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The Strange Connection Between Vision Loss and Alzheimer's

Analysis by Dr. Joseph Mercola | January 15, 2024

STORY AT-A-GLANCE

- Loss of vision is associated with a higher risk for certain subtypes of Alzheimer's; having distance vision worse than 20/40 is associated with a nearly threefold higher risk of cognitive impairment
- Amyloid beta protein plays an important role in both vision loss and Alzheimer's, and studies suggest noninvasive ocular testing may help with early Alzheimer's diagnosis
- Microwaves emitted from wireless devices trigger a chemical cascade that significantly raises your risk for neurodegenerative diseases like Alzheimer's

Editor's Note: This article is a reprint. It was originally published September 4, 2017.

Alzheimer's disease, a severe form of dementia, affects an estimated 6.7 million Americans;[1] 300,000 to 360,000 of whom are under the age of 65.[2] In 2019 Alzheimer's disease killed 121,499 Americans,[3] making Alzheimer's the sixth leading killer in the U.S. for people age 65 and older.

Since there's no conventional cure, it's really important to take prevention seriously. There are also few if any successful medical treatments available once Alzheimer's sets in. For example, memantine (sold under the brand name Namenda) is approved for moderate to severe cases of Alzheimer's, but doctors also prescribe it off-label for mild cases. Unfortunately, the drug has been found to be practically useless for mild to moderate Alzheimer's.[4]

Other go-to drugs for the treatment of Alzheimer's include cholinesterase inhibitor drugs such as Aricept, Exelon and Reminyl. These too may do more harm than good as they provoke slower heart rates, significantly increasing your chances of getting a permanent pacemaker. They also raise your risk of hip fracture.

Surprising Link Between Vision Loss and Alzheimer's

Interestingly, research[5] shows loss of vision is associated with a higher risk for certain subtypes of Alzheimer's. As reported by Reuters:[6]

> *"Based on data from two large studies of older Americans … [T]he research team found that having distance vision worse than 20/40 and even the perception of having bothersome vision problems were associated with almost threefold higher odds of cognitive impairment.*
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> *Near-vision problems were less associated with higher odds of dementia or cognitive impairment … Regular vision screening of older Americans could help to catch people at greater risk of cognitive problems and dementia, the study team writes …"*

This isn't the first time this link has been made. Two papers[7,8] published in 2016 also concluded that ocular changes could be used as biomarkers for Alzheimer's, suggesting noninvasive retinal imaging and visual testing could help with earlier diagnosis. In the first of these papers, the authors present a hypothesis for the link between declining vision and dementia:

> *"Alzheimer's disease … has multiple cognitive subtypes. These are usually broken down into memory, language, executive, attention and visuospatial functioning. The variant of AD in which visual symptoms are prominent due to the localized pathology in the parieto-occipital region is often referred to as visual variant Alzheimer's disease …*
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> *The interconnection between eye and brain suggests that it is reasonable to look for ocular manifestations of neurodegenerative disease and regard the eye*

as an extension of the [central nervous system] CNS. In embryological development, the eyes and brain have a similar origin. The eyes are formed from the anterior neural tube, an area that later gives rise to the forebrain.

Ocular development occurs through specification of the eye field post-neural induction. This process involves specific transcription factors that are also conserved in brain development.

One such factor, a 'master regulator' gene of the development of the eye field, Pax6, plays an essential role in neural development. When expressed ectopically, Pax6 can induce ocular formation in other parts of the body, whereas its impairment or knockout disrupts neurogenesis in the cortex."

The Role of Amyloid Beta in Vision Loss and Dementia

Amyloid beta also plays an important role in both conditions. One of the hallmarks of Alzheimer's disease is the buildup of amyloid beta protein in the brain. The subsequent formation of brain plaque leads to progressive decline in cognitive and social functioning. Research has also linked amyloid beta deposition to neurodegeneration in the retina.[9]

For example, amyloid beta has been found in retinal drusen[10] (yellow-colored fatty protein deposits beneath the retina) and is a hallmark of age-related macular degeneration (AMD), the most common cause of blindness among the elderly.

As noted in a 2015 study:[11] "Multiple studies now link amyloid beta with key stages of AMD progression, which is both exciting and potentially insightful, as this identifies a well-established toxic agent that aggressively targets cells in degenerative brains."

Peripheral drusen has also been linked to a higher risk for Alzheimer's.[12] In short, researchers suggest that by analyzing the presence of amyloid in the eye, one may be able to predict amyloid buildup in the brain with a fair degree of accuracy. As noted in the second paper published last year:[13]

"Kerbage et al. suggested using a florescent amyloid binding ligand in order to maximize the chances of detecting Aβ [amyloid beta] in the lens.

By compounding the ligand substance (aftobetin hydrochloride) into a sterile ophthalmic ointment suitable for topical application; in combination with an in vivo pulsed laser fluorescent spectroscopy in a group of 20 AD [Alzheimer's disease] and 20 healthy controls the authors were able to detect supranuclear amyloid in the lens of most AD patients with a sensitivity of 85 percent and specificity of 95 percent …

The authors found a correlation between fluorescence uptake values in the lens with amyloid burden in the brain detected quantified using PET."

Zinc and Antioxidants Help Protect Against Vision Loss and Alzheimer's

Health conditions that have similar or identical causes can typically benefit from identical treatments. When it comes to vision loss and dementia, both are heavily influenced by nutrition. Zinc, in particular, appears to be a very important component. Previous research[14] suggests abnormal zinc mobilization in cells plays a role in the development of Alzheimer's. According to this study:

"Zinc, copper and iron have recently been reported to be concentrated to 0.5 to 1 mmol/L in amyloid plaque. In vitro, rapid Abeta [amyloid beta] aggregation is mediated by [zinc],[15] promoted by the alpha-helical structure of Abeta, and is reversible with chelation.

In addition, Abeta produces hydrogen peroxide in a [copper/iron]-dependent manner, and the hydrogen peroxide formation is quenched by [zinc]. Moreover, zinc preserves the nontoxic properties of Abeta.[16]

Although the zinc-binding proteins apolipoprotein E epsilon4 allele and alpha(2)-macroglobulin have been characterized as two genetic risk factors for

AD, zinc exposure as a risk factor for AD has not been rigorously studied.

Based on our findings, we envisage that zinc may serve twin roles by both initiating amyloid deposition and then being involved in mechanisms attempting to quench oxidative stress and neurotoxicity derived from the amyloid mass."

Studies have also shown that supplementation with antioxidants plus zinc and copper helps slow the progression of the wet version of AMD.[17] The study in question looked at two different antioxidant formulations. The first contained vitamins C and E, beta carotene. The second contained lutein and zeaxanthin in lieu of beta carotene. Both were effective against early-stage AMD.

Antioxidants have also been investigated for the prevention and/or treatment of Alzheimer's. One such study,[18] published earlier this year, found that cryptocapsin, cryptocapsin-5,6-epoxide (a carotenoid found in ripe red mamey[19]) and zeaxanthin effectively inhibited the aggregation of amyloid beta.

According to the authors, "Our studies provided evidence that cryptocapsin, cryptocapsin-5,6-epoxide and zeaxanthin have anti-amyloidogenic potential and could be used for prevention and treatment of AD."

The Role of Iron in Alzheimer's

Iron also plays a role in Alzheimer's, but in this case, excessive iron is the problem. As explained in an article published by the University of Melbourne,[20] "Iron has a special property that allows it to exchange electrons, which is crucial in allowing our bodies to generate energy from oxygen and fuels such as sugars. But it can also damage neurons in the same way that iron metal rusts in the presence of oxygen."

Using magnetic resonance imaging, the researchers measured iron levels in the brain, finding that people who had high levels of both iron and amyloid experienced a rapid decline in cognition over the course of six years.

Memory loss was particularly pronounced when the iron buildup was located in the hippocampus, which controls short-term memory. Similarly, when the iron was primarily located in the temporal and front lobes — associated with language processing — language skills rapidly declined.

Those with high levels of amyloid but low levels of iron, on the other hand, remained stable. Interestingly, the amount of iron found in the brain does not clearly correlate with the amount of iron a person ingests or the level found in blood. Hence, serum level of iron cannot be used as an indicator of Alzheimer's risk.

That said, I've often stressed that **high iron** is a significant risk factor for chronic disease, and recommend getting your iron tested annually to make sure it's not too high. People with hemochromatosis, a genetic disease that results in chronically elevated iron, also appear to be at increased risk for Alzheimer's, according to some research.[21] So, the link between iron and dementia is definitely worth considering.

Ideally, your serum ferritin should be somewhere between 20 and 80 nanograms per milliliter (ng/mL), certainly no higher than that. As a general rule, somewhere between 40 and 60 ng/mL is the sweet spot for adult men and non-menstruating women.

Here, antioxidants may again be helpful. For example, astaxanthin has been shown to upregulate heme oxygenase-1 expression (a protein produced in response to stress, including oxidative stress), thereby protecting against beta-amyloid-induced cytotoxicity and inhibiting amyloid beta-mediated production of harmful ROS.[22] Previous research has also shown astaxanthin helps improve cognitive function in seniors complaining of age-related forgetfulness.

Astaxanthin — A Potent Brain Food That May Protect Against Dementia

This natural pigment, a cousin of beta-carotene (albeit a far more powerful one) has also been found to reduce the accumulation of phospholipid hydroperoxidases, better known as PLOOH — compounds known to accumulate in the red blood cells of people who

suffer from dementia[23] — and some scientists believe astaxanthin could help prevent dementia, including Alzheimer's.

As a fat-soluble nutrient, astaxanthin readily crosses your blood-brain barrier. One study[24] found it may help prevent neurodegeneration associated with oxidative stress, as well as make a potent natural "brain food." According to yet another article in the Alternative Medicine Review:[25]

> *"Astaxanthin improved cognition in a small clinical trial and boosted proliferation and differentiation of cultured nerve stem cells ... Astaxanthin's clinical success extends beyond protection against oxidative stress and inflammation, to demonstrable promise for slowing age-related functional decline."*

The human diet does not contain very high amounts of astaxanthin, unless you eat loads of microalgae and sea creatures that consume the algae (such as salmon, shellfish, red trout and krill).

The typical dose of astaxanthin when taken in supplement form is 2 to 4 milligrams (mg), but emerging evidence suggests you may need a lot more, depending on your health status. Dr. Robert Corish, author of "A Guide to Men's Health: Easy Tips for a Long and Healthy Life," believes 12 mg may be an optimal dose for brain and heart health.

Mitochondrial Dysfunction Is at the Heart of Alzheimer's

I interviewed Dr. Dale Bredesen, director of neurodegenerative disease research at the UCLA School of Medicine, and author of "The End of Alzheimer's: The First Program to Prevent and Reverse Cognitive Decline."

Bredesen has identified more than four dozen variables that can have a significant influence on Alzheimer's, but at the heart of it all is mitochondrial dysfunction. This makes logical sense when you consider that your mitochondria are instrumental in producing the energy currency in your body, and without energy, nothing will work properly.

Your mitochondria are also where a majority of free radicals are generated, so when your lifestyle choices produce higher amounts of free radicals, dysfunctions in mitochondria are to be expected.

The accumulation of mutations in mitochondrial DNA are also a primary driver of age-related decline. Importantly, Bredesen's work sheds light on why amyloid is created in the first place. Amyloid production is actually a protective response to different types of insults, each of which is related to a specific subtype of Alzheimer's. As Bredesen explains:

> *"If you've got inflammation going on, you are making the amyloid because … it is a very effective endogenous antimicrobial. [I]n that case, it's not really a disease … [It's] a falling apart of the system. You're making amyloid because you're fighting microbes, because you're … inflamed, because you are decreased in your trophic support (insulin resistance, and so on) or because [you're toxic].*
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> *Guess what amyloid does beautifully? It binds toxins like metals, mercury and copper. It's very clear you're making [amyloid] to protect yourself. It's all well and good if you want to remove it, but make sure to remove the inducer of it before you remove it. Otherwise, you're putting yourself at risk."*

The program Bredesen developed is a comprehensive approach that addresses the many variables of Alzheimer's at their roots.

Alzheimer's Screening Tests

Bredesen also recommends a number of screening tests to help tailor a personalized treatment protocol. For example, if you have insulin resistance, you want to improve your insulin sensitivity. If you have inflammation, then you'll work on removing the source of the proinflammatory effect. If your iron is elevated, you'll want to donate blood to lower it, and so on.

Alzheimer's Screening Tests

Test	Recommended range
Ferritin	12 to 232 ng/mL
GGT	5 to 40
25-hydroxy vitamin D	60 to 80 ng/mL
High-sensitivity CRP	Less than 0.9 mg/L (the lower the better)
Fasting Insulin	Less than 4.5 mg/dL (the lower the better)
Omega-3 index and omega 6:3 ratio	Omega-3 index should be above 8% and your omega 6-to-3 ratio between 0.5 and 3.0

EMFs — A Wildly Underestimated Contributor to Alzheimer's

In 2016, Dr. Martin Pall published a review[26] in the Journal of Chemical Neuroanatomy showing how microwave radiation from cell phones, Wi-Fi routers and computers and tablets not in airplane mode is clearly associated with many neuropsychiatric disorders, including Alzheimer's. My interview with him was published yesterday.

What Pall discovered is that **microwaves emitted from devices** such as these increase intracellular calcium through voltage gated calcium channels (VGCCs), and the tissue with the highest density of VGCCs is your brain.

Once these VGCCs are stimulated they trigger the release of neurotransmitters, neuroendocrine hormones and highly damaging reactive oxygen species (ROS), significantly raising your risk for anxiety and depression and neurodegenerative diseases like Alzheimer's and brain cancer.

Based on this mechanism, it seems clear that chronic exposure to electromagnetic fields (EMFs) can play a significant role in dementia and that as a society, we need to take this very seriously. On a personal level, be sure to limit your exposure to wireless technology. Simple measures include turning your Wi-Fi off at night, not carrying your cellphone on your body and not keeping portable phones, cellphones and other electric devices in your bedroom.

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