

Air pollution and Alzheimer's disease prevention: The science and a prevention plan

Journal of Alzheimer's Disease

2025, Vol. 108(3) 953–968

© The Author(s) 2025



Article reuse guidelines:

sagepub.com/journals-permissions

DOI: 10.1177/1387287251386482

journals.sagepub.com/home/alz



Dharma Singh Khalsa¹ , Helen Lavretsky², Meghan Y Reddy², Annie Fenn³
and Christopher Walling⁴

Abstract

Today, approximately 7.2 million people have Alzheimer's disease in the United States. That number is expected to double by 2060 if a preventive approach is not undertaken. Globally, those figures are also predicted to increase. As we'll soon discover, air pollution and other aspects of climate change are a predominant cause of this remarkable surge. The purpose of this article is to examine the scientific basis of pollution and suggest that an evolutionary lifestyle medicine program may help mitigate and slow down its negative effects on brain health. This review examines existing literature on pollution, and lifestyle as well as other aspects of a lifestyle medicine program, including diet, exercise, stress reduction especially via yoga and meditation, good sleep and essential and spiritual well-being, thus investigating their potential to enhance awareness of the issue and prevent the increase in Alzheimer's disease predicted to arise from air pollution and other forms of climate stress. Air pollution, and the potential for global warming, presents a critical emergency. Scientific evidence indicates that these factors contribute to an elevated risk of Alzheimer's disease, along with other neurological, mental, and physical health challenges. Our hypothesis is that an evidence-based lifestyle medicine program can potentially prevent, slow, or reverse multiple medical problems that are tied to Alzheimer's disease prevention. It is hypothesized that once practitioners of this program become aware, they can reach out to their family, friends, communities, country and globally to attend to the issue with strength, wisdom and clarity.

Keywords

Alzheimer's disease, environmental pollution, essential well-being, exercise, lifestyle medicine, meditation, nutrition, prevention, spiritual fitness, yoga

Received: 30 March 2025; accepted: 26 August 2025

Introduction

An 11-year-old boy tragically lost his life after being struck by a vehicle while crossing the street in Mexico City, one of the most polluted places on earth. At autopsy it was seen that he had multiple brain findings consistent with someone much older and prone to Alzheimer's disease (AD) such as positive neurites and tangles stained for phosphorylated tau.¹

The theory that AD may start many decades before symptoms develop should now be adjusted to include the new reality that AD may, in fact, begin in childhood in the many highly polluted areas of the globe, which essentially puts hundreds of millions more people at increased risk. This article explores how environmental stressors such as pollution, nanoparticles, and climate change impact AD and overall well-being. Secondly, a plan to prevent pathological changes and reduce the risk of AD and other neurodegenerative disorders will be presented.

According to the preface of the *Journal of Alzheimer's Disease* book (IOS Press) on Alzheimer's disease and air pollution, "Our only hope for prevention lies in protecting millions of people exposed to air pollutants and ameliorate their brain effects, we will be on the right path to have a

¹Alzheimer's Research and Prevention Foundation, Tucson, AZ, USA

²Department of Psychiatry & Biobehavioral Sciences, David Geffen School of Medicine at UCLA, and Semel Institute for Neuroscience and Behavior, UCLA, Los Angeles, CA, USA

³Private practice and author, Wilson, WY, USA

⁴Department of Research Psychology, California Institute of Integral Studies, San Francisco, CA, USA

Corresponding author:

Dharma Singh Khalsa, MD, Alzheimer's Research and Prevention Foundation, P.O. Box 30783, Tucson, AZ 85751-0783, USA.

Email: drdharma@alzheimersprevention.or

clean planet free of Alzheimer's disease." Following this plan will also decrease dramatically health care costs at this critical time and perhaps most importantly stop the progression of climate stress.¹

That is our goal and the purpose of this article.

The science

Pathological basis of air pollution and climate change on brain health

Climate change is a pressing global concern, frequently highlighted as a direct threat to the future of human health and society. Average surface temperatures are steadily rising, with predictions of a 2–3°C increase by 2090.² This global warming phenomenon has already been linked to extreme climatic events, including droughts, floods, heat waves, and wildfires, resulting in devastating societal and economic consequences.^{3,4} Despite persistent climate skepticism, the expansive effects of climate change are undeniable. The 2024 annual report from the United States National Oceanic and Atmospheric Administration (NOAA) recounts the toll of extreme weather and climate disasters, indicating that the country has sustained over 400 major weather and climate disasters since 1980, with overall damages exceeding \$2.9 trillion and resulting in thousands of deaths.⁵ Globally impactful events, such as the recent Los Angeles wildfires in January 2025, exemplify this reality, with the Palisades and Eaton fires causing catastrophic losses. Preliminary estimates suggest the destruction of 16,240 properties, translating to capital losses ranging from \$95 to \$164 billion.⁶ Moreover, recent wildfires have unusually occurred in North and South Carolina, Georgia, and Texas.⁷ Beyond the financial toll, the physical, emotional, and mental health burden of the fires on affected communities will likely be profound and enduring. Systematic reviews have extensively documented that the environmental consequences of climate change, such as wildfires, significantly worsen human health outcomes.⁸ Specifically, climate change has been associated with increased mortality, neurological diseases, and various other bodily problems.^{8–10} Over the past three decades, the global incidence of neurological disorders has steadily risen, particularly in regions with higher socioeconomic development.¹¹ As the global population ages and life expectancy continues to increase, neurological disorder diagnoses are expected to rise significantly in the coming decades.¹¹ The underlying mechanisms driving these disorders are diverse, including: (1) proteinopathies, (2) vascular pathologies, and (3) alterations in neurotransmitter levels. Proteinopathies, for instance, involve the accumulation of misfolded proteins in brain regions, such as amyloid- β plaques and neurofibrillary tangles in AD, abnormal

intracytoplasmic α -synuclein protein accumulation in Lewy body dementia and Parkinson's disease, or mHTT aggregation in the striatal region in Huntington's disease.¹² Similarly, vascular pathologies compromise blood flow, leading to localized brain parenchymal injuries, as seen in vascular dementia.¹² Finally, depletion of neurotransmitters such as acetylcholine, dopamine, and serotonin leads to specific symptoms of neurodegenerative diseases, including movement disorders, tremor, rigidity, bradykinesia, memory dysfunction, and cognitive defects.¹³ While these mechanisms are well-studied, mounting evidence suggests that air pollution and climate change may accelerate and intensify these processes.

As described at the outset of this article, air pollution plays a critical role in neurodegenerative disease progression. Oxidative stress, heavy metals, and environmental pollution have been reported to significantly reduce neurotransmitters in the central nervous system, such as acetylcholine and dopamine, increasing the incidence of AD, Parkinson's disease, and vascular dementia.¹³ Common air pollutants such as carbon monoxide, lead, nitrogen oxides, tropospheric ozone, fine particulate matter (PM), and sulfur dioxide often results from vehicle exhaust, the burning of vehicle exhaust, fires, and power plants among other sources (Table 1).

Many of these air pollutants have direct effects on neurological health. More specifically, air pollutants, including lead, ozone, polycyclic aromatic hydrocarbons, and particulate matter in urban environments, can negatively impact neuronal hormones and neurons.¹³ Fine particulate matter PM can induce nervous system injury by crossing the blood-brain barrier, triggering inflammatory responses, and promoting processes such as amyloid- β accumulation, tau protein phosphorylation, and synaptic dysfunctions, which contribute to AD pathology.^{25,26} Fu et al. found a 95% increase in AD risk with higher levels of PM 2.5.²⁷ Building on this Zhang et al. reported that higher PM 2.5 levels from agriculture and wildfires were associated a robust increase in the rates of dementia in the U.S.²⁸ Studies based out of China, the United Kingdom, and Germany have also linked fine PM to cognitive decline.^{29–31} For this reason air pollution specifically has been identified as a modifiable risk factor for AD by the Lancet Commission Report in 2020, and the reduction of exposure to air pollution and second-hand tobacco smoke have been set as specific actions for across the life course.³² Recognizing this danger, governmental agencies, such as the United States Environmental Protection Agency (EPA), had implemented stricter air pollution regulations, aiming to prevent thousands of premature deaths annually.³³ However, these regulations have since been rescinded.³⁴

Through the same mechanisms, and depending on individual susceptibility, microplastics could contribute to the increasing incidence of neurodegenerative diseases. In recent years, plastic pollution has become ubiquitous, and

Table 1. Common air pollutants.

Type	Description	Source	Affected Organ Systems	Average Environmental Levels
Carbon Monoxide ^a	Colorless, nonirritating, odorless, and tasteless gas found in outdoor and indoor air	Motor vehicle exhaust, appliances that burn natural gasoline, cigarette smoke, petroleum refineries, gas and coal burning power plants	Cardiovascular, Respiratory, Neurologic, Developmental	0.12 ppmv in the northern hemisphere and 0.04 ppmv in the southern hemisphere
Lead ^a	Bluish-gray metal found in small amounts in the earth's crust	Burning fossil fuels, mining, manufacturing (production of batteries, ammunition, metal products)	Cancer, Developmental, Hematological, Neurologic, Renal, Reproductive	0.20 µg/m ³ for source oriented and 0.012 µg/m ³ for non-source-oriented in the US between 2008 and 2010
Nitrogen Oxides (NO and NO ₂) ^a	Nonflammable gas that is colorless to brown at room temperature	Motor vehicles exhaust; burning of coal, oil, or natural gas; arc welding, electroplating, engraving, dynamite blasting	Respiratory, Neurological	3338.52 ppb globally in February of 2025 ^b
Tropospheric Ozone ^a	Ground level ozone	Chemical reactions between nitrogen oxides (see above) and volatile organic compounds catalyzed by sunlight	Cardiovascular, Respiratory, Neurologic,	93.4 µg/m ³ per measures in China ^c
Particulate Matter (PM ₁₀ and PM _{2.5}) ^a	PM ₁₀ : inhalable particles with < 10 micrometer diameter PM _{2.5} : fine inhalable particles with < 2.5 micrometers diameter	Direct - construction sites, unpaved roads, fields, smokestacks, fires Indirect - complex reactions of chemicals such as sulfur dioxide and nitrogen oxides, which are pollutants emitted from power plants, industries and automobiles	Cardiovascular, Respiratory, Neurologic,	PM _{2.5} : 1.6 µg/m ³ across 207 cities in the US ^d
Sulfur Dioxide ^a	Colorless gas with a pungent odor	Burning of fossil fuels (coal and oil), copper smelting, volcanic eruptions	Respiratory	11.7 µg/m ³ across 399 cities globally ^e
Other ^a – Acrolein, asbestos, benzene, carbon disulfide, creosote, fuel oils/ kerosene, polycyclic aromatic hydrocarbons, synthetic vitreous fibers, total petroleum hydrocarbons				

^aAdapted from data from the Environmental Protection Agency, Centers for Disease Control, and the Agency for Toxic Substances and Disease Registry^{14,15,16,17,19,21,23}

^bAdapted from Lan et al.¹⁸

^cAdapted from Chen et al.²⁰

^dAdapted from Kioumourtzoglou et al.²²

^eAdapted from O'Brien et al.²⁴

what is more concerning is that such contamination is enduring, taking between 100 to 1000 years to decompose.³⁵ Microplastics are small plastic particles that come from the degradation of plastics and these tiny particles of plastic can be found in the air we breathe, the water we drink, and the food we consume.³⁶ Scientists have even identified microplastics in the atmosphere, where they are exposed to ultraviolet radiation and contribute to the production of toxic greenhouse gases.³⁶ Studies have indicated that increased levels of environmental plastics have been linked to higher levels of micro- and nano-plastics (MNP) found in human tissue samples.^{37,38} Specifically, polyethylene shards and flakes have been identified in the brain tissue of American adults, with a significant increase in the presence of MNPs observed between 2016 and 2024.³⁹ While the data does not prove a causative relationship, there was an association documented between dementia diagnosis and the level of MNP accumulation. Specifically, a high level of deposition was found in the cerebrovascular walls and immune cells.³⁹ Mechanistically, microplastics may induce an inflammatory response and thereby may be at the origin of oxidative stress and neurotoxicity, which is related to neurodegenerative diseases. In animal models, microplastics have been shown to impact neuronal function and behavior, as microplastics are reported to cause inhibition of acetylcholinesterase, oxidative stress with increase in lipid peroxidation levels and an increase in the anaerobic pathway of energy production.⁴⁰ Furthermore, microplastics have been linked to strokes, and Huang et al. suggest that microplastics can induce cerebral thrombosis by causing cells to obstruct capillaries in the brain cortex.⁴¹ While research in the field of MNPs is still in its early stages, these early results highlight the need to further explore the consequences of plastics on brain health.

Finally, when faced with the dire realities of climate change, it is somewhat unsurprising that psychological distress related to climate change has been documented across countries and generations.^{42,43} Qualitative studies reveal concerns that cover themes such as fear of environmental changes, grief over the loss of natural environments, and a sense of despair or hopelessness.⁴⁴ At sufficiently high levels, this distress can manifest as mental health disorders such as depression and anxiety.⁴⁵ Mental health is intricately linked with the progression and prognosis of neurodegenerative diseases. Depression can cause a significant decline in cognition, affecting domains such as memory, executive function, and language performance,⁴⁶ and has been independently linked with increased rates of dementia.⁴⁷ When depression and dementia co-occur, patients experience reduced quality of life, increased chances of early institutionalization, greater health care utilization, and higher mortality rates.⁴⁸ The intricate link between climate-induced psychological distress and neurodegenerative diseases underscores the urgent need for comprehensive strategies to address both environmental and mental health challenges.^{43,49,50}

Current predictions indicate that many of these air pollution and climate change challenges, as well as, their consequent health effects will intensify over time, particularly impacting urban populations in crowded cities with large manmade structures.⁵¹ Addressing climate change through policy and individual-level interventions is crucial. Global solutions to reduce emissions and pollutants are crucial, but adopting brain-healthy lifestyles—through diet, supplements, exercise, sleep, and stress reduction—can help mitigate and even reverse these effects.⁵² This article will delve into these mechanisms and interventions, highlighting the connection between environmental factors and brain health.

The evidence-based plan

As described above, research shows that pollution and climate stress damage brain function, increasing the risk of cognitive decline, anxiety, depression, and cardiovascular, cerebrovascular, and pulmonary diseases.

This review offers an evidence-based plan to prevent and reverse these medical conditions, including proper diet, supplements, mental and physical exercise, stress management through yoga and meditation, and spiritual fitness. This concept of linking neurodegeneration and AD to environmental pollution and climate stress, alongside a preventive and healing plan, is unprecedented. Obviously since it is hypothetical but backed by other proven brain health programs hopefully future research will pointedly address this situation.

Published in 1997, the book *Brain Longevity* introduced a lifestyle medicine plan to prevent AD through a holistic approach.⁵³ This was markedly enhanced in 2017 with the first publication of the now world-famous FINGER study, from Finland, which has resulted in a multitude of significant positive papers and has been expanded to become World-Wide Fingers.⁵⁴

The participants in FINGER followed a healthy Nordic diet, which included fish, participated in physical and cognitive exercise, enjoyed social activity and managed vascular and metabolic risk factors. According to FINGER's lead investigator, Miia Kivipelto, MD, PhD, "The idea was that several risk factors needed to be targeted at the same time to get an optimal preventive effect." Additionally, new FINGER-based studies will include stress management and meditation (Miia Kivipelto, 2024, personal communication).

Furthermore, FINGER outcomes suggested that after 24 months, global cognition was 25% higher than the control group. It also resulted in a 13–20% reduction in cardiovascular and cerebrovascular disease.⁵⁵ The study also indicated that individuals with more subjective memory complaints might experience greater benefits from the intervention.^{56–58} FINGER also assessed quality of life or essential well-being (EWB), which is a component of Spiritual Fitness, and found it to be enhanced.⁵⁹

In 2024, The Ornish Study, a randomized lifestyle medicine approach, suggested strongly that cognitive decline and AD may be prevented, and in some cases reversed. Ornish differed from FINGER in that it included a vegan diet as well as yoga, and meditation. It was one of the first randomized controlled trials to compare a whole foods plant-based diet with a control diet on early dementia. Amongst other plant-based diet observational studies, this study stands out because it also included strategic supplementation. The intervention participants were given these supplements: omega-3 fatty acids, curcumin, a multivitamin, coenzyme Q10, vitamin C, vitamin B12, magnesium L-threonate, lion's mane mushroom extract, and a probiotic—a total of 14 pills a day.⁶⁰

The study concludes that “comprehensive changes can significantly improve cognition and function after 20 weeks in many patients with mild cognitive impairment or early Alzheimer's disease.”⁶⁰

It demonstrated improvement in cognitive tests, cognitive function, and showed less progression relative to the usual care- control group. Additionally, although there was a significant improvement in the plasma A β _{42/40} ratio, the microbiome, and telomere measurements, no scans were utilized.⁶⁰ FINGER also did not use scans, and their telomere measurements suggested that subjects with the shortest telomeres at the beginning of the study had the most significant improvements in telomere length, thus signifying the importance of a multimodal program in boosting not only brain health but also an improvement in immune function and the possibility of enhanced longevity.

As suggested, research indicates that a lifestyle program may help in preventing neurodegeneration, memory loss, AD, prostate and cardiovascular diseases, stroke, anxiety, depression, diabetes, menopausal symptoms like brain fog, and sleep disorders. It's currently said that “what's good for the heart is good for the brain.”⁶¹ Clearly, it's evident that the brain, mind, body, and spirit are interconnected. What impacts one impacts them all down to the cellular level via second and third messengers such as Cyclic CAMP, Cyclic GMP and hormones including cortisol, testosterone, estrogen, human growth hormone and various peptides.⁶² Clearly health care costs can be reduced by the utilization of a lifestyle medicine program.⁶³

Nutrition

Brain-protective dietary patterns

There is more than one dietary pattern proven to protect the brain from AD and other neurodegenerative diseases. All the science-based brain-protective dietary patterns, thus far, however, are spin-offs of the Mediterranean diet (MeDi). The MeDi is characterized by a high intake of vegetables, beans and legumes, whole grains, fruit, fish and seafood, and unsaturated fatty acids (mostly in the

form of olive oil), a low intake of dairy products, meat, and processed foods. The MeDi is also a lifestyle that includes enjoying meals with friends and family, moving naturally throughout the day, and socializing often.

Scarmeas et al. (2006) were the first to suggest that a community of people outside the Mediterranean countries could lower their risk of AD by following a MeDi diet.⁶⁴ In this longitudinal study of community-based individuals in New York, adherence to the MeDi diet reduced AD risk in a dose-response effect; the more closely participants follow a MeDi diet, the lower their risk of AD.

PREDIMED

One of the first randomized controlled nutrition intervention trials, the PREDIMED study in 2018, paved the way for looking at brain health through the lens of nutrition.⁶⁵ PREDIMED showed that following a MeDi diet could reduce the risk of cardiovascular events by as much as 30%. Previously, the PREDIMED-NAVARRA trial, a sub-study of the larger PREDIMED trial, showed a protective role in following a MeDi diet on cognition and brain health.⁶⁶ These findings lend strong support to the idea that while the MeDi could enhance longevity and protect from cardiovascular events, it may also prevent or delay age-related cognitive decline.

MIND

Dr Martha Clare Morris, a nutritional epidemiologist from Rush University who had been studying the impact of nutrition on brain health for over 20 years, and her colleagues created the first brain-specific MeDi spin-off. The MIND (Mediterranean-DASH Intervention for Neurodegenerative Delay) is a hybrid of the MeDi and the DASH (Dietary Approaches to Stop Hypertension) diet.⁶⁷ This dietary intervention was applied to 923 participants of the Rush Memory and Aging Project (MAP), ages 58 to 98, and followed for 4.5 years to assess cognitive health and the development of AD.

The MIND diet defines 10 brain-healthy food groups: berries, leafy greens, vegetables, nuts, fish and seafood, poultry, legumes, whole grains, olive oil, and red wine. The MIND diet differs from the MeDi one by carving out a few specific food groups as neuroprotective, namely berries and green leafy vegetables. In addition, the MIND diet is more specific than MeDi regarding which foods to limit or avoid: fast and fried food (up to one serving per week), pastries and sweets (up to five servings per week), butter (under one tablespoon per day), cheese (less than one ounce per week), and red meat (under four three-ounce servings per week).

Upon completion, the MIND diet results were published as two papers in 2015. In the first, those who followed the guidelines most closely reduced their AD risk by 53% after 4.5 years. Even those who ate from the diet just sometimes reduced their risk by 35%. And, in a separate paper, MIND diet eaters actually improved memory and thinking skills over time.⁶⁸ This was an astoundingly positive finding for an AD prevention study. Researchers determined that close adherence to the MIND diet slows cognitive aging by 7.5 years.

In a postmortem examination of MIND and MeDi eaters, researchers found that following either diet closely had almost 40% reduced risk of having either beta-amyloid or tau pathology.⁶⁹ In fact, close adherence to the MeDi resulted in brains that appeared 18 years younger at autopsy, while MIND diet guidelines yielded brains that looked 12 years younger.

The DIRECT-PLUS trial and green MED diet

The Green MED diet is another brain-specific spin-off of the MeDi, this time by boosting it with polyphenol-rich foods. Polyphenols are a class of bioactive substances in plant foods that perform specific brain health functions, such as reducing oxidative stress, enhancing blood vessel health, and nurturing a healthy gut microbiome.

The Green MED diet strategically includes these foods known to be high in polyphenols in a specific daily dose (number of servings): 3 to 4 cups of green tea, ¼ cup walnuts, and a smoothie made with a protein-rich leafy green. In addition, the Green MED diet emphasizes getting protein from plant sources instead of animal products and it specifically recommends avoiding poultry, red and processed meat.

This polyphenol-boosted MeDi diet was evaluated as part of the Dietary Intervention Randomized Controlled Trial Polyphenols Unprocessed Study (DIRECT-PLUS), a randomized controlled trial of metabolic and brain health comparing a traditional MeDi diet, a Green MED diet, and another healthful diet.⁷⁰ Participants were encouraged to exercise, limit meat consumption (especially processed meat), and were followed with cognitive testing and brain magnetic resonance imaging to assess total brain volume and hippocampal volume over 18 months.

The brains of the MeDi and Green MED diet followers both had significantly reduced brain shrinkage over 18 months. The group who maintained the most brain volume, however, was the Green MED group. Not only did they hold onto more gray and white matter, they had a more robust volume in the hippocampus. Researchers were able to demonstrate a linear positive effect on the hippocampus with each serving of polyphenol rich foods consumed.

The trial of the MIND

In 2015 when Dr Martha Clare Morris published data from the MIND diet study, plans were subsequently made to conduct a randomized controlled trial comparing the MIND diet with another healthful diet. The Trial for the MIND aimed to compare the MIND diet with another diet combined with other lifestyle interventions, tracking a group of participants over three years to assess changes in cognitive health.⁷¹

For this new study, 300 people were asked to follow MIND diet guidelines, but with 250 fewer calories. Another 300 people—the control group—were asked to eat the way they usually do but also with the same reduction in calories. Each group met with a nutritionist at frequent intervals, and the MIND diet arm was also provided with blueberries, mixed nuts, and olive oil. Everyone was asked to exercise as they usually do.

All the study participants had measurable health gains by the end of the study—they all ate healthier, lost weight, and had improvements in brain health parameters. The MIND diet arm of the study started to break away from the control group at year 2 with higher cognitive scores, just as was seen in the FINGER study.⁵⁴ When the global pandemic disrupted the study resulting in a shift to virtual meetings with some participant dropout, researchers saw a general reduction in cognitive scores in both groups. By the study's completion, both groups' cognitive function scores had started to improve but the MIND group had only a slight edge over the control group.

The brain health kitchen food pyramid

The Brain Health Kitchen Food Pyramid, published in *The Brain Health Kitchen: Preventing Alzheimer's Through Food* by Annie Fenn, MD (@Artisan Books 2023) reflects all the science-based brain-protective dietary patterns in one user-friendly graphic. It aligns most closely with MeDi and MIND diet guidelines, while reflecting recent data about the importance of consuming more fiber-rich and fermented foods and fewer servings of animal products. Red wine is dropped as a brain-healthy food group based on recent data that shows moderate drinking is associated with accelerated brain atrophy and there is no safe level of alcohol for the brain (Figure 1).^{72,73}

Instead, polyphenol-rich coffee and tea (especially green tea) make up the tenth brain healthy food group. This Brain Health Kitchen approach is inclusive of many dietary proclivities, including pescatarians, omnivores, vegetarians, vegans, whole-food plant-based, and traditional ethnic dietary patterns. Strategic supplementation is encouraged for those limiting or totally avoiding animal products.⁷⁴

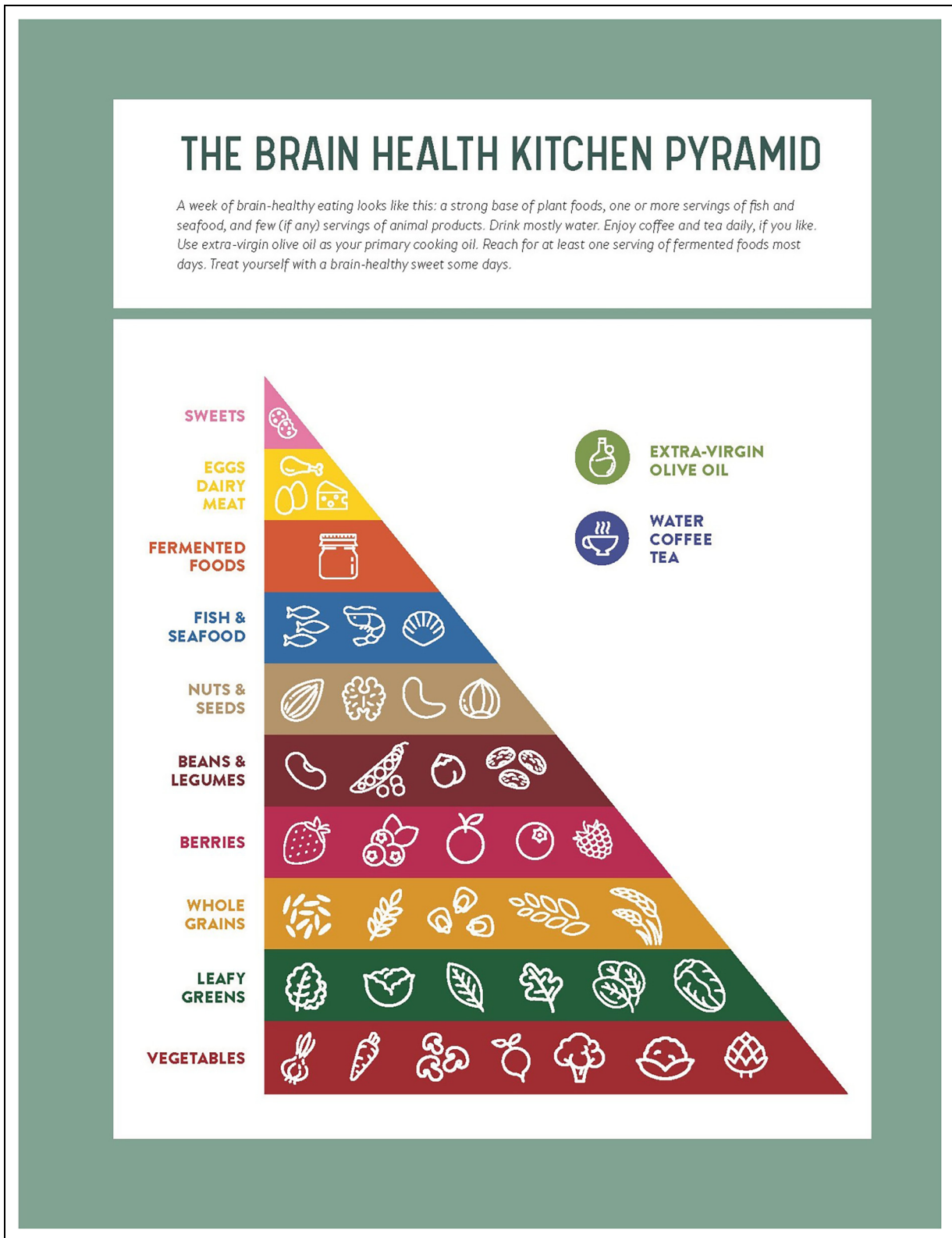


Figure 1. The brain health kitchen pyramid.

Note. Image Excerpted from *The Brain Health Kitchen: Preventing Alzheimer's Through Food* by Annie Fenn, MD. Copyright © 2023 by Annie Fenn. Available from Artisan Books, an imprint of Hachette Book Group, Inc.

Physical and cognitive exercise

It is known that frailty is a risk factor for AD as is reduced grip strength, balance and mobility. Recent studies indicate that a significant proportion of AD cases may be preventable through increased levels of physical activity.⁷⁵ Moreover, people who are more physically active can maintain cognition for a longer time than sedentary people.⁷⁶ In a large meta-analysis, including several cohort studies and 33,816 individuals, higher physical activity levels were associated with a significant reduction in the onset of dementia in a linear dose–response manner.⁷⁷ Additional research has also reported that exercise can delay the transition from mild cognitive impairment (MCI) to dementia.⁷⁷

Exercise was able to significantly improve global cognition and specific cognitive tests in MCI.⁷⁸ In people already affected by dementia, exercise improves global cognition because exercise improves the management of cardiovascular risk factors (e.g., diabetes, hypertension, dyslipidemia, and obesity), which are traditionally associated with poor cognitive performance.⁷⁹ Aerobic exercise is also associated with an increase in brain-derived neurotrophic factor, which stimulates neuronal cell growth and helps maintain neurons in an optimal status.⁸⁰ Moreover, using neuroimaging techniques, additional evidence for the impact of physical activity on brain function and structure has been reported.⁸¹ Exercise is also a good predictor of long-term changes in brain structure, such as brain volume.^{82,83}

For people with dementia, exercise interventions may also reduce the risk of disability, falls, and neuropsychiatric symptoms by approximately 31%. Since falls significantly contribute to disability, physical exercise can reduce fall risk and improve daily activities. Physical activity also reduces the presence and severity of neuropsychiatric symptoms, especially depression.

As seen above, physical exercise is critically important to maintain cognition even in frank AD. According to an article in *Scientific Reports* in 2023, exercising outdoors augments its many positive effects.⁸⁴ Due to rising pollution, it is important to check air quality before exercising outdoors, particularly for those with heart or lung conditions.

Exercise regimens have made great strides recently with the advent of high intensity interval training, the use of treadmills and elliptical machines and strength training regimens. Walking may be the best exercise for many to begin and stay active.

Beyond physical exercise, it is also clear that staying mentally engaged by incorporating brain exercises, or brain aerobics, results in positive brain changes and help in memory maintenance. Physical and mental exercises can be combined through awareness walking, which involves walking outdoors while being consciously aware of your surroundings.

Sleep

Restorative nightly sleep is a first-line defense against neurodegeneration, yet up to 50–70 million Americans live with a chronic sleep disorder such as insomnia or obstructive sleep apnoea.⁸⁵ Roughly half of adults aged 65 years and older report frequent sleep problems, and prevalence exceeds 70 % in some community samples.^{86,87} These already high background rates are aggravated by environmental pollution: each 10 µg/m³ rise in annual PM2.5 is linked to a 6-point reduction in sleep-quality indices and a 13 % higher obstructive sleep apnea incidence⁸⁸; long-term traffic noise above 55 dB elevates odds of insomnia and self-reported “high sleep disturbance” by roughly 25%⁸⁹; and wildfire smoke, nitrogen dioxide, and urban sky-glow shorten slow-wave sleep or fragment sleep architecture.⁹⁰

COVID-19 compounded the problem. Although early lockdowns briefly reduced outdoor traffic emissions, stay-at-home mandates increased time spent in poorly ventilated dwellings, heightened exposure to indoor volatile organic compounds and biomass cooking smoke, and unleashed unprecedented psychosocial stress—collectively producing the global surge in “coronomnia” documented in 2020.⁹¹

Mechanistic studies clarify why pollutant-induced sleep loss matters for dementia prevention. Deep slow-wave sleep facilitates glymphatic clearance of amyloid-β and tau; its erosion accelerates cortical deposition of these neurotoxic proteins.^{93,95} Sleep fragmentation also amplifies pollutant-primed inflammatory cascades: older adults with disturbed sleep exhibit up-regulation of IL-6, TNF-α, and NF-κB signalling.⁹⁴ These same inflammatory pathways are independently linked to cognitive decline and AD pathology.^{32,95,96} Prospective cohort data therefore converge on the conclusion that habitual short or poor-quality sleep doubles the risk of incident dementia.^{92,93}

Pharmacologic responses may inadvertently worsen long-term brain health. Benzodiazepines and Z-drug hypnotics are used nightly by 11.9 % of U.S. women aged ≥ 80 years; chronic exposure nearly doubles AD risk and impairs memory via down-regulation of GABA-A receptors.^{97,98} Before initiating hypnotics, clinicians should first target modifiable environmental drivers—installing HEPA air filtration, minimizing nocturnal noise and light, and addressing evening screen use.

Lifestyle-medicine strategies offer non-pharmacologic leverage. The multimodal program detailed in this review, daylight physical activity, Mediterranean–MIND nutrition, strategic supplementation, and twice-daily Kirtan Kriya meditation—has been shown to normalize sleep architecture or improve insomnia severity.⁹⁹ Implemented together, these interventions can restore sleep, counteract pollutant-triggered inflammation, and thereby reduce downstream dementia risk.

Yoga and meditation: a brief history and modern research

To fully appreciate the concept of yoga and meditation, it is important to know that Dr Walter Hess won the Nobel Prize in 1949 for revealing two spots in the hypothalamus: a stress center and a relaxation center.¹⁰⁰

In 1975, Harvard researcher Herbert Benson, MD, demonstrated that anyone could activate the relaxation center by following 4 steps: a comfortable position, a quiet environment, a mental device, and focused concentration. He called this “the elicitation of relaxation response.”¹⁰¹ Over four decades this simple technique has been shown to affect positively many parts of the body, mind, immune system, and genes.^{102–104} Further research indicates yoga and meditation offer numerous benefits and are part of advanced lifestyle medicine programs.^{60,105}

The medical textbook *The Principles and Practice of Yoga in Health Care* covers multiple AD risk factors such as insomnia, post-traumatic stress disorder, chronic stress, anxiety,¹⁰⁶ depression,¹⁰⁷ cardiovascular disorders, diabetes, and neurological issues,¹⁰⁸ especially improving attention and verbal memory. Yoga may also improve cognitive functioning by enhancing mood, sleep, neural connectivity, and reducing stress,¹⁰⁹ which is a known risk factor for cognitive decline and was inextricably and mistakenly not included in the recent Lancet journal on AD risk.⁶³ Chronic stress is linked to cognitive decline and decreased personal well-being, mainly due to its connection with cortisol. This connection results in toxicity to the hippocampus and limbic system, as well as epigenetic damage. These pathological findings lead to neuronal inflammation, loss or decrease in synaptic transmission, and brain cell death in significant brain areas such as the hippocampus, which are crucial for maintaining high levels of cognition and perhaps preventing cognitive decline and AD. Outcomes include inhibition of memory consolidation, memory retrieval, and working memory all of which may lead to an increased risk for AD.

Like yoga, meditation has been studied in relation to disorders associated with AD risk.^{110–125} Kirtan Kriya, a very easy 12-min singing meditation, which includes specific sounds, as well as prescribed fingertip movements in sequence from the thumb to the little finger, has shown many positive effects. Kirtan Kriya involves singing these specialized sounds for 2 min out loud, 2 min in a whisper, 4 min silently, then 2 min whispering and 2 min out loud. The sounds activate the hypothalamus and pituitary gland, while fingertip touching stimulates the brain’s homunculus, thereby significantly enhancing brain activity and providing optimal protection against neurodegeneration.¹²⁶

Kirtan Kriya has been researched for three decades at leading universities including UCLA, West Virginia University and The University of Pennsylvania, with findings widely published, as mentioned above. This work

suggests many positive benefits on subjective cognitive decline, MCI, and early AD. Studies suggest that long-term Kirtan Kriya practitioners have more gray matter than non-meditators.^{114,118,127} Kirtan Kriya provides similar benefits to a comprehensive lifestyle medicine program with just 12 min of daily practice. According to PROTECT, a long-term study involving 1000 participants, the musical aspect of Kirtan Kriya may also have many positive effects on brain health.¹²⁸ Kirtan Kriya has been associated with improved telomere health, increased amyloid levels in plasma, suggesting lower amyloid in the brain, decreased inflammatory markers, and reversed memory loss.^{115,123}

Multiple studies with Kirtan Kriya utilizing a variety of scans, along with cognitive and mental health evaluations, suggested positive changes in multiple brain areas associated with memory and mood including the dentate gyrus, anterior and posterior cingulate gyrus, hippocampus, amygdala, frontal lobe and pre-frontal cortex.¹²⁹ Synaptic transmission is also enhanced with important neurotransmitters such as acetylcholine, dopamine, serotonin also increasing as well as anandamide, the bliss chemical. This is significant because a decrease in synaptic transmission is a hallmark of AD. Moreover, our research suggests an activation of the brain’s glymphatic system as evidenced by an increase in plasma amyloid concentration.^{120,130,131}

A recent study, *The Pink Brain Project* conducted at UCLA, found that menopausal women with MCI and cardiovascular risk factors also showed increased gray matter, improved mood, and better memory after an 8-week yoga and Kirtan Kriya program compared to a Memory Enhancement Training control group. Kirtan Kriya lowered the age marker exotoxin-1, enhanced brain connectivity, and reduced inflammatory markers, aligning with our prior research.^{132–135}

Spiritual fitness

As previously described by Khalsa, Spiritual Fitness, although once discussed by the US military, is a new concept in medicine combining multiple aspects of personal religious involvement (if there is one), psychological well-being, and spirituality or a search for the sacred.¹³⁶ Having a spiritual connection may reduce AD risk and slow its progression.¹³⁷ Spiritual Fitness has also been shown to reduce risk factors for AD including cardiovascular disease, strokes, and diabetes. It promotes healthy behaviors such as proper diet, exercise, and stress management, and reduces negative behaviors such as smoking, drug use, alcohol abuse, and is associated with enhanced cognitive function with age (Figure 2).^{138,139}

Essential wellbeing

EWB, as enhanced by Kirtan Kriya and other aspects of a lifestyle medicine program, helps create a buffer against

Benefits of 12 Minutes a Day of Kirtan Kriya	
Effects	Outcomes
1. Increased Energetics	1. Less Stress
2. Improved Epigenetics	2. Better Sleep
3. Increased Synaptic Function	3. Less Inflammation
4. Increased Gray Matter Volume	4. Increased Well Being
5. Upregulation of Immune Function	5. Reversal of Memory Loss
6. Reduces Multiple Risk Factors for AD	6. Increased Spiritual Fitness
7. Increased Telomerase/Telomere Length	7. Enhanced Executive Function
8. Down Regulation of Inflammatory Genes	8. Improved Memory in SCD, MCI, early AD
9. Increased Cerebral Blood Flow to Significant Brain Areas	9. Enhanced Mood with Less Anxiety and Depression

Figure 2. Benefits of Kirtan Kriya chart.

illness, inflammation, anxiety, depression, and cognitive decline.^{139,140}

It includes the following:^{141–143}

1. **Acceptance:** You accept life and people as they are. You change what you can and drop what you cannot.
2. **Self-Belief:** The deep knowledge, faith, and expectation that what you do makes a difference in your health and well-being. Self-belief may also extend to actions that are taken to help others and make a positive change in the world, especially when it comes to climate stress.
3. **Independence:** You live free from the influence of others' negative thoughts towards you and your own negative thoughts and look forward to living independently for as long as possible.
4. **Persistent personal growth:** The continued practice of health-promoting behaviors described above and have new experiences that challenge how you think about yourself and the world.¹⁴⁴
5. **Good relationships** with others are essentially being with like-minded people with whom you share love and support. Maintaining healthy family ties along with a strong community is integral to good relationships. Great relations with others involve close connections, intimacy, and unconditional love.¹⁴⁵ Sometimes, however, it may be necessary and important to interact with people outside of your "group" or "comfort zone" to affect a positive change.
6. **Purpose in life:** Experts agree that everyone has a true purpose. Discovering that purpose is shown independently to reduce AD risk.¹⁴⁶ It also lowers the risk of myocardial infarction, stroke, and disability.^{147,148}

Spiritual Fitness integrates EWB with four new non-sectarian elements, promoting enhanced cognitive health and elevated consciousness.

1. **Patience:** Patience simply means waiting and allowing the time to come where your purpose is realized. It also leads to maintaining regular healthy habits such as proper diet, exercise, and stress management.
2. **Awareness:** Awareness fosters an enhanced sense of self-recognition, self-appreciation, and heightened consciousness including a connection to the divine. This results in gratitude and forgiveness, releasing anger, a harmful toxin to your brain and immunity as anger has been associated with AD risk factors including elevated glycosylated hemoglobin, elevated waist-hip ratio, increases of visceral adipose tissue, and lower levels of HDL cholesterol.^{149–151}
3. **Compassion:** Compassion or living with love is a strength and conveys kindness and empathy. Indeed, as suggested by Perez-Bret, compassion originates as an emphatic response to suffering, which nurtures well-being, through specific ethical actions directed to finding a solution to that suffering. Compassion also forms the cornerstone of forgiveness of oneself and others.^{152–155} Compassion is an excellent brain, heart and immune system healer.¹⁵⁶
4. **Surrender:** The stretch of surrender is to your spirit, your soul. During these chaotic and stressful times, many people are searching for peace within themselves.¹³⁶ When you align with your inner self, you can provide support and assistance to others without expecting any personal gain. Moreover, you can strive to make the world a better place. This altruism brings with it a deep and lasting

happiness and serenity, which conveys peace of mind, a much-needed brain health and immune system balm.^{157–160} Progressing on one's spiritual path also evokes an experience of what health psychologist/philosopher Stephen Post, PhD, calls “unlimited love”, which he considers the “ultimate reality.”¹⁴⁴



Environmental wellness: our new future

This article explores the possibility that air and other aspects of environmental pollution and climate stress—resulting from climate change and potentially global warming—could accelerate the onset of AD at a younger age than traditionally believed. Recent policy changes, including the substitution of paper straws with plastic ones and various other actions that counter environmental protection efforts, may pose risks to both environmental integrity and human health. Autopsy studies, in fact, show that levels of microplastic and other nanoparticles in human brains are rapidly rising and beyond that, plastics were found in increasing numbers of the brains of AD patients. This evidence-based lifestyle medicine program promotes health and psychological well-being, creates spiritual fitness, prevents AD, and helps address environmental pollution and climate stress. By committing to global conservation efforts, we can address climate concerns and advance environmental sustainability for present and future generations. We do not have much time to lose.

Acknowledgements

We are grateful to Kirti Khalsa, CEO of Alzheimer's Research and Prevention Foundation for her help in manuscript preparation.

ORCID iDs

Dharma Singh Khalsa  <https://orcid.org/0000-0002-6373-5173>
Christopher Walling  <https://orcid.org/0000-0003-0175-1108>

Author contribution(s)

Dharma Singh Khalsa: Conceptualization; Formal analysis; Project administration; Supervision; Validation; Writing – original draft; Writing – review & editing.

Helen Lavretsky: Formal analysis; Methodology; Supervision; Writing – original draft; Writing – review & editing.

Meghan Y Reddy: Conceptualization; Data curation; Writing – original draft; Writing – review & editing.

Annie Fenn: Conceptualization; Writing – original draft; Writing – review & editing.

Christopher Walling: Conceptualization; Writing – original draft; Writing – review & editing.

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: Kirtan

Kriya Meditation studies and some of the FINGER studies were partially funded by The Alzheimer's Research and Prevention Foundation of which Dr Khalsa is President/Medical Director and receives a stipend. Dr Lavretsky currently receives grant funding from the NCCIH/NIH; NIA/NIH, Dept of Defense.

Declaration of conflicting interests

The authors declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: DSK is an Editorial Board Member of this journal but was not involved in the peer-review process of this article nor had access to any information regarding its peer-review. AF is the author of *The Brain Healthy Kitchen*, from which the pyramid comes. The remaining authors received no financial support for the research, authorship, and/or publication of this article.

References

1. Calderón-Garcidueñas L. *Alzheimer's Disease and Air Pollution: The Development and Progression of a Fatal Disease from Childhood and the Opportunities for Early Prevention*. Amsterdam: IOS Press, 2021.
2. Costello A, Abbas M, Allen A, et al. Managing the health effects of climate change: lancet and university college London institute for global health commission. *Lancet* 2009; 373: 1693–1733.
3. Zheng J, Guo N, Huang Y, et al. High temperature delays and low temperature accelerates evolution of a new protein phenotype. *Nat Commun* 2024; 15: 2495.
4. Rossiello MR and Szema A. Health effects of climate change-induced wildfires and heatwaves. *Cureus* 2019; 11: e4771.
5. NOAA National Centers for Environmental Information (NCEI) U.S. Billion-Dollar Weather and Climate Disasters. <https://www.ncei.noaa.gov/access/billions/> (2025, Accessed March 2, 2025).
6. Li Z and Yu W. Economic impact of the Los Angeles Wildfires. UCLA Anderson Forecast, <https://www.anderson.ucla.edu/about/centers/ucla-anderson-forecast/economic-impact-los-angeles-wildfires> (February 4, 2025, accessed March 2, 2025.).
7. Mellen R and Noll B. Fires are burning throughout U.S. Here's where there's greater risk. The Washington Post. <https://www.washingtonpost.com/weather/2025/03/06/fire-season-texas-carolinas-wildfires/> (March 6, 2025).
8. Rocque RJ, Beaudoin C, Ndjaboue R, et al. Health effects of climate change: an overview of systematic reviews. *BMJ Open* 2021; 11: e046333.
9. Ternes K, Iyengar V, Lavretsky H, et al. Brain health innovation diplomacy: a model binding diverse disciplines to manage the promise and perils of technological innovation. *Int Psychogeriatr* 2020; 32: 955–979.
10. Lavretsky H. Eco-psychiatry: air pollution is associated with depression in older adults. *Am J Geriatr Psychiatry* 2019; 27: 1097–1098.

11. Huang Y, Li Y, Pan H, et al. Global, regional, and national burden of neurological disorders in 204 countries and territories worldwide. *J Glob Health* 2023; 13: 04160.
12. Mehta RI and Schneider JA. Neuropathology of the common forms of dementia. *Clin Geriatr Med* 2023; 39: 91–107.
13. Ayeni EA, Aldossary AM, Ayejoto DA, et al. Neurodegenerative diseases: implications of environmental and climatic influences on neurotransmitters and neuronal hormones activities. *Int J Environ Res Public Health* 2022; 19: 12495.
14. CDC. Air Pollutants. Air Quality. <https://www.cdc.gov/air-quality/pollutants/index.html> (Published April 21, 2024).
15. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Carbon Monoxide*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services, 2012, <https://www.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=1145&tid=253> (accessed July 2, 2025).
16. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Lead*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, 2020, <https://www.atsdr.cdc.gov/toxprofiles/tp13.pdf> (accessed July 2, 2025).
17. CDC. Nitrogen Oxides Toxic Substances | Toxic Substance Portal ATSDR. <https://www.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=69> (February 10, 2021, accessed July 2, 2025).
18. Lan X, Thoning KW and Dlugokencky EJ. Trends in globally-averaged CH₄, N₂O, and SF₆ determined from NOAA Global Monitoring Laboratory measurements. *Version* 2025-06.
19. EPA. Ground-level Ozone Basics US EPA. US EPA. <https://www.epa.gov/ground-level-ozone-pollution/ground-level-ozone-basic> (May 29, 2015, accessed July 2, 2025).
20. Chen C, Li T, Sun Q, et al. Short-term exposure to ozone and cause-specific mortality risks and thresholds in China: evidence from nationally representative data, 2013–2018. *Environ Int* 2023; 171: 107666.
21. United States Environmental Protection Agency. Particulate Matter (PM) Basics US EPA. US EPA. <https://www.epa.gov/pm-pollution/particulate-matter-pm-basics#PM> (November 14, 2018, accessed July 2, 2025).
22. Kioumourtzoglou MA, Schwartz J, James P, et al. PM_{2.5} And mortality in 207 US cities: modification by temperature and city characteristics. *Epidemiology* 2016; 27: 221–227.
23. CDC. Sulfur Dioxide Toxic Substances | Toxic Substance Portal ATSDR. [www.cdc.gov. https://www.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=46](https://www.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=46) (February 10, 2021, accessed July 2, 2025).
24. O'Brien E, Masselot P, Sera F, et al. Short-term association between sulfur dioxide and mortality: a multicountry analysis in 399 cities. *Environ Health Perspect* 2023; 131: 37002.
25. Calderón-Garcidueñas L, Solt AC, Henríquez-Roldán C, et al. Long-term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of the blood-brain barrier, ultrafine particulate deposition, and accumulation of amyloid beta-42 and alpha-synuclein in children and young adults. *Toxicol Pathol* 2008; 36: 289–310.
26. Calderón-Garcidueñas L, Calderón-Garcidueñas A, Torres-Jardón R, et al. Air pollution and your brain: what do you need to know right now. *Prim Health Care Res Dev* 2015; 16: 329–345.
27. Fu P and Yung KKL. Air pollution and Alzheimer's disease: a systematic review and meta-analysis. *J Alzheimers Dis* 2020; 77: 701–714.
28. Zhang B, Weuve J, Langa KM, et al. Comparison of particulate air pollution from different emission sources and incident dementia in the US. *JAMA Intern Med* 2023; 183: 1080–1089.
29. Ranft U, Schikowski T, Sugiri D, et al. Long-term exposure to traffic-related particulate matter impairs cognitive function in the elderly. *Environ Res* 2009; 109: 1004–1011.
30. He F, Tang JJ, Zhang T, et al. Impact of air pollution on cognitive impairment in older people: a cohort study in rural and suburban China. *J Alzheimers Dis* 2020; 77: 1671–1679.
31. Russ TC, Cherrie MPC, Dibben C, et al. Life course air pollution exposure and cognitive decline: modelled historical air pollution data and the lothian birth cohort 1936. *J Alzheimers Dis* 2021; 79: 1063–1074.
32. Livingston G, Huntley J, Sommerlad A, et al. Dementia prevention, intervention, and care: 2020 report of the lancet commission. *Lancet* 2020; 396: 413–446.
33. Furlow B. US EPA strengthens PM_{2.5} air pollution limits. *Lancet Respir Med* 2024; 12: 271.
34. EPA Press Office. EPA Launches Biggest Deregulatory Action in U.S. History US EPA. US EPA. <https://www.epa.gov/newsreleases/epa-launches-biggest-deregulatory-action-us-history> (March 12, 2025, accessed July 2, 2025).
35. Jaishankar M, Tseten T, Anbalagan N, et al. Toxicity, mechanism and health effects of some heavy metals. *Interdiscip Toxicol* 2014; 7: 60–72.
36. Saha SC and Saha G. Effect of microplastics deposition on human lung airways: a review with computational benefits and challenges. *Heliyon* 2024; 10: e24355.
37. Leslie HA, van Velzen MJM, Brandsma SH, et al. Discovery and quantification of plastic particle pollution in human blood. *Environ. Int* 2022; 163: 107199.
38. Persiani E, Cecchetti E, Ceccherini E, et al. Microplastics: a matter of the heart (and vascular system). *Biomedicine* 2023; 11: 264.
39. Babaremu KO, Okoya SA, Hughes E, et al. Sustainable plastic waste management in a circular economy. *Heliyon* 2022; 8: e09984.
40. Barboza LGA, Vieira LR, Branco V, et al. Microplastics cause neurotoxicity, oxidative damage and energy-related changes and interact with the bioaccumulation of mercury in the European seabass, *dicentrarchus labrax* (linnaeus, 1758). *Aquat Toxicol* 2018; 195: 49–57.

41. Nihart AJ, Garcia MA, El Hayek E, et al. Bioaccumulation of microplastics in decedent human brains. *Nat Med* 2025; 31: 1114–1119.
42. Huang H, Hou J, Li M, et al. Microplastics in the bloodstream can induce cerebral thrombosis by causing cell obstruction and lead to neurobehavioral abnormalities. *Sci Adv* 2025; 11: eadr8243.
43. Ballew MT, Uppalapati SS, Myers T, et al. Climate change psychological distress is associated with increased collective climate action in the U.S. *NPJ Clim Action* 2024; 3: 88.
44. Ogunbode C, Doran R, Hanss D, et al. Climate anxiety, wellbeing and pro-environmental action: correlates of negative emotional responses to climate change in 32 countries. *J Environmental Psychology* 2022; 84: 101887.
45. Seethalakshmy A, Kaviya P, Shaji A, et al. A qualitative study on eco-anxiety, climatic change distress, climate resilience, and intentional activities among vulnerable adults. In: Garg M, Kumar D, Samanta D, et al. (eds) *Impact of climate change on social and mental well-being*. London: Elsevier, 2024, pp.137–145.
46. Uppalapati S, Ballew M, Campbell E, et al. The prevalence of climate change psychological distress among American adults. In: *Yale University and George Mason University*. New Haven: CT: Yale Program on Climate Change Communication, 2023. <https://climatecommunication.yale.edu/publications/climate-change-psychological-distress-prevalence/>.
47. O'Shea DM, Fieo RA, Hamilton JL, et al. Examining the association between late-life depressive symptoms, cognitive function, and brain volumes in the context of cognitive reserve. *Int J Geriatr Psychiatry* 2015; 30: 614–622.
48. Brommelhoff JA, Gatz M, Johansson B, et al. Depression as a risk factor or prodromal feature for dementia? Findings in a population-based sample of Swedish twins. *Psychol Aging* 2009; 24: 373–384.
49. Cipriani G, Lucetti C, Carlesi C, et al. Depression and dementia. A review. *Eur Geriatr Med* 2015; 6: 479–486.
50. Leiserowitz A and Lowe S. Yale experts explain climate anxiety. 2023. <https://sustainability.yale.edu/explainers/yale-experts-explain-climate-anxiety>.
51. Yoder K. It's not just you: Everyone is Googling climate anxiety. 2021. <https://grist.org/language/climate-anxiety-google-search-trends/>.
52. Luber G and Prudent N. Climate change and human health. *Trans Am Clin Climatol Assoc* 2009; 120: 113–117.
53. Khalsa DS. *Brain Longevity*. New York: Warner Books, 1997.
54. Kivipelto M, Mangialasche F, Mangialasche F, et al. World-Wide FINGERS network: a global approach to risk reduction and prevention of dementia. *Alzheimers Dement* 2020; 16: 1078–1094.
55. Ngandu T, Ngandu T, Lehtisalo J, et al. A 2 year multidomain intervention of diet, exercise, cognitive training, and vascular risk monitoring versus control to prevent cognitive decline in at-risk elderly people (FINGER): a randomised controlled trial. *Lancet* 2015; 385: 2255–2263.
56. Rosenberg A, Ngandu T, Ngandu T, et al. Multidomain lifestyle intervention benefits a large elderly population at risk for cognitive decline and dementia regardless of baseline characteristics: the FINGER trial. *Alzheimers Dement* 2018; 14: 263–270.
57. Solomon A, Solomon A, Turunen H, et al. Effect of the apolipoprotein E genotype on cognitive change during a multidomain lifestyle intervention: a subgroup analysis of a randomized clinical trial. *JAMA Neurol* 2018; 75: 462–470.
58. Lehtisalo J, Rusanen M, Solomon A, et al. Effect of a multidomain lifestyle intervention on cardiovascular risk in older people: the FINGER trial. *Eur Heart J* 2022; 43: 2054–2061.
59. Lehtisalo J, Ngandu T, Mangialasche F, et al. Health related quality of life in an at-risk population during an intervention study and extended follow-up - the Finnish geriatric intervention study to prevent cognitive impairment and disability (FINGER). *Alzheimers Dement* 2024; 20: e090679.
60. Ornish D, Madison C, Kivipelto M, et al. Effects of intensive lifestyle changes on the progression of mild cognitive impairment or early dementia due to Alzheimer's disease: a randomized, controlled clinical trial. *Alzheimers Res Ther* 2024; 16: 122.
61. Kivipelto M, Ngandu T and Ngandu T. Good for the heart and good for the brain. *Lancet Neurol* 2019; 18: 327–328.
62. Khalsa DS. Stress, meditation, and Alzheimer's disease prevention: where the evidence stands. *J Alzheimers Dis* 2015; 48: 1–12.
63. Livingston G, Huntley J, Liu KY, et al. Dementia prevention, intervention, and care: 2024 report of the lancet standing commission. *Lancet* 2024; 404: 572–628.
64. Scarmeas N, Stern Y, Tang MX, et al. Mediterranean Diet and risk for Alzheimer's disease. *Ann Neurol* 2006; 59: 912–921.
65. Martínez-González MÁ, Ros E and Estruch R. Primary prevention of cardiovascular disease with a Mediterranean diet supplemented with extra-virgin olive oil or nuts. *N Engl J Med* 2018; 378: 1388–1389.
66. Martinez-Lapiscina EH, Clavero P, Toledo E, et al. Mediterranean Diet improves cognition: the PREDIMED-NAVARRA randomised trial. *J Neurol Neurosurg Psychiatry* 2013; 84: 1318–1325.
67. Morris MC, Tangney CC, Wang Y, et al. MIND Diet associated with reduced incidence of Alzheimer's disease. *Alzheimers Dement* 2015; 11: 1007–1014.
68. Morris MC, Tangney CC, Wang Y, et al. MIND Diet slows cognitive decline with aging. *Alzheimers Dement* 2015; 11: 1015–1022.
69. Agarwal P, Leurgans S, Agrawal S, et al. Association of Mediterranean-DASH intervention for neurodegenerative delay and Mediterranean diets with Alzheimer disease pathology. *Neurology* 2023; 100: e2259–e2268.

70. Kaplan A, Zelicha H, Yaskolka Meir A, et al. The effect of a high-polyphenol Mediterranean diet (green-MED) combined with physical activity on age-related brain atrophy: the dietary intervention randomized controlled trial polyphenols unprocessed study (DIRECT PLUS). *Am J Clin Nutr* 2022; 115: 1270–1281.
71. Barnes LL, Dhana K, Liu X, et al. Trial of the MIND diet for prevention of cognitive decline in older persons. *N Engl J Med* 2023; 389: 602–611.
72. Daviet R, Aydogan G, Jagannathan K, et al. Associations between alcohol consumption and gray and white matter volumes in the UK biobank. *Nat Commun* 2022; 13: 1175.
73. Jeon KH, Han K, Jeong S, et al. Changes in alcohol consumption and risk of dementia in a nationwide cohort in South Korea. *JAMA Netw Open* 2023; 6: e2254771.
74. Fenn A. *The Brain Health Kitchen*. New York: Artisan Books, 2023.
75. Vancampfort D, Solmi M, Firth J, et al. The impact of pharmacologic and nonpharmacologic interventions to improve physical health outcomes in people with dementia: a meta-review of meta-analyses of randomized controlled trials. *J Am Med Dir Assoc* 2020; 21: 1410–1414.e2.
76. Livingston G, Sommerlad A, Orgeta V, et al. Dementia prevention, intervention, and care. *Lancet* 2017; 390: 2673–2734.
77. Sofi F, Valecchi D, Bacci D, et al. Physical activity and risk of cognitive decline: a meta-analysis of prospective studies. *J Intern Med* 2011; 269: 107–117.
78. Demurtas J, Schoene D, Torbahn G, et al. Physical activity and exercise in mild cognitive impairment and dementia: an umbrella review of intervention and observational studies. *J Am Med Dir Assoc* 2020; 21: 1415–1422.
79. de la Torre JC. Cardiovascular risk factors promote brain hypoperfusion leading to cognitive decline and dementia. *Cardiovasc Psychiatry Neurol* 2012; 2012: 367516.
80. Currie J, Ramsbottom R, Ludlow H, et al. Cardio-respiratory fitness, habitual physical activity and serum brain derived neurotrophic factor (BDNF) in men and women. *Neurosci Lett* 2009; 451: 152–155.
81. Chen LZ, Yuan X, Zhang Y, et al. Brain functional specialization is enhanced among tai chi chuan practitioners. *Arch Phys Med Rehabil* 2020; 101: 1176–1182.
82. Lautenschlager NT, Cox K and Cyarto EV. The influence of exercise on brain aging and dementia. *Biochim Biophys Acta* 2012; 1822: 474–481.
83. Valenzuela PL, Castillo-García A, Morales JS, et al. Exercise benefits on Alzheimer's disease: state-of-the-science. *Ageing Res Rev* 2020; 62: 101108.
84. Boere K, Lloyd K, Binsted G, et al. Exercising is good for the brain but exercising outside is potentially better. *Sci Rep* 2023; 13: 1140.
85. National Heart, Lung, and Blood Institute. Sleep Deprivation and Deficiency: What are sleep deprivation and deficiency?, www.nhlbi.nih.gov/health/sleep-deprivation (2022, accessed 20 March 2025).
86. Johns Hopkins Medicine. Sleep Deprivation: What is sleep deprivation?, www.hopkinsmedicine.org/health/conditions-and-diseases/sleep-deprivation (2019, accessed 20 March 2025).
87. Miner B and Kryger MH. Sleep in the aging population. *Sleep Med Clin* 2017; 12: 31–38.
88. Li X, Zheng Y, Ma X, et al. Long-term exposure to PM2.5, sleep quality and obstructive sleep apnoea in Chinese adults. *Environ Int* 2020; 139: 105685.
89. Schubert J, Rieger J, Klingelhöfer D, et al. Road-traffic noise, insomnia and high sleep disturbance: results from the German Gutenberg health study. *Environ Res* 2023; 231: 116103.
90. Johnson L, Reid C, Dimitriadis K, et al. Wildfire smoke, nocturnal light pollution and nitrogen dioxide exposure are associated with fragmented slow-wave sleep. *Sleep* 2023; 46: zsad169.
91. Partinen M. Sleep research in 2020: COVID-19-related sleep disorders. *Lancet Neurol* 2021; 20: 15–17.
92. Gaur A, Kaliappan A, Balan Y, et al. Sleep and Alzheimer: the link. *Maedica (Bucur)* 2022; 17: 177–185.
93. Sabia S, Fayosse A, Dumurgier J, et al. Association of sleep duration in middle and old age with incidence of dementia. *Nat Commun* 2021; 12: 2289.
94. Piber D, Cho JH, Lee O, et al. Sleep disturbance and activation of cellular and transcriptional mechanisms of inflammation in older adults. *Brain Behav Immun* 2022; 106: 67–75.
95. Ahmad MA, Kareem O, Khushtar M, et al. Neuroinflammation: a potential risk for dementia. *Int J Mol Sci* 2022; 23: 616.
96. Sartori AC, Vance DE, Slater LZ, et al. The impact of inflammation on cognitive function in older adults: implications for healthcare practice and research. *J Neurosci Nurs* 2012; 44: 206–217.
97. Billioti de Gage S, Moride Y and Ducruet T. Benzodiazepine use and risk of Alzheimer's disease: case-control study. *Br Med J* 2014; 349: S205.
98. Etcheto M, Olloquequi J, Sánchez-López E, et al. Benzodiazepines and related drugs as a risk factor in Alzheimer's disease dementia. *Front Aging Neurosci* 2020; 11: 344.
99. Innes KE, Innes KE, Selfe TK, et al. Effects of meditation versus music listening on perceived stress, mood, sleep, and quality of life in adults with early memory loss: a pilot randomized controlled trial. *J Alzheimers Dis* 2016; 52: 1277–1298.
100. Hess WR. Ueber die wechselbeziehungen zwischen psychischen und vegetativen funktionen. *Schweiz Arch Neurol Psychiatr* 1925; 16: 3655.
101. Benson H, Beary JF and Carol MP. Relaxation response. *Psychiatry* 1974; 37: 37–46.
102. Dusek JA, Hibberd PL, Buczynski B, et al. Stress management versus lifestyle modification on systolic hypertension and medication elimination: a randomized trial. *J Altern Complement Med* 2008; 14: 129–138.

103. Dusek JA, Otu HH, Wohlhueter AL, et al. Genomic counter-stress changes induced by the relaxation response. *PLoS One* 2008; 3: e2576.
104. Bhasin M, Bhasin M, Dusek JA, et al. Relaxation response induces temporal transcriptome changes in energy metabolism, insulin secretion and inflammatory pathways. *PLoS One* 2013; 8: e62817.
105. Orme-Johnson D, Barnes VA and Schneider RH. Effects of the transcendental meditation program on cardiovascular risk factors and clinical events. In: Allan R and Fisher J (eds) *Heart and Mind: The Practice of Cardiac Psychology*. 2nd ed. Washington: DC: American Psychological Association, 2011, pp.365–379.
106. Pilkington K, Gerbarg PL, Brown RP, et al. Yoga therapy for anxiety. In: Khalsa SBS, Cohen L and Telles S (eds) *The principles and practice of yoga in health care*. 1st ed. London: Handspring Publishing, 2016, pp.95–114.
107. Uebelacker LA, Lavretsky H, Tremont G, et al. Yoga therapy for depression. In: Khalsa SBS, Cohen L and Telles S (eds) *The principles and practice of yoga in health care*. 1st ed. London: Handspring Publishing, 2016, pp.73–91.
108. Kozasa E, Santaella D, Mishra S, et al. Yoga therapy for neurological and immune conditions. In: Khalsa SBS, Cohen L, Telles S, et al. (eds) *The principles and practice of yoga in health care*. 1st ed. London: Handspring Publishing, 2016, pp.185–202.
109. Varambally S, Kutty BM, Gangadhar BN, et al. Yoga therapy for other mental health conditions. In: Khalsa SBS, Cohen L and Telles S (eds) *The principles and practice of yoga in health care*. 1st ed. London: Handspring Publishing, 2016, pp.119–131.
110. Lazar SW, Bush CG, Gollub RL, et al. Functional brain mapping of the relaxation response and meditation. *Neuroreport* 2000; 11: 1581–1585.
111. Samuelson M, Foret M, Baim M, et al. Exploring the effectiveness of a comprehensive mind-body intervention for medical symptom relief. *J Altern Complementary Med* 2010; 16: 187–192.
112. Dusek JA, Chang BH, Zaki J, et al. Association between oxygen consumption and nitric oxide production during the relaxation response. *Med Sci Monit* 2005; 12: CR1–C10.
113. Wells RE, Wells RE, Kerr CE, et al. Meditation for adults with mild cognitive impairment: a pilot randomized trial. *J Am Geriatr Soc* 2013; 61: 642–645.
114. Jacobs TL, Epel ES, Lin J, et al. Intensive meditation training, immune cell telomerase activity, and psychological mediators. *Psychoneuroendocrinology* 2011; 36: 664–681.
115. Lavretsky H, Epel ES, Siddarth P, et al. A pilot study of yogic meditation for family dementia caregivers with depressive symptoms: effects on mental health, cognition, and telomerase activity. *Int J Geriatr Psychiatry* 2013; 28: 57–65.
116. Newberg AB, Wintering N, Khalsa DS, et al. Meditation effects on cognitive function and cerebral blood flow in subjects with memory loss: a preliminary study. *J Alzheimers Dis* 2010; 20: 517–526.
117. Newberg AB, Wintering N, Waldman MR, et al. Cerebral blood flow differences between long-term meditators and non-meditators. *Conscious Cogn* 2010; 19: 899–905.
118. Khalsa DS. Alzheimer disease. In: Rakel DP (ed.) *Integrative Medicine*. 2nd ed. Philadelphia: Elsevier Saunders, 2001, pp.133–141.
119. Moss AS, Wintering N, Roggenkamp H, et al. Effects of an 8-week meditation program on mood and anxiety in patients with memory loss. *J Altern Complement Med* 2012; 18: 48–53.
120. Streeter CC, Jensen JE, Perlmutter RM, et al. Yoga asana sessions increase brain GABA levels: a pilot study. *J Altern Complement Med* 2007; 13: 419–426.
121. Luders E, Cherbuin N, Kurth F, et al. Forever young(er): potential age-defying effects of long-term meditation on gray matter atrophy. *Enzyme Microb Technol* 2015; 58: 30–31.
122. Pomykala KL, Silverman DH, Geist C, et al. A pilot study of the effects of meditation on regional brain metabolism in distressed dementia caregivers. *Aging Health* 2012; 8: 509–516.
123. Innes KE, Selfe TK, Selfe TK, et al. Effects of meditation and music-listening on blood biomarkers of cellular aging and Alzheimer's disease in adults with subjective cognitive decline: an exploratory randomized clinical trial. *J Alzheimers Dis* 2018; 66: 947–970.
124. Innes KE, Selfe TK, Khalsa DS, et al. Meditation and music improve memory and cognitive function in adults with subjective cognitive decline: a pilot randomized controlled trial. *J Alzheimers Dis* 2017; 56: 899–916.
125. Eyre HA, Eyre HA, Acevedo BP, et al. Changes in neural connectivity and memory following a yoga intervention for older adults: a pilot study. *J Alzheimers Dis* 2016; 52: 673–684.
126. Newberg AB, Wintering N, Khalsa DS, et al. Meditation effects on cognitive function and cerebral blood flow in subjects with memory loss: a preliminary study. *J Alzheimers Dis* 2010; 20: 517–526.
127. Kaliman P, Álvarez-López MJ, Cosín-Tomás M, et al. Rapid changes in histone deacetylases and inflammatory gene expression in expert meditators. *Psychoneuroendocrinology* 2014; 40: 96–107.
128. The University of Exeter. The PROTECT Study, <https://www.protectstudy.org.uk> (2025, accessed March 14, 2025).
129. Khalsa DS, Amen D, Hanks C, et al. Cerebral blood flow changes during chanting meditation. *Nucl Med Commun* 2009; 30: 956–961.
130. Newberg AB and Iversen J. The neural basis of the complex mental task of meditation: neurotransmitter and neurochemical considerations. *Med Hypotheses* 2003; 61: 282291.
131. de la Monte SM and Tong M. Brain metabolic dysfunction at the core of Alzheimer's disease. *Biochem Pharmacol* 2014; 88: 548–559.

132. Grzenda A, Siddarth P, Milillo MM, et al. Cognitive and immunological effects of yoga compared to memory training in older women at risk for Alzheimer's disease. *Transl Psychiatry* 2024; 14: 96.
133. Kilpatrick LA, Siddarth P, Krause-Sorio B, et al. Impact of yoga versus memory enhancement training on hippocampal connectivity in older women at risk for Alzheimer's disease. *J Alzheimers Dis* 2023; 95: 149–159.
134. Krause-Sorio B, Siddarth P, Kilpatrick LA, et al. Yoga prevents gray matter atrophy in women at risk for Alzheimer's disease: a randomized controlled trial. *J Alzheimers Dis* 2022; 87: 569–581.
135. Lavretsky H. Changes in peripheral AD biomarkers in a randomized controlled trial of yoga vs. Memory training in older women at risk for Alzheimer's disease. *Alzheimers Dement* 2024; 20: e086035.
136. Khalsa DS and Newberg AB. Spiritual fitness: a new dimension in Alzheimer's disease prevention. *J Alzheimers Dis* 2021; 80: 505–519.
137. Sheardova K, Nedelska Z, Šumec R, et al. The effect of spiritual well-being (transcendental and non-transcendental domain) on regional brain atrophy in non-demented subjects with memory complaints: 3-year follow up data from the Czech brain aging study. *Alzheimers Dement* 2018; 14: P587–P588.
138. Koenig HG, VanderWeele V and Peeter J. *Handbook of Religion and Health*. 3rd ed. New York: Oxford University Press, 2021.
139. Lavretsky H. Spirituality and aging. *Aging Health* 2010; 6: 749–769.
140. Ryff CD. Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *J Pers Soc Psychol* 1989; 57: 1069–1081.
141. Ryff CD and Keyes CLM. The structure of psychological well-being revisited. *J Pers Soc Psychol* 1995; 69: 719–727.
142. Khalsa DS and Perry G. The four pillars of Alzheimer's prevention. *Cerebrum* 2017: 03–17.
143. Robitschek C, Ashton MW, Spering CC, et al. Development and psychometric evaluation of the personal growth initiative scale-II. *J Couns Psychol* 2012; 59: 274–287.
144. Post SG. *Is ultimate reality unlimited love?* West Conshohocken: Templeton Press, 2014.
145. Boyle PA, Buchman AS, Barnes LL, et al. Effect of a purpose in life on risk of incident Alzheimer disease and mild cognitive impairment in community-dwelling older persons. *Arch Gen Psychiatry* 2010; 67: 304–310.
146. Musich S, Wang SS, Kraemer S, et al. Purpose in life and positive health outcomes among older adults. *Popul Health Manag* 2017; 21: 139–147.
147. Ahlberg AC, Ljung T, Rosmond R, et al. Depression and anxiety symptoms in relation to anthropometry and metabolism in men. *Psychiatry Res* 2002; 112: 101–110.
148. Landen M, Baghaei F, Rosmond R, et al. Dyslipidemia and high waist-hip ratio in women with self-reported social anxiety. *Psychoneuroendocrinology* 2004; 29: 1037–1046.
149. Raikonen K, Matthews KA, Kuller LH, et al. Anger, hostility, and visceral adipose tissue in healthy postmenopausal women. *Climacteric* 1999; 2: 291–292.
150. Perez-Bret E, Altisent R and Rocafort J. Definition of compassion in healthcare: a systematic literature review. *Int J Palliat Nurs* 2016; 22: 599–606.
151. Dewar B, Pullin S and Tocheris R. Valuing compassion through definition and measurement. *Nurs Manag (Harrow)* 2011; 17: 32–37.
152. Torralba Rosello F. Constructos eticos del cuidar. *Enferm Intensiva* 2000; 11: 136–141.
153. Nouwen HJM, McNeill DP and Morrison DA. *Compassion: A reflection on the Christian Life*. New York: Image Books Doubleday, 2006.
154. Jimenez A, Triana M and Washburn J. Compasion y salud. *Isegoria* 2002; 27: 211–223.
155. Koenig HG. Maintaining health and well-being by putting faith into action during the COVID-19 pandemic. *J Relig Health* 2020; 59: 2205–2214.
156. Hart CW and Koenig HG. Religion and health during the COVID-19 pandemic. *J Relig Health* 2020; 59: 1141–1143.
157. Ryff CD and Singer BH. Know thyself and become what you are: a eudaimonic approach to psychological well-being. *J Happiness Stud* 2008; 9: 13–39.
158. Post SG. *Unlimited love*. Radnor: Templeton Press, 2013.
159. Kimble MA. Beyond the biomedical paradigm: generating a spiritual vision of ageing. *J Relig Gerontol* 2002; 12: 31–41.
160. Dolan P, Krekel C, Shreedhar G, et al. Happy to help: the welfare effects of a nationwide micro-volunteering programme. Happy to help: the welfare effects of a nationwide micro-volunteering programme. *CEP Discussion Papers* (1772). London, UK: London School of Economics and Political Science. Centre for Economic Performance, 2021.