**Summary of initial research on cognitive decline and Alzheimers**

**By Peter Senior, 13 October 2019**

**The objective:** find out how to reverse cognitive decline.

**Hypothesis:** the standard medical view that cognitive decline and Alzheimers can at best be alleviated and that the is no way to reverse it is false.

**Initial task:** to check if Dr Bredesen’s protocol, or some variation, could assist achievement of this objective.

This paper comprises an **initial** chronological list of findings that appear to be of interest. The most promising to date is the first listed together with several aspects of Dr Bredesen’s protocol. SharpAgainNaturally (page 38) references Dr Bredesen’s work and includes several similar protocols.

Further updates will be provided shortly.

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(Analysis, Conclusions and summary – to come)

**Dr Dale Bredesen’s book: End of Alzheimer’s**

[https://www.amazon.com.au/s/ref=nb\_sb\_noss\_2?url=search-alias%3Dstripbooks&field-keywords=dr+dale+bredesen](https://www.amazon.com.au/s/ref%3Dnb_sb_noss_2?url=search-alias%3Dstripbooks&field-keywords=dr+dale+bredesen)

<https://www.apoe4.info/wiki/Bredesen_Protocol> :

[Dr. Dale Bredesen](https://www.drbredesen.com/thebredesenprotocol/) has created the ReCODE protocol that involves multiple strategies to address specific health issues that contribute to cognitive decline and Alzheimer's Disease (AD). The results of each strategy are measured by using blood tests, cognitive evaluations, and other markers of overall health improvements. Actions are tweaked over time to aim for optimal lab and evaluation results. His analogy is to think of AD as a leaky roof - there are as many as 36 leaks in the AD roof that need to be addressed to stop the problem. Not every patient will have the same leaks, and the protocol is customized based on the patient’s genetics, current health, and lifestyle.

 In 2014, his first published paper on the protocol, [Reversal of Cognitive Decline](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4221920/), highlighted 10 case studies. Of those 10 people, nine showed enough improvement to return to normal life activities. Several hundred people with cognitive impairment have since followed the protocol, and most have seen a reversal of cognitive impairment. He published results of reversing various levels of cognitive decline in [Reversal of Cognitive Decline: 100 patients](https://www.omicsonline.org/open-access/reversal-of-cognitive-decline-100-patients-2161-0460-1000450.pdf), published October 2018. His book *The End of Alzheimer's*, published August 2017 discusses his protocol and explains many of the mechanisms of Alzheimer's.

 Bredesen’s protocol has not been tested as a preventative, however in a May 2019 podcast interview, Dr Bredesen did say that he’s never had someone at risk come in for prevention and develop even mild cognitive impairment. Research has shown that amyloid-β is deposited in E4 carriers as early as their thirties, so addressing components prior to experiencing cognitive impairment symptoms will likely lead to better health and cognition in aging. Members on the APOE4.Info forum who follow the protocol report improvements not only in health but also in cognition, even if they do not have an SCI or MCI diagnosis.

 Although Bredesen does not see private patients, he has made his protocol available to those seeking doctor assistance through [AHNP: Precision Health](https://www.ahnphealth.com/). MPI Cognition, his previous affiliation, was acquired by AHNP and his prior affiliation with [Muses Labs](https://museslabs.com/) has ended.

 Dr Dale Bredeson End of Alzheimer’s book:  AUD4.99 on Kindle.  He has been researching Alzheimers with a highly qualified team for 25 years. Their view is that what is measured, in particular plaques and tangles (all explained in the book) are only the indirect cause of brain cells being killed deliberately.  The real cause, he explains, is that the brain has a natural defence mechanism that tries to stop the plaques and tangles forming and, if these build up too much, it goes haywire and starts killing too many brain cells.  The trick is to stop the mechanisms that cause the plaque and tangle growth. They have identified 36 so far (see below) and expect just a few more to be found.  These come in categories of inflammatory, toxic and nutrients.  Their programme is to identify which are the major causes for each individual – it varies it seems – and tackle each one starting with the most prominent.  Their program is called ReCODE, and they are training numerous nurses and doctors to apply it.  The reason ReCODE hasn’t made the main-stream is they are not allowed to carry out formal test approval programs, as required for all approved medical drugs.  Big Pharma only want to sell huge numbers of pills.  Problem is, it isn’t a matter of one super pill as each person’s needs vary as which of the 36+ are the major causes, so testing just one would be a waste of time and only appear to prove the treatment doesn’t work.  Ie stop the plaques and tangles forming and the brain’s defence mechanism reckons all is now well, stops killing cells and shuts down until needed.  But the official approval mechanism only allows one factor at a time to be tested, so the approval mechanism for multiple factors has not been approved.  They have had many successes when doctors who knew of this program sent their patients to Dr Bredersen – with amazing full recoveries. The list below links to summaries of why each strategy is important, what you can do, and a selection of research references. NB: Cognoscopy, chapter 7

 **Diet Strategies**

[Optimize diet](https://www.apoe4.info/wiki/Optimize_diet)

[Enhance autophagy and ketogenesis](https://www.apoe4.info/wiki/Enhance_autophagy%2C_ketogenesis)

[Improve GI Health](https://www.apoe4.info/wiki/GI_health)

  **Lifestyle Strategies**

[Reduce stress](https://www.apoe4.info/wiki/Reduce_stress)

[Optimize sleep](https://www.apoe4.info/wiki/Optimize_sleep)

[Exercise](https://www.apoe4.info/wiki/Exercise)

[Rule out sleep apnea](https://www.apoe4.info/wiki/Ensure_nocturnal_oxygenation)

[Optimize mitochondrial function](https://www.apoe4.info/wiki/Optimize_mitochondrial_function)

 **Lab Tests to Track and Treat**

[Homocysteine](https://www.apoe4.info/wiki/Homocysteine_less_than_6)

[B vitamins](https://www.apoe4.info/wiki/Serum_B12_greater_than_500)

[Inflammation](https://www.apoe4.info/wiki/CRP_less_than_1.0%3B_A/G_greater_than_1.8)

[Insulin sensitivity](https://www.apoe4.info/wiki/Fasting_insulin_less_than_5;_HgbA1c_less_than_5.6) (insulin and blood glucose)

[Hormones](https://www.apoe4.info/wiki/Hormone_balance)

[Zn:fCu ratio](https://www.apoe4.info/wiki/Optimize_Zn%3AfCu_ratio)

[Vitamin D](https://www.apoe4.info/wiki/25OH-D3_%3D_50-80ng/ml)

[Rule out heavy metal toxicity](https://www.apoe4.info/wiki/Exclude_heavy_metal_toxicity)

[Optimize antioxidants](https://www.apoe4.info/wiki/Optimize_antioxidants)  ??

 **Brain Strategies**

[Brain stimulation](https://www.apoe4.info/wiki/Brain_stimulation)

[Reduction of Aß](https://www.apoe4.info/wiki/Reduction_of_A%C3%9F)

[Cognitive enhancement](https://www.apoe4.info/wiki/Cognitive_enhancement)

[Increase NGF](https://www.apoe4.info/wiki/Increase_NGF)

[Provide synaptic structural components](https://www.apoe4.info/wiki/Provide_synaptic_structural_components)

[Increase focus](https://www.apoe4.info/wiki/Increase_focus)

[Increase SirT1 function](https://www.apoe4.info/wiki/Increase_SirT1_function)

[Inhalational Alzheimer's](https://www.apoe4.info/wiki/Inhalational_Alzheimer%27s) (editing note: update to types of AD)

# 2016 paper <https://www.aging-us.com/article/100981/text>

# Reversal of cognitive decline in Alzheimer's disease

#### **Dale E. Bredesen**[**1**](https://www.aging-us.com/article/100981/text)**,**[**2**](https://www.aging-us.com/article/100981/text)**, Edwin C. Amos**[**3**](https://www.aging-us.com/article/100981/text)**, Jonathan Canick**[**4**](https://www.aging-us.com/article/100981/text)**, Mary Ackerley**[**5**](https://www.aging-us.com/article/100981/text)**, Cyrus Raji**[**6**](https://www.aging-us.com/article/100981/text)**, Milan Fiala**[**7**](https://www.aging-us.com/article/100981/text)**, Jamila Ahdidan**[**8**](https://www.aging-us.com/article/100981/text)

* 1 Easton Laboratories for Neurodegenerative Disease Research, Department of Neurology, University of California, Los Angeles, CA 90095, USA
* 2 Buck Institute for Research on Aging, Novato, CA 94945, USA
* 3 Department of Neurology, University of California, Los Angeles, CA 90095, USA
* 4 Memory Clinic, California Pacific Medical Center, San Francisco, CA 94115, USA
* 5 Private Practice of Psychiatry, Tucson, AZ 85718, USA
* 6 Department of Radiology, University of California, Los Angeles, CA 90095, USA
* 7 Department of Surgery, University of California, Los Angeles, CA 90095, USA
* 8 Brainreader, Horsens, Denmark

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### Abstract

Alzheimer's disease is one of the most significant healthcare problems nationally and globally. Recently, the first description of the reversal of cognitive decline in patients with early Alzheimer's disease or its precursors, MCI (mild cognitive impairment) and SCI (subjective cognitive impairment), was published [1]. The therapeutic approach used was programmatic and personalized rather than monotherapeutic and invariant, and was dubbed metabolic enhancement for neurodegeneration (MEND). Patients who had had to discontinue work were able to return to work, and those struggling at work were able to improve their performance. The patients, their spouses, and their co-workers all reported clear improvements. Here we report the results from quantitative MRI and neuropsychological testing in ten patients with cognitive decline, nine ApoE4+ (five homozygous and four heterozygous) and one ApoE4-, who were treated with the MEND protocol for 5-24 months. The magnitude of the improvement is unprecedented, providing additional objective evidence that this programmatic approach to cognitive decline is highly effective. These results have far-reaching implications for the treatment of Alzheimer's disease, MCI, and SCI; for personalized programs that may enhance pharmaceutical efficacy; and for personal identification of ApoE genotype.

# Dr. Dale Bredesen on Preventing and Reversing Alzheimer's Disease

<https://www.foundmyfitness.com/episodes/dale-bredesen> - 58 min video

Dale E. Bredesen, M.D., is a professor of neurology at the Easton Laboratories for Neurodegenerative Disease Research at the David Geffen School of Medicine at the University of California, Los Angeles (UCLA).

Dr. Bredesen’s laboratory focuses on identifying and understanding basic mechanisms underlying the neurodegenerative process and the translation of this knowledge into effective treatments for Alzheimer’s disease and other neurodegenerative conditions. He has collaborated on the publication of more than 220 academic research papers.

He and his colleagues have identified several subtypes of Alzheimer’s disease and has developed ReCODE – reversal of cognitive decline – a protocol that offers a new approach to treatment that has reversed symptoms in patients with mild cognitive impairment and Alzheimer’s disease.

Dr. Bredesen received his undergraduate degree from the California Institute of Technology and his medical degree from Duke University. He served as Resident and Chief Resident in Neurology at the University of California, San Francisco (UCSF). He was the Founding President and CEO of the Buck Institute for Research on Aging and Adjunct Professor at UCSF.

## The major subtypes of Alzheimer’s disease.

Identified just over a century ago, Alzheimer’s disease is a complex, multifaceted condition that affects nearly 44 million people worldwide. In this episode, Dr. Dale Bredesen identifies the defining characteristics of Alzheimer’s disease and enumerates its primary subtypes:

1. **The inflammatory subtype of Alzheimer’s disease.**
	* A type characterized by systemic inflammation, reflected in such laboratory results as a high hs-CRP (high-sensitivity C-reactive protein), low albumin:globulin ratio, and high cytokine levels such as interleukin-1 and interleukin-6.
2. **The atrophic subtype of Alzheimer's disease** — a reduction in support for synaptogenesis.
	* A type characterized by an atrophic profile, with reduced support from molecules such as estradiol, progesterone, brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), testosterone, insulin, and vitamin D, often accompanied by increased homocysteine and insulin resistance, the last feature of which Dr. Bredesen refers to as type 1.5 or glycotoxicity.
3. **The cortical subtype of Alzheimer's disease** — an environmental toxin-related type associated with chronic Inflammatory response syndrome (CIRS) that presents with more general cerebral atrophy and frontal-temporal-parietal abnormalities, resulting in an emphasis on executive deficits, rather than the more amnestic quality of hippocampal impairment.

Although the subtypes vary in their causes and manifestation and often overlap to some degree, Dr. Bredesen explains that the underlying pathological features – the accumulation of amyloid beta plaques and tau tangles – are unifying aspects of the disease. He adds that how these features play out in the somewhat fragile environment of the brain depends on a wide array of contextual parameters, such as genetics and lifestyle factors, including diet, sleep, exercise, and environmental exposures.

[*Click here to read Dr. Bredesen’s paper summarizing these subtypes of Alzheimer's disease*](https://www.ncbi.nlm.nih.gov/pubmed/26870879).

## The emergence of new properties of amyloid-beta.

["We think more and more of amyloid as being like napalm." - Dale Bredesen, M.D.](https://twitter.com/intent/tweet?text=We%20think%20more%20and%20more%20of%20amyloid%20as%20being%20like%20napalm.%22%20-%20%40Dr_Bredesen%20https%3A%2F%2Fwww.foundmyfitness.com%2Fepisodes%2Fdale-bredesen)

#### Amyloid-beta as an antimicrobial response.

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. But in recent decades research has revealed interesting characteristics that suggest amyloid-beta can [play a protective role against fungal, bacterial, and viral infections](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6075814/).

One example is seen in the herpes virus, which upregulates the production of amyloid-beta protein in vitro. In turn, the protein binds to and agglutinates the viral particles. Perhaps more importantly, increased production of amyloid-beta improves survival in animals subjected to a viral assault, a phenomenon that strongly supports an [antimicrobial protection hypothesis of Alzheimer’s disease](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6075814/). There may be good reason for this antimicrobial property too: 90% of glioblastomas, a type of brain cancer, have been shown to express a herpes type of virus known as cytomegalovirus. Most people harbor latent herpes virus infections, and some evidence suggests that [reactivation of the latent virus in the brain, particularly in APOE4 carriers, might increase the risk of developing Alzheimer’s disease](https://www.sciencedirect.com/science/article/pii/S0140673696101495).

#### The interaction of metals with amyloid-beta.

One of the roles amyloid-beta may also play as “protector” is that of binder of transition metals like zinc, copper, or iron. Animal experiments demonstrate that [chelating agents can even reduce deposition of amyloid-beta](https://www.researchgate.net/publication/8024602_Amyloid-beta_metal_interaction_and_metal_chelation). These interactions with metals become important in the discussion of Dr. Bredesen’s protocol where a combination of early-onset, non-amnestic cognitive changes, and biomarkers like altered copper-to-zinc ratio, especially low serum zinc, might be suggestive of the “cortical” or “toxic” subtype of Alzheimer’s disease.

[*Click here for a FoundMyFitness episode featuring guest Dr. Gordon Lithgow discussing metals in the context of aging, protein aggregation, and neurodegenerative disease*](https://www.foundmyfitness.com/episodes/gordon-j-lithgow).

## The role of the APOE4 polymorphism in Alzheimer’s disease.

["In the past, people said, 'Don't check because there's nothing you can do about it,' and that has completely changed." - Dale Bredesen, M.D.](https://twitter.com/intent/tweet?text=%22In%20the%20past%2C%20people%20said%2C%20%27Don%27t%20check%20because%20there%27s%20nothing%20you%20can%20do%20about%20it%2C%27%20and%20that%20has%20completely%20changed.%20-%20%40Dr_Bredesen%20https%3A%2F%2Fwww.foundmyfitness.com%2Fepisodes%2Fdale-bredesen)

More than 75 million people in the US carry at least one allele for APOE4, a version of apolipoprotein E that is the major genetic risk factor for Alzheimer’s disease, which some studies show may increase the odds of developing Alzheimer’s disease by [as much as 2- to 3-fold in the case of heterozygotes and as much as 15-fold in homozygotes](https://www.ncbi.nlm.nih.gov/pubmed/9343467). The old mantra – that little could be done to prevent APOE-related Alzheimer’s disease – is now being challenged, however. Dr. Bredesen’s research indicates that, armed with knowledge, we can make dietary and lifestyle changes to prevent or at least delay the cognitive losses that once seemed to be one’s destiny.

A key element in the acquisition of knowledge about our risks factors and what can be done to manage our risks is what Dr. Bredesen calls a “cognoscopy” – a term he coined that describes a battery of assessments, including biochemical tests, which measure some of the key biomarkers for Alzheimer’s disease.

[*Learn more about the ReCODE baseline testing available through ahnphealth.com*](https://www.ahnphealth.com/inform.html).

## The biomarkers of Alzheimer’s disease and the Bredesen Cognoscopy.

["[Alzheimer’s disease] should essentially decrease to a very low level with the current generation. If everybody gets checked, we recommend that everybody 45 or over get a cognoscopy." - Dale Bredesen, M.D.](https://twitter.com/intent/tweet?text=%5BAlzheimer%E2%80%99s%20disease%5D%20should%20essentially%20decrease%20to%20a%20very%20low%20level%20with%20the%20current%20generation%2C%20if%20everybody%20gets%20checked.%20We%20recommend%20that%20everybody%2045%20or%20over%20get%20a%20cognoscopy.%22%20-%20%40Dr_Bredesen%20https%3A%2F%2Fwww.foundmyfitness.com%2Fepisodes%2Fdale-bredesen)

In this episode, Dr. Bredesen proposes a pretty radical idea: Alzheimer’s disease as we know it could be largely ended with the current generation. The key to doing this? By treating the prevention of Alzheimer’s in much the same way we treat colon cancer — with screening to detect the first signs of trouble.

However, a recurring theme a recurring theme throughout our discussion with Dr. Bredesen is that what most labs and clinicians call normal may actually be different than what is optimal. In fact, perhaps the key take-home message from this episode is that not only should we potentially get tested and keep an eye on certain biomarkers in particular, but more importantly, for some of these tests, our goal should be to keep our ranges even healthier than what the laboratory references may indicate as “normal.”

Whether a person is at risk of developing the disease versus actively manifesting symptoms is often reflective of the number of their suboptimal biomarkers: as few as three to five suboptimal lab values may be observed in an at-risk pre-symptomatic person versus up to 25 in a symptomatic person. (See chapter 7, The "Cognoscopy" — Where Do You Stand? in Dr. Bredesen’s book [*The End of Alzheimer’s*](https://www.penguinrandomhouse.com/books/551532/the-end-of-alzheimers-by-dale-e-bredesen-md/9780735216204/))

In case you’re wondering what just a few (not all!) of those key biomarkers might be, here are a few from the episode that stand out if for no other reason than Dr. Bredesen mentioned them and provided the ranges he thinks are more optimal than the standard “within normal limits” ranges:

* hsCRP (less than 1.0 mg/L)
* Fasting insulin (less than 7 mIU/L)
* Hemoglobin A1c (less than 5.5%)
* Homocysteine (less than 7 μmol/L)

[*View a more comprehensive list at popular APOE4 community site apoe4.info*](https://www.apoe4.info/wiki/Bredesen_Protocol).

***In this episode, we also discuss...***

* [00:00:53](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h00m53s) - The defining pathological hallmarks of Alzheimer’s disease, the imperfectness of these markers, and the overall prevalence of the disease.
* [00:01:56](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h01m56s) - How understanding amyloid-beta in the context of a protective response helps us make sense of asymptomatic deposition of amyloid-beta. [Herpes study](https://www.ncbi.nlm.nih.gov/pubmed/30001512).
* [00:02:41](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h02m41s) - The antimicrobial and metal-binding characterics of amyloid-beta, the latter of which includes metals like copper, zinc, and iron. [Metal-binding study](https://link.springer.com/chapter/10.1007/0-387-23226-5_12).
* [00:03:32](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h03m32s) - The inclusion of inflammation as a special determining factor that seems to often make the difference between asymptomatic and symptomatic deposition of amyloid-beta.
* [00:03:50](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h03m50s) - The three to four major subtypes that Alzheimer’s disease falls into based on around 36 different factors identified by Dr. Bredesen’s group. These subtypes include: inflammation, glycotoxicity (insulin resistance), lack of trophic support, and an early onset, toxicity-related “cortical” subtype.
* [00:05:08](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h05m08s) - The atrophic subtype of Alzheimer’s disease, which Dr. Bredesen describes as a lack of proper cell signalling as a consequence of long-term reductions in nerve growth factor, brain-derived neurotrophic factor and other hormones that result in a fall in synaptoblastic-to-synaptoclastic ratio (in other words, synapse-generationg versus synapse-destroying activities).
* [00:06:36](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h06m36s) - The glycotoxic subtype of Alzheimer’s disease, which is associated with a loss of trophic support due to insulin resistance even in patients that have otherwise normal insulin responsiveness in peripheral tissues.
* [00:07:41](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h07m41s) - The cortical or “toxic” subtype of Alzheimer’s disease, which presents earlier in age and less amnestically and may be associated with various environmental issues outlined by Dr. Bredesen, such as environmental molds or metals. See also [*00:12:30*](https://www.foundmyfitness.com/episodes/dale-bredesen) for an exhaustive description of this unique subtype.
* [00:09:30](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h09m30s) - Low serum zinc or high copper-to-zinc ratios, possibly in combination with low triglycerides, as unique biomarkers associated with the type 3 “cortical” Alzheimer’s disease pathogenesis.
* [00:10:10](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h10m10s) - Zinc deficiency as a downstream effect of changes in gastric acidity, potentially from the taking of proton pump inhibitors and also as a consequence of overexposure to copper.
* [00:13:48](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h13m48s) - ApoE4 as a risk factor for Alzheimer’s disease and Dr. Bredesen’s extremely optimistic take on the opportunity for mitigating that risk and even near-elimination of the Alzheimer’s disease phenomena.
* [00:15:48](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h15m48s) - The key to Dr. Bredesen’s risk reduction strategy: embracing what he terms as a “cognoscopy,” the suite of tests, including blood tests and more, that he believes should be checked and monitored by everyone aged 45+ (in much the same way we screen for colon cancer with regular colonoscopies in older adults).
* [00:17:23](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h17m23s) - The MEND protocol and its successor, the ReCODE protocol, which is now used in over 3,000 patients and stands for “reversal of cognitive decline.” [Learn more about Dr. Bredesen’s ReCODE protocol (or “cognoscopy”) by clicking here](http://www.ahnphealth.com/inform.html).
* [00:18:06](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h18m06s) - The 2014 publication featuring the case reports of 10 patients that experienced strong reversal of functional decline, returning to work, and even improved hippocampal volume. [2014 MEND study](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4221920/). Dr. Bredesen also shares some details on his upcoming publication, namely, that it will include a more substantial 50 patient cohort.
* [00:18:46](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h18m46s) - The problem of trying to treat a disease without knowing the underlying cause and, similarly, only approaching the condition reactively instead of proactively.
* [00:19:58](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h19m58s) - The role some pathogens may have in Alzheimer’s disease, such as the Borrelia of lyme disease or it’s many co-infections like Babesia, Borrelia, Bartonella, and Ehrlichia.
* [00:20:21](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h20m21s) - Some of the key blood-based biomarkers that are useful for tracking and projecting future cognitive health, including: homocysteine (<7µml/L preferred), which Dr. Bredesen indicates may be a mediator of cerebral atrophy, and also fasting insulin (<5% preferred), hemoglobin A1c, fasting glucose, vitamin D, and a number of other hormones relevant for trophic support.
* [00:21:54](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h21m54s) - The combination of blood markers that defines the Alzheimer’s disease subtype that Dr. Bredesen refers to as type 1.5 or “glycotoxic” Alzheimer’s.
* [00:23:00](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h23m00s) - The biomarkers, particularly hormones, that make up the “atrophic” subtype of Alzheimer’s disease, which Dr. Bredesen refers to as type 2 Alzheimer’s. Note: Some of these signaling factors, namely BDNF, are not amenable to being assayed, but are produced robustly in response to exercise.
* [00:24:03](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h24m03s) - The environmental-historical context behind the prevalence of APOE4 that might help explain why a gene variant thought to be highly deleterious exists in a whopping 25% of the population.
* [00:25:38](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h25m38s) - The specific toxins Dr. Bredesen is most concerned with as potential risk factors for Alzheimer’s disease, particularly the “cortical” variety he refers to as type 3.
* [00:25:52](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h25m52s) - How certain molds may actually increase their toxins in response to stresses we place on them, like fungicides that are added to treated wood.
* [00:26:32](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h26m32s) - The chronic inflammatory condition associated with water-damaged buildings called chronic inflammatory response syndrome or “CERS.” [CERS study](https://www.sciencedirect.com/science/article/pii/S0892036214001329).
* [00:27:28](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h27m28s) - The Bredesen Protocol recommended diet, which Dr. Bredesen refers to as “Ketoflex 12/3”, so-named to highlight three points of emphasis: 1) mild ketosis, 2) a “flexitarian” approach that treats meat as a condiment instead of a main course, 3) At least twelve-hours of daily fasting starting three-hours before bed.
* [00:29:00](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h29m00s) - An interesting clinical example of surprise mercury toxicity, possibly from diet and other factors, including genetic, associated with early mild cognitive impairment that was PET scan-positive for amyloid deposition.
* [00:31:17](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h31m17s) - A slightly more aggressive daily time-restricted eating routine for APOE4 carriers, including 14 to 16 hours of daily fasting. [*See the episodes with Dr. Panda for a great discussion of daily time-restricted eating*](https://www.foundmyfitness.com/episodes/satchin-panda).
* [00:33:53](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h33m53s) - Dr. Bredesen’s clinical observation that patients with higher ketone levels, in the range of 1.5mmol to 4mmol/L beta-hydroxybutyrate, do better as a whole (even when carriers of APOE4).
* [00:34:09](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h34m09s) - Tips for easing into dietary ketosis, potential pitfalls to look out for, and biomarkers to keep an eye on for further tailoring after the early adaptation period.
* [00:37:16](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h37m16s) - Some of the improvements seen in rodent research in terms of healthspan, but also memory and brain function, from a cyclical ketogenic diet. [*See the episode with Dr. Eric Verdin for a discussion on this research*](https://www.foundmyfitness.com/episodes/eric-verdin).
* [00:38:42](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h38m42s) - A subtle way that utilizing ketones to meet more energetic needs in the brain may be beneficial: it leaves more glucose to be used by the pentose phosphate pathway, possibly resulting in increased production of the cellular antioxidant glutathione.
* [00:41:20](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h41m20s) - How a “leaky gut” may be playing a role in diseases of chronic inflammation. Note: The term “leaky gut” is a colourful way of describing a gut with poor protective barrier function, which hypothetically enables translocation of inflammatory endotoxin from enterobacteria that naturally reside there that would otherwise be ordinarily benign.
* [00:42:42](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h42m42s) - The propensity of those with cognitive decline to exhibit peripheral macrophage immune cells that are characteristically poor at phagocytosing and clearance of amyloid-beta and how this may be affected by omega-3 supplementation. [Relevant study](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5161513/).
* [00:46:29](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h46m29s) - The involvement of omega-3 derived signaling molecules called specialized pro-resolving mediators, including molecules like the resolvins and maresins, in resolving inflammation.
* [00:48:50](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h48m50s) - Differences in omega-3 DHA transport that may affect APOE4 carriers. [Relevant study](https://www.ncbi.nlm.nih.gov/pubmed/24345162).
* [00:50:23](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h50m23s) - The reductions in glucose transport in the brain that may occur in the context of omega-3 DHA-deficiency. [Relevant study](https://www.ncbi.nlm.nih.gov/pubmed/12068080).
* [00:50:58](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h50m58s) - Some of the recent research into how amyloid-beta is produced as an antimicrobial response to the Herpes virus and the association this infection seems to have with Alzheimer’s disease.
* [00:52:33](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h52m33s) - The recent research out of Finland showing frequent sauna use was associated with a 65% reduction in Alzheimer’s disease and some of the research showing how induced sweating as [a uniquely good method of eliminating certain metals](https://www.ncbi.nlm.nih.gov/pubmed/21057782) like cobalt, cadmium, aluminum, and lead. [*Listen to this episode with Dr. Jari Laukkanen to learn more about the science of sauna use*](https://www.foundmyfitness.com/episodes/jari-laukkanen).
* [00:54:52](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h54m52s) - The challenge in making targeted monotherapeutic approaches work, like directly targeting amyloid-beta with something like monoclonal antibodies, and the importance of addressing underlying causes for which amyloid-beta is being produced in response to.
* [00:59:57](https://www.foundmyfitness.com/episodes/dale-bredesen?t=00h59m57s) - Dr. Bredesen’s take on why tau tangles, also called neurofibrillary tangles, represent generalized synaptoclastic activity that is generated as a response to the insults targeted by the protocol.
* [01:00:59](https://www.foundmyfitness.com/episodes/dale-bredesen?t=01h00m59s) - The subtypes of Alzheimer’s disease Dr. Bredesen encounters most frequently and why insulin resistance can still be crucial, even if it’s not as obvious peripherally.
* [01:03:09](https://www.foundmyfitness.com/episodes/dale-bredesen?t=01h03m09s) - How to find out your ERMI score, which is an index created by the EPA to assess relative safety based on mold concentration in your home.
* [01:05:39](https://www.foundmyfitness.com/episodes/dale-bredesen?t=01h05m39s) - The tests Dr. Bredesen recommends for assessing various aspects gut health.

## People mentioned

* [Bruce Ames](https://en.wikipedia.org/wiki/Bruce_Ames) - [*episode with Dr. Ames*](https://www.foundmyfitness.com/episodes/bruce-ames)
* [Eric Verdin](https://en.wikipedia.org/wiki/Eric_M._Verdin) - [*episode with Dr. Verdin*](https://www.foundmyfitness.com/episodes/eric-verdin)
* [Terry Wahls](https://terrywahls.com/) - [*episode with Dr. Wahls*](https://www.foundmyfitness.com/episodes/terry-wahls)
* [Alois Alzheimer](https://en.wikipedia.org/wiki/Alois_Alzheimer)
* [Rudolph Tanzi](http://www.hms.harvard.edu/dms/neuroscience/fac/tanzi.php)
* [Robert D. Moir](https://www.massgeneral.org/neurology/researcher_profiles/moir_robert.aspx)
* [Ashley Bush](https://pursuit.unimelb.edu.au/individuals/professor-ashley-bush)
* [Ed Goetzl](https://www.eurekalert.org/pub_releases/2010-12/uoc--uo121310.php)
* [Mario F. Mendez](https://www.uclahealth.org/mario-mendez)
* [Robert Mahley](https://profiles.ucsf.edu/robert.mahley)
* [Mark Hyman](https://en.wikipedia.org/wiki/Mark_Hyman_%28doctor%29)
* [Julie Gregory](https://www.apoe4.info/wp/board-of-directors/)
* Mary Newport
* [Caleb “Tuck” Finch](https://en.wikipedia.org/wiki/Caleb_Finch)
* [Ritchie Shoemaker](https://www.survivingmold.com/about/ritchie-shoemaker-m-d)
* [Milan Fiala](https://www.eurekalert.org/pub_releases/2008-04/ip-mfm042608.php)
* [Charles Serhan](https://www.hms.harvard.edu/dms/bbs/fac/Serhan.php)
* [Paul Clayton](http://www.drpaulclayton.com/)
* [Richard Wurtman](https://en.wikipedia.org/wiki/Richard_Wurtman)
* [Norman Salem](https://scholar.google.com/scholar?hl=en&as_sdt=0%2C5&q=author%3A%22N+Salem+Jr%22&btnG=)
* [Aristo Vojdani](http://www.yourmedicaldetective.com/public/148.cfm)
* [Stephen J Genuis](https://www.stephengenuis.com/)

Taken from Dr Bredesen's book ***The End of Alzheimer's*** provided for quick reference, refer to the book for specific information

| **Genetics** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | ApoE | Negative for ApoE4 | Whole genome, exome, or SNPs | Saliva or blood |

| **Blood Tests** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
| Inflammation vs. cellular protection | Hs-CRP | <0.9 | IL-6, TNFalpha |  |
|  | Homocysteine | <7 |  |  |
|  | Vit. B6, B12, folate | 60-100 (B6) 500-1500 (B12)10-25 (folate) |  |  |
|  | Vit. C, D, E | 1.3-2.5 (C) 50-80 (D) 12-20 (E) |  | Vit. D is measured as 25-hydroxy-cholecalciferol |
|  | Omega-6: omega-3 ratio | 0.5-3.0 |  |  |
|  | A/G ratio (albumin:globulin ratio) | ≥ 1.8 > 4.5 (albumin) |  |  |
|  | Fasting insulin, glucose, hemoglobin A1c | ≤ 4.5 (fasting insulin) 70-90 (fasting glucose) <5.6 (HbA1c.) Although in a 2019 interview Dr Bredesen updated to: fasting insulin < 5.0, and A1c of 4.5 to 5.2. | Neural exosome studies (p-tau, AB42, REST, cathepsin D, and IRS-1 phos. Ratio) |  |
|  | Body mass index (BMI) | 18-25 |  |  |
|  | LDL-p or sdLDL or oxidized LDL | 700-1000 (p) <20 (sd) <60 (ox) |  |  |
|  | Cholesterol, HDL, triglycerides | >150 (cholesterol) >50 (HDL) <150 (TG) |  |  |
|  | Glutathione | 5.0-5.5 |  |  |
|  | RBC thiamine pyrophosphate | 100-150 |  |  |
|  | Leaky gut, leaky bloodbrain barrier, gluten sensitivity, autoantibodies | NegativeTest: Cyrex Array 3 & 4 |  |  |

| **Trophic support** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | Vit D. | 50-80 |  |  |
|  | Estradiol (E2), progesterone (p) | 50-250 (E2) 1-20 (p) |  |  |
|  | Pregnenolone, cortisol, DHEA-sulfate | 50-100 (preg) 10-18 (cort) 350-430 (DHEA, women) 400-500 (DHEA, men) |  |  |
|  | Testosterone, free testosterone | 1500-1000 6.5-15 (free) |  |  |
|  | Free T3, free T4, reverse T3, TSH | 3.2-4.2 (fT3) 1.3-1.8 (fT4) <20 (rT3) <2.0 (TSH) Ft3:rt3 ≥20 |  |  |

| **Toxin related** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | Mercury, lead, arsenic, cadmium | <5, <2, <7, <2.5, respectively | <50th percentile (Quicksilver) |  |
|  | Copper:zinc ratio | 0.8-1.2 | RBC zinc; ceruloplasmin |  |
|  | C4a, TGF-B1, MSH | <2830 (C4a) <2380 (TGF-B1) 35-81 (MSH) | MMP9, VEGF, leptin, VIP, ADH, osmolality | If abnormal, add MARCoNS culture and VCS testing |
|  | HLA-DR/DQ | Benign HLA-DR/DQ |  |  |

| **Metals Excluding those listed above** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | RBC-magnesium | 5.2-6.5 |  |  |
|  | Copper, zinc | 90-110 (both) |  |  |
|  | Selenium | 110-150 |  |  |
|  | Potassium | 4.5-5.5 |  |  |
|  | Calcium | 8.5-10.5 |  |  |

| **Cognitive performance** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | CNS Vital Signs, BrainHQ, or equivalent | >50th percentile for age, improving w/ practice | Novel object recognition |  |

| **Imaging** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | MRI w/ volumetrics | Hippocampal, cortical volume percentiles steady (or increasing) for age, >25th percentile |  |  |

| **Sleep** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | Sleep study | AHI <5/h |  |  |

| **Microbiomes** | **Critical tests** | **Target values** | **Optional tests** | **Comments** |
| --- | --- | --- | --- | --- |
|  | Gut, oral, nasal | No pathogens |  |  |

This article presents a handy summary of Dr Bredesen’s approach.

<https://www.news.com.au/lifestyle/health/groundbreaking-research-into-dementia/news-story/9b20f91c8751b80199c35671075c329a>

Groundbreaking research could unlock the answer to a heartbreaking condition that affects about 345,000 Australians, Adam MacDougall, The Health Hacker.

What if I told you that the answer to combating one of the world’s most devastating brain diseases was not something sitting on the shelf in a pharmacy, but something on the end of your fork?

That’s the philosophy behind Dr Dale Bredesen’s groundbreaking research into dementia, a heartbreaking and wide-reaching disease that affects about 447,115 Australians. Up to 70 per cent of those people likely to develop Alzheimer’s.

Those numbers are climbing, too. Dementia Australia predicts that the number of people living with dementia will grow to 589,807 by 2028 and 1.07 million by 2058. And that’s where Dr Bredesen comes in.

Dr Bredesen, the author of the groundbreaking book *The End of Alzheimer’s*, is in Sydney this week at the 7th BioCeuticals Symposium to share his amazing research.

Dr Bredesen believes dementia and its associated diseases are not just preventable, but reversible. The answer, he says, lies in simple lifestyle changes.

“Thing about Alzheimer’s disease is that we always look at the ‘how’ of treatment, but we never spent enough time focusing on the ‘why’ of it occurring in the first place,” Dr Bredesen says.

“That’s what I’ve done, and what my book covers. It’s a protocol for preventing, and helping treat, dementia and Alzheimer’s disease.”

Adam MacDougall with Dr Dale Bredesen.

His research is predictably complex, but can be most easily summarised with idea that three key factors — lifestyle, diet and sleep — are major contributors to the onset of dementia, and by making simple changes to the way we live, we can drastically reduce the risk of developing the disease, and help reduce its impacts in those already suffering.

The idea is that Alzheimer’s disease develops as a response to a build-up of amyloid proteins in the brain. Dr Bredesen’s work focuses on what causes that build-up and how to avoid it.

 “When you produce this amyloid, this is actually a protective response to several problems,” he said.

“If you have ongoing inflammation, whether it’s from problems with your gut, or problems with an infection, or whether it’s because of insulin resistance, you’re actually making this amyloid as a response.

“So, why do you have the amyloid? What we’re looking at is why did you get it?”

The answer, he says, is combination of factors — including gut health, exposure to mercury through fish or dental work, and poor nutrition — but most link back to the modern diet.

**RELATED:**[**Adam MacDougall on how daylight saving messes with your body**](https://www.news.com.au/lifestyle/health/how-daylight-saving-messes-with-your-body/news-story/6abd8538084f01cdca786c7145f016cf)

“The idea of going after something so complex with one drug makes little sense,” he said.

“We tend to eat a diet that is not the diet we ate thousands of years ago. We have way too many grains, too much dairy — and the biggest one of all — simple carbohydrates, sugars.

“So this is a protocol to stop this illness developing, and for generations to come, Alzheimer’s and dementia might be a thing of the past.”

**HACKING BRAIN HEALTH**

• **Diet:** Dr Bredesen advocates the Ketoflex 12/3 diet, which induces mild ketosis that forces the brain to burn fat (which he describes as a “clean fuel”) rather than carbohydrates. It means fasting for 12 hours after your evening meal, and finishing dinner at least three hours before bed. He also suggests limiting meat to two or three meals per week, and only eating SMASH seafood (which stands for salmon, mackerel, anchovies, sardines and herring) and avoiding tuna.

• **Exercise:**When training for your brain, Dr Bredesen says you should be exercising four or five days each week and combine aerobic fitness — including jogging, cycling or brisk walking — with weight or resistance training, aiming to hit a minimum of 150 minutes of exercise every week.

• **Sleep:** To get the maximum healing benefit of sleep, Dr Bredesen says you should be aiming to get as near as you can to a full eight hours every night. While any kind of chemical-based sleeping aid is out, he says you can use melatonin supplements, which also naturally occur in the brain.

**THE QUESTION**

**Question:** Does brain training actually work? Should I be working out my mind just like I exercise my body?

**Answer:** Your question arrived at the perfect time, because I was able to ask Dr Bredesen that very thing for you. He is a big advocate of keeping your mind in shape, and while there are plenty of mobile phone applications designed to do just that, he also recommends traditional crosswords and sudoku puzzles to give your mind a healthy workout.

*Adam MacDougall is a former NRL player and the creator of*[*The Man Shake*](https://www.themanshake.com.au/)*. Continue the conversation*[*@adammacdougall5*](https://twitter.com/adammacdougall5?lang=en)

The Buck Institute for Research on Aging is an independent biomedical research institute that researches aging and age-related disease. The mission of the Buck Institute is to extend the healthy years of life. The Buck Institute is one of eleven centres for aging research of the Glenn Foundation for Medical Research.

### [**Buck Institute for Research on Aging** https://www.buckinstitute.org](file:///C%3A%5CUsers%5CPeter2%5CDocuments%5CAfiles%5CBetter-Management%2C%20newsletters%2C%20articles%5C%0DBuck%20Institute%20for%20Research%20on%20Aging%20%20https%3A%5Cwww.buckinstitute.org%0D)

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MoCA brain test - <https://www.mybraintest.org/dl2/moca-test-english-7-1.pdf>

Brian exercises: The Brain HQ Group -   <https://www.brainhq.com/why-brainhq/about-the-brainhq-exercises/attention/double-decision/>

Also Luminosity (author has used this exercise program for 6 years), Dakim, Cogstate

Souvenaid, a multi-vitamin drink for early stages of CD

MCT oil is most commonly extracted from **coconut oil,** can help **you** stay in the fat-burning state known as **ketosi -** Addi small amounts of MCTs to any food**s**

**Ashwagandha, an Indian herb,** provides a benefits for your **body** and brain - lower blood sugar levels, reduce cortisol, boost brain function.

The **APOE gene (see also in Dr Pedesen’s book)** provides instructions for making a protein called apolipoprotein E. This protein combines with fats (lipids) in the body to form molecules called lipoproteins. Lipoproteins are responsible for packaging cholesterol and other fats and carrying them through the bloodstream. **APOE4** is the greatest genetic risk factor for late-onset Alzheimer's disease (AD), increasing the risk of developing the disease by 3-fold in the 14% of the population that are carriers. There are no approved predictive genetic tests for this form of the condition. - <https://www.apoe4.info/wp/>

## Redimind mixture / tonic : The Clinical Study

The **International Shopping List Test**, the standard in short-term memory evaluation, was administered to 50 participants to evaluate the effectiveness of  versus placebo. Huperzine A "Compared with [other acetylcholinergic substances], HupA has better penetration through the blood-brain barrier, higher oral bioavailability, and longer duration of AChE inhibitory action. HupA has been found to improve cognitive deficits in a broad range of animal models. [...] The phase IV clinical trials in China have demonstrated that HupA significantly improved memory deficits in elderly people with benign senescent forgetfulness [...] with minimal peripheral cholinergic side effects and no unexpected toxicity."

## Mayo Clinic - Alzheimer's treatments: What's on the horizon?

## <https://www.mayoclinic.org/diseases-conditions/alzheimers-disease/in-depth/alzheimers-treatments/art-20047780>

**Despite many promising leads, new treatments for Alzheimer's are slow to emerge.**

Current Alzheimer's treatments temporarily improve symptoms of memory loss and problems with thinking and reasoning. These Alzheimer's treatments boost performance of chemicals in the brain that carry information from one brain cell to another. However, these treatments don't stop the underlying decline and death of brain cells. As more cells die, Alzheimer's disease continues to progress.

# Webmd - <https://www.webmd.com/alzheimers/guide/alzheimers-disease-treatment-overview#1> Treatments for Alzheimer's Disease

Right now, there is no cure for [Alzheimer's disease](https://www.webmd.com/alzheimers/default.htm). Once a person starts showing signs – [memory loss](https://www.webmd.com/brain/memory-loss) and problems with learning, judgment, communication, and daily life -- there aren’t any treatments that can stop or reverse them. But there are medicines that can ease some of the symptoms in some people. They can slow down how quickly the disease gets worse, and help the brain work better for longer. It’s important to talk to your doctor about which option may work best for you.

**National Institute of Aging**

# <https://www.nia.nih.gov/health/how-alzheimers-disease-treated>

# How Is Alzheimer's Disease Treated?

[Alzheimer’s disease](https://www.nia.nih.gov/health/what-alzheimers-disease) is complex, and **it is unlikely that any one drug or other intervention will successfully treat it**. Current approaches focus on helping people maintain mental function, [manage behavioural symptoms](https://www.nia.nih.gov/health/managing-personality-and-behavior-changes-alzheimers), and slow down the [symptoms](https://www.nia.nih.gov/health/what-are-signs-alzheimers-disease) of disease. Several prescription drugs are currently approved by the U.S. Food and Drug Administration (FDA) to treat people who have been diagnosed with Alzheimer’s disease. Treating the symptoms of Alzheimer’s can provide people with comfort, dignity, and independence for a longer period of time and can encourage and assist their caregivers as well. Most medicines work best for people in the early or middle stages of Alzheimer’s. For example, they can slow down some symptoms, such as memory loss, for a time. It is important to understand that none of these medications stops the disease itself.

## Dementia.org – (note: worthwhile checking site)

## Preventative Measures: As research for a dementia cure continues, taking preventative measures to decrease the risk of developing dementia becomes crucial. Certain risk factors like age or genetic susceptibility cannot be changed or controlled, but there are many other factors that increase the probability of developing dementia. Alzheimer’s disease is a form of [****dementia****](https://draxe.com/dementia/) that can rob people of the ability to think clearly, perform everyday tasks and ultimately, remember who they even are. Because the disease is so devastating, and since previous treatments failed to come up with a cure, I’m always on the lookout for Alzheimer’s natural treatment options and Alzheimer’s news, scouring the medical journals for Alzheimer’s breakthroughs.

## There’s so much we still don’t know about the human brain, but thankfully, 2016 marks a year of progress and some pretty significant Alzheimer’s breakthroughs. Let me share some of them with you. There are several theories including [****free radical damage****](https://draxe.com/fighting-free-radical-damage/), an inability to use glucose properly, vitamin deficiencies or environmental toxins. This illness affects a third of people over the age of 85 in the U.S.The good news is that there are Alzheimer’s natural treatment options that can effectively improve this condition. Recently, scientists are also uncovering major Alzheimer’s breakthroughs that may, one day, lead us to a cure.

# Alzheimer’s Natural Treatment Options & 7 Notable Breakthroughs

# Standard sensible day-to-day advice, similar to Dr Bredesen, but no diagnosis

#  by [Dr. Josh Axe, DC, DMN, CNS](https://draxe.com/about-dr-josh-axe/) April 5, 2018

## **7 Notable Alzheimer’s Breakthroughs**

#### **1. What you eat TOTALLY matters**

If you’ve spent any time at all on this website, you know my mantra: [**Food is medicine**](https://draxe.com/food-is-medicine/). It’s not hocus pocus, either. Hippocrates knew the importance of food in healing the body back in 400 B.C. when he advised people to prevent and treat diseases first and foremost by eating nutrient-packed foods. Modern science is catching up. Scientists recently found that the [**Mediterranean diet**](https://draxe.com/mediterranean-diet/) seems to be protective against Alzheimer’s disease. A UCLA study published in the American Journal of Geriatric Psychiatry found that the Mediterranean diet is one of the main lifestyle factors that seems to keep the brain from developing the toxic plaques and tangles associated with the development of Alzheimer’s disease. ([2](http://aanddjournal.net/article/S1552-5260%2815%2902037-3/fulltext))

Plaque is characterized by deposits of a toxic protein called beta-amyloid in the spaces between nerve cells in the brain. Think of tangles of knotted threads of the [tau protein](http://www.sciencemag.org/news/2018/01/alzheimer-s-protein-may-spread-infection-human-brain-scans-suggest) found within brain cells. Both are considered the key indicators of Alzheimer’s.

The new study used PET imaging to study the brain for changes and is the first to demonstrate how lifestyle factors directly influence abnormal proteins in people with subtle memory loss who have not yet been diagnosed with dementia. Healthy lifestyle factors also have been shown to be related to reduced shrinking of the brain and lower rates of atrophy in people with Alzheimer’s. ([3a](http://newsroom.ucla.edu/releases/diet-and-exercise-can-reduce-protein-build-ups-linked-to-alzheimers-ucla-study-shows))

Food staples of the Mediterranean diet include:

* fresh fruits and vegetables (especially leafy greens like spinach and kale and non-starchy veggies like eggplant, cauliflower, artichokes, tomatoes and fennel)
* [**olive oil**](https://draxe.com/olive-oil-benefits/)
* nuts and seeds (like almonds and sesame seeds used to make tahini)
* legumes and beans (especially lentils and chickpeas used to make hummus)
* herbs and spices (like oregano, rosemary and parsley)
* whole grains
* eating wild-caught fish and seafood at least twice a week
* high-quality, pasture-raised poultry, eggs, cheese, [**goat milk**](https://draxe.com/goat-milk/), and probiotic-rich kefir or yogurt consumed in moderation
* red meat consumed on special occasions or about once weekly
* plenty of fresh water and some coffee or tea
* oftentimes a daily glass of red wine

One study found the [**MIND diet**](https://draxe.com/mind-diet-plan-benefits/), a hybrid of the Mediterranean diet and DASH diet, specifically designed to help reduce cognitive decline through berries, whole grains, leafy, green vegetables, other vegetables, olive oil, poultry and fish more effectively reduced incidence of Alzheimer’s disease than the two respective diets did when followed separately. ([3b](https://www.alzheimersanddementia.com/article/S1552-5260%2815%2900017-5/abstract)) Similarly, [the ketogenic diet appears to help neurological disease like Alzheimer’s](https://draxe.com/keto-diet-food-list/). For example, in one study clinical improvement was observed in Alzheimer’s patients fed a keto diet, and this was marked by improved mitochondrial function. ([3c](https://www.ncbi.nlm.nih.gov/pubmed/19664276))

#### **2. Exercise is a potent Alzheimer’s preventer**

That same UCLA-led study also produced some robust results surrounding exercise’s brain-protecting properties. Those who were more physically active on a regular basis also had the lowest levels of tangles and plaques on the PET scans, meaning they had a much lower risk of developing Alzheimer’s disease. (2) While any type of exercise is certainly better than sitting around, if you’re time strapped, [**Burst training**](https://draxe.com/7-ideas-for-burst-training-at-home/), also known as high-intensity interval training, or HIIT, is a great option. Here are 3 [**HIIT workouts**](https://draxe.com/hiit-workouts/) to help you get started. Keep in mind, though, that we need more research on how HIIT impacts the brain. We know that it does melt away fat faster than traditional steady state cardio (and a lower BMI lowers your risk of the tangles and plaques associated with Alzheimer’s, according to the latest UCLA study). However, a previous study did find that steady state cardio creates more brain neurons compared to weight training or HIIT. ([4](http://onlinelibrary.wiley.com/doi/10.1113/JP271552/abstract)) More research is needed to see if one form of exercise is best to prevent Alzheimer’s. For now, just focus on any physical activity and getting into a healthy BMI range.

**New Scientist article – worth examining in detail.**

<https://www.newscientist.com/article/2191814-we-may-finally-know-what-causes-alzheimers-and-how-to-stop-it/>

AFTER decades of disappointment, we may have a new lead on fighting Alzheimer’s disease. Compelling evidence that the condition is caused by a bacterium involved in gum disease could prove a game-changer in tackling one of medicine’s biggest mysteries, and lead to effective treatments or even a vaccine.

Now researchers from Cortexyme and several universities have reported finding the two toxic enzymes that *P. gingivalis* uses to feed on human tissue in 99 and 96 per cent of 54 human Alzheimer’s brain samples taken from the hippocampus – a brain area important for memory (*Science Advances*, [doi.org/gftvdt](http://dx.doi.org/gftvdt)). These protein-degrading enzymes are called gingipains, and they were found in higher levels in brain tissue that also had more tau fragments and thus more cognitive decline.

**UK National Health – (note: useless)**

<https://www.nhs.uk/conditions/dementia/cure/>

There is currently no "cure" for dementia. In fact, because dementia is caused by different diseases it is unlikely that there will be a single cure for dementia.

Research is aimed at finding cures for dementia-causing diseases, such as [Alzheimer's disease](https://www.nhs.uk/conditions/Alzheimers-disease/) and [vascular dementia](https://www.nhs.uk/conditions/vascular-dementia/). Developing new medicines to treat dementia takes many years and millions of pounds. Repurposing existing drugs used for other conditions is another, often quicker, way of finding medicines to treat dementia.

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**May 2017 – UCLA research – promising! Focus on life-style changes. Along the lines of Dr Bredesen which the paper refers to, but without checking body’s chemistry.**

<https://qz.com/977133/a-ucla-study-shows-there-could-be-a-cure-for-alzheimers-disease/>

Last summer, a research group from the University of California, Los Angeles (UCLA) quietly published the [results](http://www.aging-us.com/article/100981/text) of a new approach in the treatment of Alzheimer’s disease. What they found was striking. Although the size of the study was small, every participant demonstrated such marked improvement that almost all were found to be in the normal range on testing for memory and cognition by the study’s end. Functionally, this amounts to a cure.

Acknowledging these difficulties, the researchers at UCLA opted for a different approach. Beginning from the premise that Alzheimer’s disease is a particular manifestation of a highly complex system in disarray, they sought to optimize the system by changing the inputs. Put another way, the scientists chose to set aside the molecular box which has proven so vexing, and to focus instead on the context of the box itself. Although we cannot say precisely how the intervention worked, on a cellular level, the important thing is that it did work.

The method isn’t entirely novel. Researchers have already shown that multi-faceted, comprehensive lifestyle interventions can significantly improve outcomes in cardiovascular disease, diabetes and hypertension. But it’s difficult for these approaches to gain traction for two reasons. First, these protocols are more challenging than simply taking a pill at bedtime. Patients need ongoing education, counselling and support to effect meaningful change. And second, the pharmaceutical mode of treatment is deeply embedded within our current medical system. Insurance companies are set up to pay for medication, not lifestyle change; and physicians are taught pharmacology, not nutrition.

<https://www.naturalnews.com/2019-01-14-cure-for-alzheimers-disease-found-in-our-synapses.html>

**This article has several similarities to Dr Bredesen’s**

# The CURE for Alzheimer’s Disease can be found in our synapses

Monday, January 14, 2019 by: [S.D. Wells](https://www.naturalnews.com/author/sdwells)

## Understanding the neuroscience of Alzheimer’s and Parkinson’s shows us the cause and the cure at the same time

The point of connection of neurons is called a synapse, and that’s where neurotransmitters are released and communication happens in the brain. This is where we experience all of our senses and engage in thought processes, including critical thinking and memory. This is also exactly where dementia happens.

The synapse is where neurons release hormones, glutamates, and small peptides called amyloid beta. The amyloid beta are the brain’s “trash” and a prime factor involved in Alzheimer’s disease, functioning as the main component of plaques that cling to each other and clog up the neural pathway. These are the plaques found in the brains of Alzheimer’s patients.

Normally, these amyloid plaques are swept out of the neural pathway (like trash) by the “custodians of the brain” called microglea. These amazing microglea are the brain’s own immune cells and are the answer to beating brain diseases. Scientists recently discovered through [sophisticated experiments](http://brainblogger.com/2012/01/06/mighty-microglia-the-brains-immune-cells-could-be-the-key-to-treating-brain-diseases/) that these cells constantly search for brain damage, like a perpetually-running computer virus scan, running surveillance for different levels of damage. The microglea are literally capable of eating infected and damaged cells before infection spreads, while clearing out “debris” from dying cells.

Diseases of dementia therefore begin when amyloid beta begins to accumulate, because too much is released, overwhelming the microglea, and leaving waste in the neural pathways, blocking communication. The synapse piles up with plaques (trash and waste) that become sticky and bind to themselves (think of animal fat clogging your sink drain).

At a certain tipping point, when the body and brain have created too much “trash” for too long, creating massive inflammation and tangles, the microglea become overwhelmed and enter a hyper-mode, where they actually begin attacking healthy cells. Scientists believe the microglea may even, at the tipping point, begin clearing away the synapses themselves. Get it? The cure lives in keeping amyloid plaques from reaching the “tipping point.” Here’s how you do that.

## Stop consuming foods that create plaques in the brain – so your brain’s “custodians” can clear out the sticky trash that blocks your synapses

Amyloid plaque accumulation may never be “cured” with a chemical drug or vaccine, but that doesn’t matter, because you can cure the problem yourself. Are you ready to start taking your preventative medicine? It’s not very difficult you know. Let’s break it down to its simplest form, then you decide if you can “pull it off.”

You wouldn’t pick up a poisonous snake just to see if it bites you, and then start searching the internet for the anecdote, would you? You wouldn’t pick some poison ivy and rub it on your skin on purpose, would you? If you were severely allergic to peanuts, you certainly wouldn’t eat a handful just to see what happens. That’s just common sense.

So what if you knew what caused dementia, would you stop eating it? Guess what. Now is the time to stop marching for the cure and start living it, because knowledge is power. Now get this.

White foods are known to cause [excess plaque build-up](https://www.alzheimers.net/foods-that-induce-memory-loss/) in the brain, leading to dementia. These white foods include white bread, white flour, white rice (except basmati, which is naturally white), white pasta, and white sugar. Stop eating bleached food.

Processed foods and meats cause excess plaque in the synapses, fuelling dementia. Avoid processed cheeses (think American cheese especially here), and processed meats, like sausages, bacon, hot dogs, and cold cuts (especially smoked deli meats), and even beer. Nitrosamines in smoked meats cause the liver to produce fats that are toxic to the brain.

Stop eating foods that contain diacetyl, a chemical commonly found in microwave popcorn. Diacetyl increases amyloid plaques in the brain.

## Animal fat and canola oil coagulate in your blood and create tangles of plaque in the brain

You’ll hear it time and time again, that a plant-based diet cures almost every preventable disease and disorder known to humans. It’s true. If you’re a heavy meat eater, your body is struggling to process all that animal fat, creating heart and brain “trash” that your body’s “janitors” just can’t sweep away fast enough.

If you think organic or “expeller pressed” canola oil means that the oil doesn’t coagulate in your body, you’d be dead wrong. After about six weeks, any canola oil that your body hasn’t cleared out [looks like a sticky glue](https://www.naturalnews.com/2018-01-02-the-most-popular-toxic-food-ingredient-in-the-united-states-canola-oil.html) you could use to bond cement. Think of all that “trash” blocking your synapses and causing dementia, because that’s exactly what happens.

Did you know that in the U.S. alone, Alzheimer’s care already costs $2 billion a year (one out of every five Medicare dollars)? Dementia kills more people than cancer. Did you know that? Sure, Big Pharma will tell you Alzheimer’s and Parkinson’s are not preventable, but both are, and the cure lives in prevention. You may begin now.

<https://www.sciencealert.com/new-alzheimer-s-treatment-fully-restores-memory-function>

# New Alzheimer’s Treatment Fully Restores Memory Function

BEC CREW

18 MAR 2015

Australian researchers have come up with a non-invasive ultrasound technology that clears the brain of neurotoxic amyloid plaques - structures that are responsible for memory loss and a decline in cognitive function in Alzheimer’s patients.

Publishing in [Science Translational Medicine](http://stm.sciencemag.org/content/7/278/278ra33), the team describes the technique as using a particular type of ultrasound called a focused therapeutic ultrasound, which non-invasively beams sound waves into the brain tissue. By oscillating super-fast, these sound waves are able to gently open up the blood-brain barrier, which is a layer that protects the brain against bacteria, and stimulate the brain’s microglial cells to activate. Microglial cells are basically waste-removal cells, so they’re able to clear out the toxic beta-amyloid clumps that are responsible for the worst symptoms of Alzheimer’s.

See also <https://stm.sciencemag.org/content/7/278/278ra33>

<https://www.sciencedaily.com/news/mind_brain/dementia/>

# An alternate theory for what causes Alzheimer's disease

*Date:*

August 12, 2019, *Source:* University of California - Riverside

*Summary:*

Alzheimer's disease, the most common cause of dementia among the elderly, is characterized by plaques and tangles in the brain, with most efforts at finding a cure focused on these abnormal structures. But a research team has identified alternate chemistry that could account for the various pathologies associated with the disease.

"The dominant theory based on beta-amyloid build-up has been around for decades, and dozens of clinical trials based on that theory have been attempted, but all have failed," said Ryan R. Julian, a professor of chemistry who led the research team. "In addition to plaques, lysosomal storage is observed in brains of people who have Alzheimer's disease. Neurons -- fragile cells that do not undergo cell division -- are susceptible to lysosomal problems, specifically, lysosomal storage, which we report is a likely cause of Alzheimer's disease." "Long-lived proteins become more problematic as we age and could account for the lysosomal storage seen in Alzheimer's, an age-related disease," Julian said. "If we are correct, it would open up new avenues for treatment and prevention of this disease."

<https://www.sciencedaily.com/releases/2019/09/190916101856.htm>

# Alzheimer's disease risk gene APOE4 impairs function of brain immune cells

September 16, 2019, *Source:*University of Eastern Finland

*Summary:*

A study carried out with a new human stem cell-derived model reveals that the most prevalent genetic risk factor of Alzheimer's disease (AD), apolipoprotein E4 (APOE4), impairs the function of human brain immune cells, microglia. These findings pave the way for new, effective treatment approaches for AD.

APOE4 is the strongest genetic risk factor for Alzheimer's disease. Apolipoprotein, APOE, plays a critical role in the metabolism of lipids, such as cholesterol, and contributes to repairing neuronal damage in the brain. APOE is present in humans in three isoforms, and genetics determines which forms an individual carries. Only the APOE4 form predisposes for Alzheimer's disease, and over half of patients carry this form. In humans, the APOE gene is abundantly expressed in microglia, but its role specifically in these cells is poorly understood.

**Harvard Medical School**

<https://www.health.harvard.edu/mind-and-mood/protecting-against-cognitive-decline>

While there's currently no treatment that can prevent or cure dementia, researchers have identified some factors that may help protect you from cognitive decline.

Exercise offers an impressive array of health benefits. It helps prevent heart disease and type 2 diabetes; lowers the risk for high blood pressure, colon cancer, and breast cancer; and helps relieve insomnia, anxiety, and depression. In addition, it may help ward off cognitive decline and dementia. Plus, some studies have shown that engaging in a program of regular exercise improved cognitive function in people who already had memory problems. Exercise may be particularly advantageous for people who carry the APOE4 gene variant, which makes people more susceptible to Alzheimer's.

### A Mediterranean-style diet

### Alcohol There is some evidence that moderate consumption of alcohol reduces the risk for cognitive decline and dementia. A study in JAMA, for example, found that people over age 65 who drank up to one alcoholic beverage a day had about half the risk as nondrinkers over five to seven years. Another study reported that resveratrol, a compound in red wine, broke down beta-amyloid (abnormal deposits of protein associated with Alzheimer's disease) in laboratory experiments, suggesting that red wine in particular may be protective, but further study is needed. In the meantime, experts do not recommend drinking alcohol to fend off Alzheimer's disease or cognitive decline. However, experts do not recommend drinking alcohol to prevent cognitive decline. If you enjoy an occasional alcoholic beverage, you should limit your consumption to no more than two drinks a day if you are a man or one drink if you are a woman. In the JAMA study, heavy drinkers—defined as more than four drinks per day or 14 per week for men and more than three drinks per day or seven per week for women—had a 22% higher Alzheimer's risk than the non-drinkers.

### Mental stimulation - Many researchers believe that education level is less important in maintaining a healthy brain than the habit of staying mentally active as you age. In one study, mentally intact people in their 70s and 80s were asked how often they did six activities that required active mental engagement—reading, writing, doing crossword puzzles, playing board or card games, engaging in group discussions, and playing music. In the following five years, those who placed in the highest third in terms of how often they engaged in mentallystimulating activities were half as likely to develop mild cognitive impairment as those in the lowest third. An earlier report found a similar link between brain-stretching activities and a lower risk of Alzheimer's.

### To learn more about MCI, review the online guide from Harvard Medical School, [Understanding Mild Cognitive Impairment.](https://www.health.harvard.edu/mind-and-mood/understanding-mild-cognitive-impairment)

NCBI National Centre for Biological Information

<https://www.ncbi.nlm.nih.gov/pubmed/29658744>

[Psychol Aging.](https://www.ncbi.nlm.nih.gov/pubmed/29658744) 2018 Mar

# When does cognitive decline begin? A systematic review of change point studies on accelerated decline in cognitive and neurological outcomes preceding mild cognitive impairment, dementia, and death.

[Karr JE](https://www.ncbi.nlm.nih.gov/pubmed/?term=Karr%20JE%5BAuthor%5D&cauthor=true&cauthor_uid=29658744)1, [Graham RB](https://www.ncbi.nlm.nih.gov/pubmed/?term=Graham%20RB%5BAuthor%5D&cauthor=true&cauthor_uid=29658744)1, [Hofer SM](https://www.ncbi.nlm.nih.gov/pubmed/?term=Hofer%20SM%5BAuthor%5D&cauthor=true&cauthor_uid=29658744)1, [Muniz-Terrera G](https://www.ncbi.nlm.nih.gov/pubmed/?term=Muniz-Terrera%20G%5BAuthor%5D&cauthor=true&cauthor_uid=29658744)2.

### [Author information](https://www.ncbi.nlm.nih.gov/pubmed/29658744)

### Abstract

Older adults who ultimately develop dementia experience accelerated cognitive decline long before diagnosis. A similar acceleration in cognitive decline occurs in the years before death as well. To evaluate preclinical and terminal cognitive decline, past researchers have incorporated change points in their analyses of longitudinal data, identifying point estimates of how many years prior to diagnosis or death that decline begins to accelerate. The current systematic review aimed to summarize the published literature on preclinical and terminal change points in relation to mild cognitive impairment (MCI), dementia, and death, identifying the order in which cognitive and neurological outcomes decline and factors that modify the onset and rate of decline. A systematic search protocol yielded 35 studies, describing 16 longitudinal cohorts, modelling change points for cognitive and neurological outcomes preceding MCI, dementia, or death. Change points for cognitive abilities ranged from 3-7 years prior to MCI diagnosis, 1-11 years prior to dementia diagnosis, and 3-15 years before death. No sequence of decline was observed preceding MCI or death, but the following sequence was tentatively accepted for Alzheimer's disease: verbal memory, visuospatial ability, executive functions and fluency, and last, verbal IQ. Some of the modifiers of the onset and rate of decline examined by previous researchers included gender, education, genetics, neuropathology, and personality. Change point analyses evidence accelerated decline preceding MCI, dementia, and death, but moderators of the onset and rate of decline remain ambiguous due to between-study modelling differences, and coordinated analyses may improve comparability across future studies. (PsycINFO Database Record.

**Medical News Today**

<https://www.medicalnewstoday.com/articles/324457.php>

# Study finds new cognitive decline mechanism in Alzheimer's

Published Friday 15 February 2019

People with Alzheimer's disease experience poor blood flow to the brain, which affects cognitive function. A new study conducted in a mouse model has finally uncovered the reason behind this reduced blood flow.

Reduced blood flow to the brain contributes to Alzheimer's, but what mechanism leads to this vascular problem in the first place? For a while now, researchers have been aware that [Alzheimer's disease](https://www.medicalnewstoday.com/articles/159442.php) goes hand in hand with vascular dysfunction, and reduced blood flow to the brain, in particular. However, it is only recently that investigators have begun to focus their efforts on understanding just how and why poor vascular health can contribute to cognitive decline in this type of [dementia](https://www.medicalnewstoday.com/articles/142214.php).

A [study](https://www.alzheimersanddementia.com/article/S1552-5260%2818%2933495-2/fulltext) published last month in Alzheimer's and Dementia, the journal of the Alzheimer's Association, calls vascular dysfunction "the disregarded partner of Alzheimer's disease." It argues that researchers must first fully understand all the factors involved in the pathology of this type of dementia before they can develop a pluripotent treatment for it.

"Individualized, targeted therapies for [Alzheimer's disease] patients will be successful when the complexity of [this condition's] pathophysiology is fully appreciated," the study authors write.

Now, in a study in mice, a team of investigators from Cornell University in Ithaca, NY has identified a mechanism — tied to poor blood flow to the brain — that directly contributes to cognitive decline.

**TED video** - <https://www.youtube.com/watch?v=twG4mr6Jov0>

# What you can do to prevent Alzheimer's | Lisa Genova

•Published on May 20, 2017

Alzheimer's doesn't have to be your brain's destiny, says neuroscientist and author of "Still Alice," Lisa Genova.

**Dietitian Amylee Amos talk**

# ALZHEIMER’S DIET,” APOE 4 + THE BREDESEN PROTOCOL

March 13, 2018 By: Whitney E. RD. 25 min video

We’re talking to brain health expert Amylee Amos MS, RDN about The Bredesen Protocol, a clinically-tested “Alzheimer’s diet” and how it can potentially slow and even reverse cognitive decline or prevent disease in APOE4 susceptible individuals.

<https://www.whitneyerd.com/2018/03/alzheimers-diet-bredesen-protocol.html>

Note: their approaches to food a very similar to heart program protocol, but worthwhile focus on Dr Bredesen’s protocol.

# 7th BioCeuticals Research Symposium7tBioCeuticals Research Symposium

### **Date:***Fri 03 May 19 - Sun 05 May 19***Location:***488 George St SYDNEY NSW Australia 2000*

### <https://www.bioceuticals.com.au/education/event/7th-BioCeuticals-Research-Symposium>

### **This year, our keynote speakers include:**

**Dr Dale Bredesen,** internationally recognised expert in the mechanisms of neurodegenerative diseases, who’s research has shown that Alzheimer’s disease stems from an imbalance in nerve cell signaling and has opened the door to a new therapeutic approach. Dr Bredesen will cover the mechanisms underlying the neurodegenerative process, and the translation of this knowledge into effective therapeutics for Alzheimer’s disease and other neurodegenerative conditions.

**Dr Jay Lombard,** internationally acclaimed neurologist, author, and keynote speaker who creates solutions for brain health and intractable neurological disorders. Dr Lombard integrates biological, psychological, and existential components in his holistic treatment approach. His discoveries have been regarded by key opinion leaders as fundamentally shifting the paradigm of psychiatric medicine.

**Ms Amanda Archibald**, dietitian, nutritionist and public health advocate, who has pioneered the combination of the science of nutrigenomics with the culinary arts. Amanda will draw particular attention to the mechanisms of food-gene relationships related to the core issues known to affect long-term health – inflammation, oxidative stress, blood sugar and fats, and gut health – while emphatically encouraging taste and enjoyment as culinary treasures in the process.

**Dr Brandon Brock**, chiropractic neurologist, family nurse practitioner and nutritionist, has a passion for providing easy-to-comprehend skills that can be utilised in a clinical setting. He received the most outstanding functional neurology teacher of the year award from the ACA Council of Neurology for five years and twice from IAFNR. Dr Brock will present his unique and integrated understanding of functional neurology blending nutrition, pharmacology, immunology and endocrinology to provide a comprehensive approach to clinical presentations.

# TED talk. 2015

# Note: compelling viewing

<https://www.youtube.com/watch?v=tkIg-SxPzTA> – 8 min video

# Alzheimer’s Is Not Normal Aging — And We Can Cure It | Samuel Cohen | TED Talks

•Published on Oct 17, 2015

More than 40 million people worldwide suffer from Alzheimer’s disease, and that number is expected to increase drastically in the coming years. But no real progress has been made in the fight against the disease since its classification more than 100 years ago. Scientist Samuel Cohen shares a new breakthrough in Alzheimer’s research from his lab as well as a message of hope. “Alzheimer’s is a disease,” Cohen says, “and we can cure it.”

**Collective Evolution**

<https://www.collective-evolution.com/2018/04/27/82-year-old-woman-with-dementia-gets-her-memory-back-after-changing-her-diet/>

Article text includes:

A change in diet, which was comprised of high amounts of blueberries and walnuts, has proven to have had a strong impact on Sylvia’s condition that her recipes are now being shared by the Alzheimer’s Society.

Recently, an 82-year-old woman who suffered from dementia, who couldn’t recognize her own son has miraculously got her memory back after changing her diet.

When his mother’s condition became so severe that for her own safety she had to be kept in the hospital, Mark Hatzer almost came to terms with losing another parent.

Sylvia had lost her memory and parts of her mind, she had even phoned the police once accusing the nurse who were caring for her of kidnap.

A change in diet, which was comprised of high amounts of blueberries and walnuts, has proven to have had a strong impact on Sylvia’s condition that her recipes are now being shared by the Alzheimer’s Society.

Sylvia also began incorporating other health foods, including broccoli, kale, spinach, sunflower seeds, green tea, oats, sweet potatoes and even dark chocolate with a high percentage of cacoa. All of these foods are known to be beneficial for brain health.

Mark and Sylvia devised to diet together after deciding that the medication on it’s own was not enough, they looked into the research showing that rates of dementia are much lower in mediterranean countries and copied a lot of their eating habits.

*“In certain countries Alzheimer’s is virtually unheard of because of their diet.*

*“Everyone knows about fish but there is also blueberries, strawberries, Brazil nuts and walnuts – these are apparently shaped like a brain to give us a sign that they are good for the brain.”*

*There were also some cognitive exercises that Mark and his mother would do together like jigsaw puzzles crosswords and meeting people in social situations, Sylvia would also exercise by using a pedaling device outfitted for her chair.*

In an article titled, [**Strong evidence linking Aluminum to Alzheimer’s**](http://www.greenmedinfo.com/blog/%22), recently published in The Hippocratic Post website, Exley explained that:

***“We already know that the aluminium content of brain tissue in late-onset or sporadic Alzheimer’s disease is significantly higher than is found in age-matched controls. So, individuals who develop Alzheimer’s disease in their late sixties and older also accumulate more aluminium in their brain tissue than individuals of the same age without the disease.***

**The NeuroDevelopment Center,** Providence, Rhone Island, USA

<https://www.neurodevelopmentcenter.com/psychological-disorders/cognitive-decline/cognitive-decline-prevention/>

## You CAN stay sharp as you age: Options for cognitive decline prevention and treatment

The stakes are high. Research clearly shows gradual mental decline with aging, affecting:

* memory
* thinking skills
* concentration
* problem solving

But research also shows that you can stay sharp as you age. A rapidly growing number of studies in the neuroscience of brain aging are showing us new methods and techniques to help you maintain your mental fitness as you age. Cognitive decline prevention is coming into prominence.

At the NeuroDevelopment Center, we are devoted to helping you maintain your mental fitness as you age.  Our staff professionals comb the cutting edge of this science to uncover new knowledge and techniques to help you get sharp and stay sharp. Everyday, new studies are published that point the way toward cognitive decline prevention. We find them and translate the complexities of advanced brain science into practical approaches you can use to stay sharp.

The research is increasingly clear: mental fitness protects you from the effects of age related cognitive decline and from the effects of Alzheimer’s Disease and other forms of dementia. Mental fitness is a form of cognitive decline prevention.

## Choose from these options to prevent or treat age related decline

Cogmed working memory training is a research-supported way to increase concentration and improve working memory. With this online brain training program, there are a series of engaging working memory games and challenges. The challenges, like most popular video games, get progressively harder as your skills improve. Cogmed Working Memory Training provides scientifically designed working memory exercise and practice. Just like physical exercise, it leads to improved skill and capacity. Repeated studies show lasting gains. [Learn more](https://neurodevelopmentcenter.com/treatment/cogmed/).

Neurofeedback or EEG biofeedback is a research supported way to train the brain to function better. Advanced neuroscience has shown that feedback-guided brain training can result in measurable change in brain networks. With neurofeedback, you can activate and exercise brain networks devoted to memory, attention, and problem solving. Early research is showing improved mental function and even some benefit for Alzheimer’s disease.  [Learn more.](https://neurodevelopmentcenter.com/psychological-disorders/cognitive-decline/neurofeedback-for-cognitive-decline/)

## Stop stress from killing your memory cellls with HRV Biofeedbackheart rate variability

Stress contributes to mental decline. The stress hormone cortisol destroys memory neurons. Research shows that heart rate variability (HRV) biofeedback is an effective way to reduce stress.

Your heart beat is a great “stress-o-meter”. When you are stressed or “revved up” or in a negative emotional state, and your nervous system arousal is high, your heart beats in lockstep. The amount of time between heart beats changes very little. When you are calm and stable, and nervous system arousal is lower, the beat to beat interval becomes more flexible.

HRV biofeedback training provides an easy and very concrete way to learn how to reshape these heart rate rhythms for increased calm and stability. A simple sensor on your ear or finger picks up your heart beat. The computer analyzes the interval between heart beats. During HRV biofeedback sessions we will help you learn to relax your muscles and your mind, and learn to slow and regulate your breathing. As you learn, your heart rate variability will increase. The image above shows a session in progress, with the more regular signal on the right half of the top section showing increased HRV. Once you learn the techniques in sessions in our office, you can also use inexpensive software at home to build your ability to recognize when you are bothered and rapidly regain your calm.

## NeuroTracker

NeuroTracker is a new technology that isolates and trains attention and working memory, without medication! Several early studies have shown significant improvements in attention after seven 15-minute sessions of training with Neurotracker. The training is safe and non-invasive. It engages you in a series of 3D exercises that help improve attention, focus, and awareness. Neurotracker is used by elite athletes around the world to improve their attention and movement tracking while in play. Teams from the NFL, NBA, NHL and EPL employ Neurotracker as do many Olympic and college athletes. New research is showing that NeuroTracker can help you stay sharp as you age. [Learn more.](https://neurotracker.net/)

**Note: this article presents some useful information – worth examining…**

**Life Extension**

**Age-related cognitive decline**

<https://www.lifeextension.com/Protocols/Neurological/Age-Related-Cognitive-Decline/Page-01>

All aging humans will develop some degree of decline in cognitive capacity, usually including the following symptoms:

* forgetfulness
* decreased ability to maintain focus
* decreased problem solving capacity

If left unchecked, symptoms oftentimes progress into more serious conditions, such as dementia and depression, or even Alzheimer’s disease.

Fortunately, proactive lifestyle changes, cognitive training, and nutritional interventions such as **phosphatidylserine**and **glyceryl phosphoryl choline** have been shown to decrease the rate of intellectual decay and potentially reverse age-related cognitive decline.

## Causes of Age-Related Cognitive Decline

Many factors contribute to age-related cognitive decline:

* Oxidative stress and free radical damage
* Chronic low-level inflammation
* Declining hormone levels like estrogen, testosterone, DHEA, pregnenolone
* Inner arterial lining (endothelium) dysfunction
* Insulin resistance
* Excess body weight
* Suboptimal nutrition
* Loneliness, lack of social network, and high stress

## Dietary and Lifestyle Changes

Several dietary and lifestyle changes can help reduce age-related cognitive decline:

* Switch from a western diet high in simple sugars and saturated fats to a Mediterranean diet high in mono- and polyunsaturated omega-3 fats, fiber, and polyphenols
* Caloric restriction may improve learning and memory
* Cognitive stimulation and training, including playing chess and speaking more than one language, can enhance cognitive reserve and convey protection against loss of brain function
* Exercise is known to increase levels of brain-derived neurotrophic factor, which can lead to enhanced cognitive function
* Moderate alcohol consumption (up to 2 drinks/day) and caffeinated coffee consumption (~3 cups/day) may convey protection against cognitive decline

## Integrative Interventions

* **Fish Oil:**Daily omega-3 supplementation was independently associated with a dramatic reduction in cognitive decline over a 1.5-year period in an aging study population.
* **Phosphatidylserine:**Human clinical trials have found that supplementing with phosphatidylserine improves cognitive function in aging subjects with cognitive impairment.
* **Glyceryl Phosphoryl Choline (GPC):**Patients taking GPC showed neurological improvement and relief of clinical symptoms of chronic cerebral deterioration that was superior or equivalent to that obtained with prescription drugs.
* **Acetyl-L-carnitine:**A meta-analysis of data from over 21 studies shows that supplementation with acetyl-L-carnitine improves cognitive deficits observed during aging and pathological brain deterioration.
* **Huperzine A:** Patients with Alzheimer’s disease improved their scores on standard cognitive tests after supplementing with huperzine A.

Cognitive function appears to peak around age 20 and diminish steadily over the remaining years of life.1,2 With life expectancies increasing dramatically in the last century, cognitive decline and dementia have become major contributors to disability and mortality.3,4

Aging is associated with gradual changes in the brain that slow and reduce its function. As a result of these changes, it is common for elderly people, even those without neurological disease, to find it takes longer to perform mental tasks and to experience diminished memory, attention, and abilities to learn, reason, and solve problems.2 Although some cognitive decline occurs during normal aging, its rate of progression is affected by lifestyle, environmental, and genetic factors,5 some of which may be modifiable.1,6

Aging is a complex process, and age-related conditions like cognitive decline are multifactorial. Some factors that likely contribute to age-related cognitive decline are:

* stem cell senescence,
* brain oxidative stress and mitochondrial dysfunction,
* neuroinflammation (inflammation in the brain),
* circadian rhythm and metabolic disturbances,
* vascular dysfunction,
* abnormal protein accumulation,
* disordered homocysteine metabolism,
* changing hormone levels, and
* epigenetic factors—changes in the way genes are expressed.

These same mechanisms also appear to contribute to dementia and neurodegenerative diseases like Alzheimer disease and Parkinson disease.1

Currently, much is known about lifestyle factors that work together to promote healthy brain aging, such as eating a nutrient-dense diet (eg, Mediterranean-style diet), being physically active, reducing stress, getting adequate sleep, and regularly engaging in mentally and socially stimulating activities.1,4,6 In addition, a number of integrative interventions have been identified as having protective effects on brain function.7,8

This protocol will review many underlying factors that contribute to cognitive decline, and describe several novel medical strategies, lifestyle and dietary habits, and integrative interventions that can support healthy cognitive function and brain health throughout life.

## Risk Factors Associated with Cognitive Decline

Older age is the number one risk factor for age-related cognitive decline, as well as mild cognitive impairment and dementia. Women have a higher risk of dementia than men. Furthermore, a number of potentially modifiable risk factors for late-life dementia have been identified, many of which have their strongest impact on late-life cognitive function when they occur in midlife.21-23 These risk factors include:

* Sedentary lifestyle21,22
* Low educational attainment21,22
* Smoking21,22
* Obesity21,22
* Insulin resistance and type 2 diabetes21,22
* Hypertension21,22
* High cholesterol levels21,22
* Chronic kidney disease21
* Atrial fibrillation (a type of arrhythmia)21
* Cardiovascular disease24
* Depression21
* Sleep disorders25
* Sleep apnea26
* High homocysteine levels27
* Heavy metal toxicity28

Although there are currently no pharmacologic interventions specifically for age-related cognitive decline, medical approaches to underlying issues such as vascular disease and systemic inflammation may help prevent progressive cognitive loss. For example, the use of certain antihypertensive medications to lower high blood pressure may slow cognitive decline and prevent dementia; however, they also pose a risk of reducing cerebrovascular blood flow and causing more cognitive harm.105,106

A number of observational studies have noted that patients using cholesterol-lowering drugs called statins (such as simvastatin [Zocor] and atorvastatin [Lipitor]) have a lower risk of mild cognitive impairment and dementia than those not using statins107; however, other studies and case reports suggest statins may impair cognitive function in some individuals,108 and other studies have found no benefit.109 Large randomized controlled trials have found statins had no impact on cognitive decline or dementia risk.108,110-114

While once thought to hold promise for patients with mild cognitive impairment, it now seems that anti-dementia drugs, like the acetylcholinesterase inhibitors donepezil (Aricept), galantamine (Razadyne), tacrine (Cognex), and rivastigmine (Exelon), have neither been shown to restore cognitive function nor protect against dementia in this population.115,116 The use of biological and genetic markers in the future may help researchers identify those most likely to benefit from certain drug therapies.115

**Note: there is a long list of life-style, food, vitamins and medications suggested, and 472 references at the end of the article.**

**Alzheimers Association**

<https://www.alz.org/alzheimers-dementia/what-is-dementia/related_conditions/mild-cognitive-impairment>

**Treatment and outcomes**

Currently, there are no medications approved by the Food and Drug Administration (FDA) for the treatment of MCI. Drugs approved to treat symptoms of Alzheimer's disease have not shown any lasting benefit in delaying or preventing progression of MCI to dementia.

More research is needed on the biological changes associated with normal aging, MCI and Alzheimer’s and other dementias to better understand the causes of and risk factors for MCI and the prognosis for those with the condition.

Individuals who have been diagnosed with MCI should be re-evaluated every six months to determine if symptoms have progressed. Stay informed about research investigating MCI, Alzheimer’s and other dementias.

UC Berkeley School of Public Health

<https://www.healthandwellnessalerts.berkeley.edu/topics/warning-signs-of-mild-cognitive-impairment/>

Article repeating much of the standard views.

World Health Organisation (WHO) 2017

<https://www.who.int/mental_health/neurology/dementia/guidelines_risk_reduction/en/>

### Overview

The *WHO Guidelines on risk reduction of cognitive decline and dementia* provide evidence-based recommendations on lifestyle behaviours and interventions to delay or prevent cognitive decline and dementia.

Worldwide, around 50 million people have dementia and, with one new case every three seconds, the number of people with dementia is set to triple by 2050. The increasing numbers of people with dementia, its significant social and economic impact and lack of curative treatment, make it imperative for countries to focus on reducing modifiable risk factors for dementia. Action area 3 of the *Global action plan on the public health response to dementia 2017–2025* is risk reduction.

These WHO Guidelines are an important tool for health care providers as well as governments, policy-makers and other stakeholders to strengthen their response to the dementia challenge.

A long document detailing WHO’s approach and plans:

<https://apps.who.int/iris/bitstream/handle/10665/259615/9789241513487-eng.pdf;jsessionid=6850637489F26490003AD56666190CB9?sequence=1>

..Vision of a world in which dementia is prevented and people. The goal of the global action plan is to improve the lives of people with dementia, their carers and families, while decreasing the impact of dementia on them as well as on communities and countries.

**Note: little mention of finding out how to delay or prevent dementia.**

**---------------------------------------------------**

**Emory University – ADRC Research Centre**

<http://alzheimers.emory.edu/healthy_aging/cognitive-skills-normal-aging.html>

A commonly held misconception is that aging results in an inevitable loss of all cognitive abilities and that nothing can be done to halt this decline.  Research, however, does not support these claims.  While certain areas of thinking do show a normal decline as we age, others remain stable.  Moreover, interventions may actually slow some of the changes that do occur.

A previous view was that as we age, brain cells inevitably die off and are not replaced.  This concept led to the belief that nothing could be done to alter the inevitable.  We now know that certain interventions can sharpen cognitive processes.  These include:

* Reducing Stress:  Researchers have found that high stress levels impair learning and memory in both animals and humans.  Strategies to reduce stress such as exercise may be beneficial.
* Maintaining Good Health:  Regular visits to the doctor are critical to make sure that medical conditions which can themselves impair thinking are under good control.  In addition, possible interactions among medications should be evaluated by letting your physician know all of the medications you are taking, even if not prescribed by that particular doctor.  A diet rich in fruits and vegetables containing antioxidants such as blueberries, strawberries, and broccoli as well as certain fats such as olive oil may be neuroprotective.
* Keeping Mentally Stimulated:  Studies have found that engaging in challenging cognitive tasks can protect against age-related declines in thinking and the risk of developing Alzheimer’s disease.  It is important to keep oneself stimulated through activities such as playing bridge, reading, and attending adult education courses.
* Using Active Strategies:  There is evidence that some of the difficulties in storing new memories are due to the fact that older persons do not spontaneously use strategies to encode this information.  When they do, age differences are weakened.  In addition, older adults demonstrate good recognition of new information when they are helped with cues to jog their memory.  Strategies that can be helpful to facilitate memory include following a routine (e.g., always putting one’s keys in the same place), using external techniques (e.g., a calendar, a pill box), and taking more time to actively process new information (e.g., when introduced to someone, pay extra attention and try to come up with an association to recall that person’s name)

Wikipedia

<https://en.wikipedia.org/wiki/Dementia>

There is no known [cure](https://en.wikipedia.org/wiki/Cure) for dementia.[[2]](https://en.wikipedia.org/wiki/Dementia#cite_note-WHO2014-2) [Cholinesterase inhibitors](https://en.wikipedia.org/wiki/Acetylcholinesterase_inhibitor) such as [donepezil](https://en.wikipedia.org/wiki/Donepezil) are often used and may be beneficial in mild to moderate disorder.[[7]](https://en.wikipedia.org/wiki/Dementia#cite_note-Kav2007-7)[[16]](https://en.wikipedia.org/wiki/Dementia#cite_note-Bir2006-16)[[17]](https://en.wikipedia.org/wiki/Dementia#cite_note-17) Overall benefit, however, may be minor.

Agency for Healthcare Research and Quality U.S. Department of Health and Human Services

Preventing Alzheimer’s Disease and Cognitive Decline

<https://www.ahrq.gov/downloads/pub/evidence/pdf/alzheimers/alzcog.pdf>

Conclusions: The current research on the list of putative risk or protective factors is largely inadequate to confidently assess their association with AD or cognitive decline. Further research that addresses the limitations of existing studies is needed prior to be able to make recommendations on interventions.

**FamilyDoctor.org**

<https://familydoctor.org/nourish-your-brain/>

## What is cognitive decline?

Cognitive decline is when your brain doesn’t work as well as it used to. For example, a person who is experiencing cognitive decline may have trouble learning, using language, or remembering things.

Some cognitive decline is a normal part of growing older. Cognitive decline that happens quickly or that affects day-to-day activities is called [**dementia**](https://familydoctor.org/condition/dementia/). A head injury, a [**stroke**](https://familydoctor.org/condition/stroke/), or disease (for example, [**Alzheimer’s disease**](https://familydoctor.org/condition/alzheimers-disease/)) can damage brain cells and lead to dementia.

As your body gets older, so does your brain. You can’t stop normal cognitive decline, just as you can’t stop other parts of normal aging. However, you can maintain your body and brain health by making healthy choices about your lifestyle, diet, and exercise. Healthy choices can also help prevent disease.

## Path to improved health

There isn’t one specific diet that is best for brain health, but eating healthy is important for your overall health. Choosing foods that nourish your body and brain can help prevent or delay health problems, including conditions that increase your risks for dementia.

### **Tips to nourish your body and brain**

* **Manage your weight.**Studies show that [**obesity**](https://familydoctor.org/condition/obesity/), [**diabetes**](https://familydoctor.org/condition/diabetes/), [**high blood pressure**](https://familydoctor.org/condition/high-blood-pressure/), and high [**cholesterol**](https://familydoctor.org/condition/cholesterol/) can all increase your risk for dementia. To lose weight and keep it off, avoid short-term or [**“fad” diets**](https://familydoctor.org/nutrition-weight-loss-need-know-fad-diets/). Instead, adopt a healthy way of thinking about and eating food.
* **Eat fruits, vegetables, and whole grains.**A diet that includes lots of fruits, vegetables, and whole grains can reduce your risk for chronic diseases, including heart disease, diabetes, and [**cancer**](https://familydoctor.org/condition/cancer/). These same foods may also help protect brain function. The antioxidants in leafy greens, dark-skinned vegetables, and cruciferous vegetables (broccoli, cabbage, and turnips) may be especially protective. Vegetables including beets, broccoli, Brussels sprouts, cauliflower, eggplant, kale, red bell peppers, romaine lettuce, and spinach are good choices.
* **Avoid unhealthy fats.**Try not to eat any trans fats. These are man-made fats that are bad for you. Trans fats are often used in processed foods and store-bought baked goods. Read food labels carefully to check for trans fats. They will appear in the ingredient list as “hydrogenated vegetable oil” or “partially hydrogenated vegetable oil.”

Foods that are high in saturated fats (for example, red meat) can contribute to high cholesterol levels. Over time, [**high cholesterol**](https://familydoctor.org/condition/cholesterol/) can increase your risk for [**heart attack**](https://familydoctor.org/condition/heart-attack/) and stroke. When you do eat red meat, reduce your portion size. Choose poultry and fish more often.

You can also avoid unhealthy fats by using olive oil or canola oil when you are sautéing foods. Bake, broil, or roast your food instead of frying it.

* **Get your omega-3 fatty acids.**The most common source of omega-3 fatty acids is fatty fish (sardines, tuna, salmon, mackerel, and herring). Try to eat this type of fish once or twice a week.
* **Talk to your doctor about the risks and benefits of taking vitamins or supplements.**Your doctor might suggest a dietary supplement based on your overall health and the vitamins or minerals your diet lacks. If you are interested in taking another type of supplement, talk to your doctor about why you want to take it and what you hope it will do for you. He or she can help you figure out if a dietary supplement will interact with any medical conditions you have or any prescription or over-the-counter (OTC) medicine you are taking.

### **What else can I do to maintain my brain health?**

You can stay active physically, socially, and mentally. Physical activity helps prevent disease and maintain blood flow to the brain. If you don’t already exercise, try to work up to 30 minutes of moderate activity into your schedule 5 times a week. Moderate activities include anything that gets your heart rate up. Walking, hiking, bicycling, and swimming are all good options. Choose something you enjoy doing.

Any activity you do with other people helps to stimulate your brain. A social activity can be as simple as having lunch with a friend or walking around the block with a neighbor. Volunteer opportunities in your community or church are good ways to be social. Another option is finding a club or social group that focuses on a sport, hobby, or topic you enjoy.

To keep your brain cells strong and active, it’s important to stay mentally active. Challenge yourself to learn something new. Read to stay informed and for fun. Enroll in a class at a local community college or adult education center. Or, challenge yourself in a different way by playing games, completing puzzles, or trying memory exercises.

**Functional Medicine Coaching Academy**

<https://functionalmedicinecoaching.org/reversing-cognitive-decline/>

**Note: watch 3-minute video intro by Dr Bredesen.**

## ****Reversing Cognitive Decline (RCD) for Coaches****

The Functional Medicine Coaching Academy and Dr. Dale Bredesen have partnered to create Reversing Cognitive Decline for Coaches (RCD), the only course that trains Health Coaches and other healthcare professionals in the Bredesen Protocol for Reversing Cognitive Decline AND earns you NBHWC Continuing Education Credit.

**COURSE CONTENT:**

This ground-breaking course is designed by The Functional Medicine Coaching Academy and Dr. Dale Bredesen, an internationally-recognized expert in the mechanisms of neurodegenerative diseases. He developed the Bredesen Protocol to address the causes of cognitive decline in a comprehensive and personalized way that leads to improved outcomes. The Protocol is one of the few promising treatments currently available for cognitive impairment, and Dr. Bredesen believes health coaches are the key to helping cognitively-impaired clients and their caregivers create changes that stick.

In this course, you’ll build proficiency in a range of skills and topics crucial for working with cognitively-impaired populations, including but not limited to:

* Develop skills and knowledge to coach cognitive decline clients and their caregivers
* Learn how to best support practitioners who are using the Bredesen Protocol
* Effectively coach cognitively-impaired clients and caregivers in the Bredesen approach to cognitive decline
* Demonstrate understanding of ketosis, the recommended nutrition plan, and intermittent fasting
* Describe the importance of accurate assessment and differential diagnosis
* Explain how to use Brain HQ
* Choose appropriate coaching strategies to effectively partner with cognitively-impaired clients and caregivers to create goals for initiating and sustaining healthy lifestyle change
* Access to recordings of Live Town Hall Meetings where Dr. Bredesen answers questions from practitioners and patients.

**This self-paced online course is presented in 8 modules:**

* Introduction
* Overview of Cognitive Decline and Bredesen's Approach to Cognitive Decline
* Coaching Clients and Caregivers with in the Bredesen Protocol
* Nutrition Plan for the Bredesen Protocol
* Lifestyle Interventions
* Brain Training and Neuropsychological Assessment
* Coaching In Action
* Conclusion

**Why Take this Course?**

Cognitive decline, a lapse in cognitive functions like memory and reason associated with conditions like Alzheimer’s disease, dementia, and other types of mild cognitive impairment, is becoming a global epidemic. These conditions affect the sufferer’s ability to make good decisions, recall important information, access and create memories—in short, cognitive decline impacts every area of the patient’s life. Global incidence of these conditions is growing at an epidemic pace, yet few effective treatments exist and few healthcare professionals have sufficient training aimed specifically at reversing cognitive decline. Cognitively impaired patients and their caregivers need support NOW, and more and more of them are turning to health coaches looking for results.

In this first-of-its-kind course, health coaches and other healthcare professionals will:

* + Learn effective strategies and tools to help cognitively-impaired clients and their caregivers create goals for initiating and sustaining healthy lifestyle change
	+ Build the language and skills to confidently partner with doctors whose patients are undergoing the Bredesen Protocol
	+ Explore a new potential client base and coaching niche in one of the fastest-growing areas of healthcare
	+ Build a foundation in a critical skill set that, should you choose to pursue it, will set yourself apart and differentiate your practice
	+ Start to speak confidently on topics like ketosis, the recommended nutrition plan, intermittent fasting, Brain HQ, and the importance of accurate assessment and differential diagnosis
	+ Earn 10 NBHWC Continuing Education hours (if eligible)

Note: cost of course: USD649

**Getting Started:** Once you purchase Reversing Cognitive Decline for Coaches, you will receive an email invitation to the course from our educational platform, Canvas.  You will have access to the course material in Canvas for 6 months, and all materials are downloadable as well. Modules will be completed in sequential order, and students who complete the course will receive a certificate of completion.

Note: this website and articles reference Dr Bredesen and advocate many similar protocols.

SharpAgainNaturally

60-min video - <https://www.youtube.com/watch?v=t1XvjeoOK0c>

<https://sharpagain.org/a-new-approach/>

## A MULTI-CAUSAL, MULTI-THERAPEUTIC APPROACH

After learning about these causes of dementia, it would be another two years before we learned about the work of Dr. Dale Bredesen at UCLA and the Buck Institute for Research on Aging. During the past few years, Dr. Bredesen had been conducting a small study utilizing a similar multi-causal, multi-therapeutic approach. His groundbreaking results showed a 90% success rate (nine out of ten subjects) in restoring cognitive function, enabling those who had to quit or cut back on work to return to their jobs.

What Dr. Bredesen’s research made clear is that dementia has many causes and that treating all of them simultaneously often produces the best results. He uses a leaky roof with 36 holes as an analogy for AD: you can patch one hole or even several holes, but the roof will still leak until all the holes have been repaired.

Dr. Bredesen has continued to work with patients and published a follow-up paper ([Reversal of Cognitive Decline in Alzheimer’s Disease](https://sharpagain.org/wp-content/uploads/2018/03/Reversal-of-Cognitive-Decline-in-Alzheimers-Disease.pdf)) in 2016.  He now trains physicians around the world to treat memory loss using his protocol.

Another major study, the [Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGER)](https://sharpagain.org/wp-content/uploads/2018/03/Finnish-Geriatric-Intervention-Study-to-Prevent-Cognitive-Impairment-and-Disability-FINGER.pdf) from 2009-2011 showed that nutrition, exercise and mental stimulation alone had a positive and significant impact on cognitive function.

## TEN CAUSES OF DEMENTIA

Working with our [Medical and Dental Advisory Board](https://sharpagain.org/about/#advisoryboard), Sharp Again has identified 10 causes of dementia that can be evaluated and treated, potentially reversing memory loss and restoring full cognitive function:

1. [Nutritional imbalances and deficiencies](https://sharpagain.org/nutrition/)
2. [Toxins in food, water, air, work/home environments](https://sharpagain.org/environmental-toxins/)
3. [Effects of prescription medications](https://sharpagain.org/prescription-medications/)
4. [Mercury and other heavy metal toxicity](https://sharpagain.org/heavy-metal-toxicity/)
5. [Hormonal imbalances](https://sharpagain.org/hormonal-imbalances/) (T3 thyroid, HGH, estrogen, testosterone, and others)
6. [Inflammation](https://sharpagain.org/inflammation/) from low-level infections, including oral infections, mould, food sensitivities, URI’s and Lyme Disease
7. [Inadequate physical activity, mental stimulation, and social interaction](https://sharpagain.org/physical-mental-social-activity/)
8. [Prolonged stress](https://sharpagain.org/prolonged-stress/)
9. [Sleep and breathing problems](https://sharpagain.org/sleep-breathing-problems/)
10. [Physical and emotional trauma](https://sharpagain.org/trauma/)

### **Resources**

* [Alzheimer’s Disease Facts and Figures](https://www.alz.org/facts/)
* [National Institute on Aging](https://www.nia.nih.gov/)
* [Dr. Dale Bredesen, The Omega Institute](https://www.eomega.org/workshops/teachers/dale-e-bredesen)
* [Alzheimer’s Association](https://www.alz.org/)

**Healing Advocates** – You Can Heal Chronic Disease

<http://healingadvocates.org/>

<http://healingadvocates.org/alzheimers-disease/>