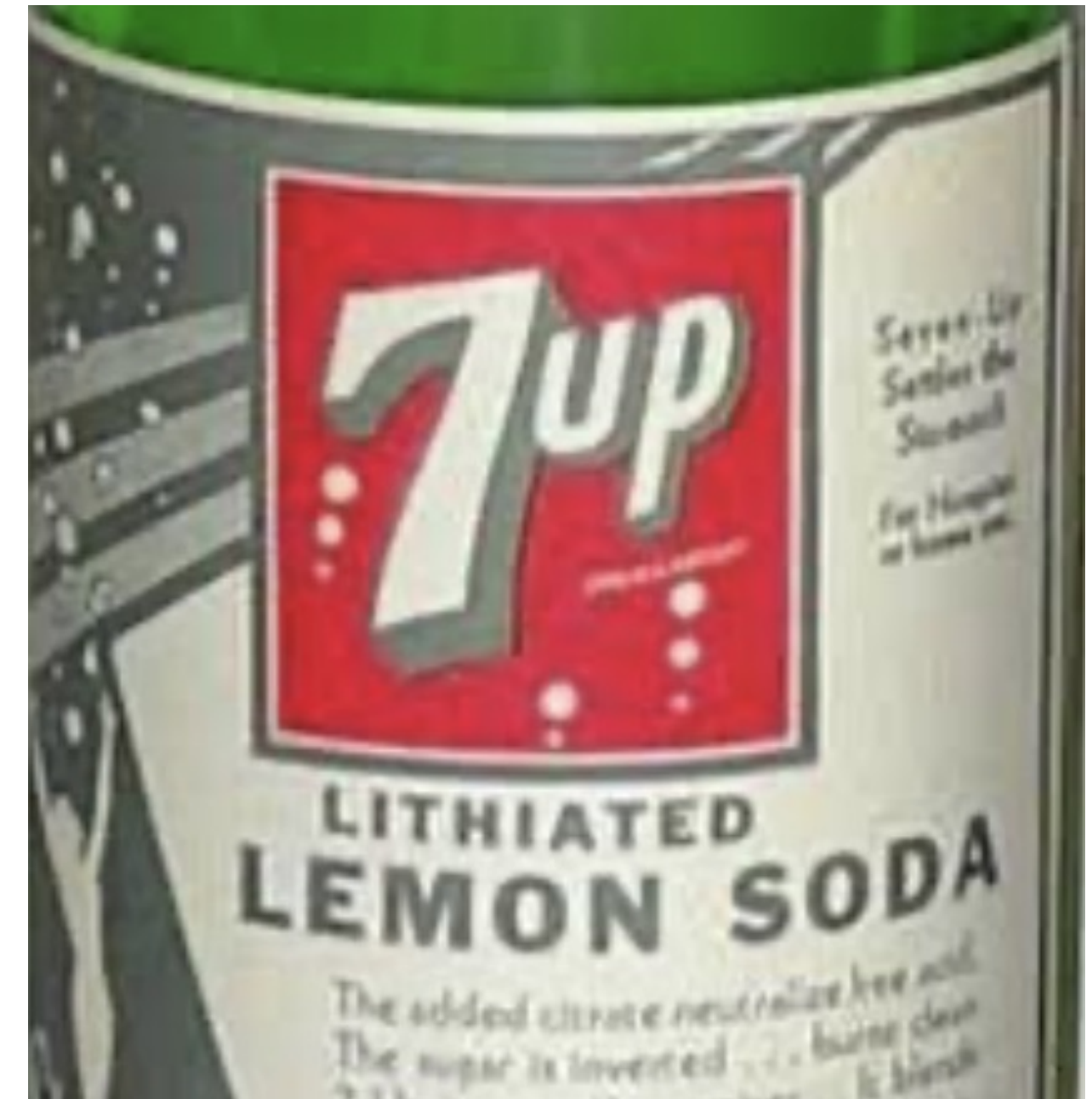
Creatine for Brain Health

- Creatine boosts brain energy metabolism, reduces oxidative stress, supports neuroprotection, and enhances synaptic plasticity
- Impaired brain energy metabolism, including dysfunction in the creatine (Cr)1 system, may contribute to the development and progression of Alzheimer's disease (Xu C)
- A study using 20 g/day of CrM in patients with AD increased brain total creatine by 11%, and was associated with cognitive improvements (Smith N)
- Creatine supplementation improves muscle strength and function and may potentially avert functional disability in older adults (Davis M)



The Wonders of Lithium

- Between the 1880s and World War I, the "Lithia water" craze saw massive popularity for bottled, lithium-rich mineral waters marketed as "nature's remedy" for ailments like gout, kidney, and liver issues
- 7Up originally contained lithium citrate, a mood-stabilizing drug, when launched in 1929 as "Bib-Label Lithiated Lemon-Lime Soda".



The Lithium Trap

- As amyloid- β plaques accumulate, they sequester or “steal” lithium. Because amyloid plaques carry a negative charge, they attract positively charged lithium ions, binding them within plaques while depriving surrounding neurons.
- In mouse models, a lithium-deficient diet reproduced AD-like changes, including plaque formation and cognitive decline



Cracking Alzheimer's Code: How Lithium Shields the Brain

By Aarushi Sahni, Annabelle Lu, Jacen Lopez, Nisha Ganesan, William Dong, Yuva Krishanapillai | September 24, 2025

Lithium Deficiency May Be An Early Driver of Alzheimer's Disease

- A recent meta-analysis from 2024, which reviewed seven observational studies—including case control studies and cohort studies—found that lithium treatment was associated with a substantial decrease in dementia risk. (Lu Q)
- Lithium users had about a 41% lower risk of developing Alzheimer's disease and roughly a 34% lower risk of all-cause dementia compared to those who didn't use lithium. (Lu Q)
- A 12-month trial found that MCI patients taking low-dose lithium maintained better cognitive function than those on placebo (Forienza O)
- Lithium treatment also significantly reduced the Alzheimer's biomarker phosphorylated tau
- Microdose lithium shows promise in stabilizing cognitive decline (Nunez) (Martsunaga S)

Lithium Orotate

- Enhances autophagy, boost mitochondrial function, and plays a role in protecting cells from premature aging by attenuating telomere shortening and influences DNA repair. (Martinson L) (Hamatra S)
- Lithium Orotate 5-30mg/ day
- Orotate doses are microdoses (5–30 mg) vs. prescription carbonate (600–1,800 mg daily with blood monitoring)
- No serious adverse effects reported at low doses



Daily multivitamin slows signs of biological ageing

- The COSMOS randomized clinical trial's ancillary study showed that daily multivitamin-multimineral (MVM) supplementation slowed biological aging as measured by epigenetic clocks, reducing it by approximately 2.7 to 5.1 months over two years compared to placebo, with statistically significant effects on two mortality-predictive clocks (Li S 2026)



Taking multivitamins daily was associated with changes in epigenetic ageing 'clocks'. Credit: Halfpoint Images/Getty

Supplements to Help Optimize Brain health

- **Omega 3** promotes cognition, neuronal preservation, protection against neuro-degeneration & optimizes synaptic plasticity
- **Resveratrol** upregulates autophagy & is neuroprotective effects in the inhibition of β -amyloid production, aggregation and in the destabilization of the A β fibrils and is anti-inflammatory, scavenges free radicals and increases BDNF
- **Li Orotate** is neuroprotective and anti-inflammatory
- **Magnesium:** helps with muscle and nerve function, regulating blood pressure, and supporting the immune system
- **Melatonin:** has antioxidant, anti-inflammatory, and immunomodulatory properties
- **Curcumin:** increases BDNF, reduces oxidative stress and it has anticoagulant, antiplatelet and fibrinolytic properties. Curcumin reduces cellular senescence and has anti-inflammatory and anti-prion effects

Supplements to Enhance Brain Health

- **CDP choline** is a naturally occurring choline source for the synthesis of acetylcholine, enhances the release of dopamine, norepinephrine and serotonin. Choline converts homocysteine into methionine, which has several beneficial effects including helping to lower homocysteine levels (Shea T)
- **Acetyl-carnitine** does cross the BBB and is a powerful anti-oxidant and can aid in directing fatty acids to the cell mitochondria, assisting in the creation of new cell energy. Stimulates Ach receptors in the brain
- **Fisetin** supports the aging brain by inducing autophagy. It was found to be helpful in animal models of PKD, ALS, Huntington's, TBI, CVA and age-related memory loss
- **Flavonoids** boost NGF and BDNF (neurotrophic factors for the brain)
- **Vitamin E:** protects cells from oxidative stress (200mg/d)
- **Vit D** : optimize levels , may help clear amyloid. Deficiency linked to brain volume loss and dementia
- **Omega 3** supports the BBB and is associated with better brain health

Supplements

- **Resveratrol** has neuroprotective effects in the inhibition of β -amyloid production and aggregation and in the destabilization of the $A\beta$ fibrils/ anti-inflammatory, scavenges free radicals. It also upregulates autophagy and increases BDNF
- **Melatonin** reduces oxidative stress, increases BDNF, lowers inflammation, has immunomodulating properties, improves memory primarily through its neuroprotective effects, enhancement of synaptic plasticity, and modulation of glutamate signaling (Sadir N 2025)
- **Magnesium Threonate** blocks the NMDA receptor/ modulates glutamate and is essential for maintaining brain neural plasticity. Mg deficiency has been shown in NDC disease and linked to low brain volumes.
- **NAD+** (nicotinamide adenine dinucleotide) and its precursors like nicotinamide riboside (NR) show promise in preclinical studies for improving cognitive decline by reducing neuroinflammation, protecting mitochondria, and enhancing memory function
- **Urolithin A** enhances cellular health by increasing mitophagy and mitochondrial function and reducing detrimental inflammation

Supplements

- **Urolithin** stimulates this crucial recycling and cleansing process - ultimately protecting cells from age-associated decline. Long-term use significantly improved learning, memory, and olfactory function in different AD transgenic mice & reduced A β and Tau pathology. UA activates autophagy/mitophagy (crucial for recycling and cleansing protecting cells from age associated decline)
- **Nattokinase, Serrapetpase and Lumbrokinase** can dissolve prions
- **Taurine** is critical for normal brain function. It can spur the growth of new brain cells, even in old age and may help protect the brain from neurotoxins that damage the nervous system. Taurine has also been shown to help protect against factors involved in neurological decline and stroke damage. It blocks glutamate
- **Uridine** with Choline and Omega 3 (plays a vital role in the health of neurons and synapses)
- **Vinpocetine** boosts neuroplasticity, blood flow, & reduces neuroinflammation (do not use if on blood thinners or with low BP/ stop 2-3 weeks prior to surgery)

Herbs for Brain Health

- **Lion's Mane** has proven neuroprotective qualities through its ability to stimulate the production of nerve growth factor (NGF)
- **Bacopa** may be neuroprotective thru its role in modulating the NMDA. Numerous studies show benefit with memory
- **Ashwagandha** is anti-inflammatory, blocks A β production, inhibits neural cell death, dendrite extension, neurite outgrowth and restores synaptic function, neural regeneration, reverses mitochondrial dysfunction, improves auditory-verbal working memory, executive function, processing speed etc.
- **Gotu Kola** has been used for centuries to enhance memory and cognitive functioning and to combat the effects of aging in the brain. It's also known for its calming properties.
- **Whole coffee fruit extract (WCFE)** increases BDNF
- **Ginkgo biloba** (neuroprotection) it has antioxidant and anti-amyloid properties

The Power of Music in Dementia : Beatie Wolfe



Time to Act on Dementia, Time to Act on Alzheimers

The World Alzheimer's Month 2024 campaign, Time to act on dementia, Time to act on Alzheimer's, will focus raising awareness and on addressing the stigma and discrimination which still exists around dementia.

IMAGINE A WORLD WITHOUT ALZHEIMER'S

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