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Wellness Institute

Alzheimer's Part 1

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Introduction

In 1901 Alois Alzheimer came into contact with Auguste Deter. She exhibited strange behavior and loss of memory. Alzheimer made arrangement for her care in exchange for her brain and records after her death She died April 8, 1906.

Using staining techniques Alzheimer was the first to identify the amyloid plaques and neurofibrillary tangles that are commonly used to identify the disease named after him.

Alzheimer's original paper on the disease noted an atherosclerotic appearance to the blood vessels in Auguste's brain. The concept of cardiogenic dementia appeared in the medical literature in the 1970's. This is the idea that dementia develops because damage to the arteries cuts off blood supply and nutritional support for brain cells resulting in thir death. There is little doubt today that blood vessel damage can be a major factor contributing to the death of brain cells.

In the 1990's genetic researchers discovered a gene variant called APOE4 or adapolipoprotein E4 which greatly enhances the chances of developing Alzheimer's. A single copy of the gene increases the risk of developing the disease three-fold. A double copy of the gene, one copy from each parent, increases the odds of developing Alzheimer's nine-fold. This genetic marker is considered the highest risk factor for developing the disease.

This could be very depressing, however, there is a "Nigerian paradox." Nigerians have one of the highest occurences of this nasty gene, yet one of the lowest incidences of Alzheimer's in the world. What this tells us is that environment trumps genetics when it comes to Alzheimer's.

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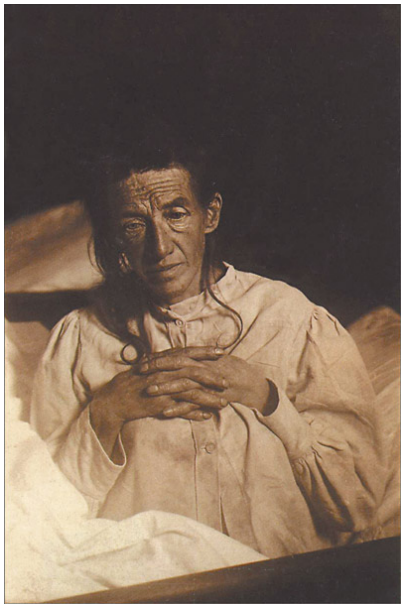
Auguste Deter, the first Alzheimer patient: https://commons.wikimedia.org/wiki/Alois_Alzheimer#/media/File:Auguste_D_aus_Marktbreit.jpg



The APOE4 Gene

One of the leading names in Alzheimer research is Dale Brednesen. His insight into Alzheimer's resulted from research on as substance called amyloid precursor protein (or APP). This protein is produced by brain cells. Enzymes can split the protein in two ways. One of these results in amyloid beta which is a major contributor to the development of Alzheimer's as it contributes to pruning back and killing brain cells.

But amyloid precursor protein can be split another way resulting in the formation of CTFa which protects brain cells and promotes synaptic growth.

With this molecule as a guideline, Brednesen identified 36 different factors which influence how APP is split. He then gathered these factors under several major headings or categories. He has concluded that Alzheimer's develops as a result of inflammation/infection, malnutrition, and toxic exposure. Cardiovascular disease and trauma can reinforce the damage done by these other factors.

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Inflammation/ Infection

Amyloid beta upregulates the inflammatory response and actually has antimicrobial properties. This protein

can preserve life when individuals live in unsantiary environments where they are exposed to bacteria, fungi, and viruses. Foods to which an individual is intolerant can also promote inflammation.

Even though in the long run, when excessive, amyloid beta can destroy brain cells, in the short run it can be protective. The APOE4 gene increases the tendency to produce an inflammatory response by encouraging the split of APP to produce amyloid beta. Thus it increases the chances of survival of the young in a dangerous microbial environment, at the cost of a shorter life. The problem with amyloid beta is that once it is produced it tends to encourage the production of more amyloid beta.

Measuring Inflammation

The best measurement for inflammation is high sensitivity C reactive protein or hs-CRP. An optimal measure for this marker is less than one.

Searching for the Cause

The first step in dealing with inflammation and infection is to try to avoid the cause. A number of infectious agents have been identified in those with Alzheimer's including herpes viruses, bacteria causing gum disease, lyme disease and associated microbes, and pathogens in the sinuses. Intolerances to gluten and dairy can also be triggers.

Infections can be dealt with by the use of antibiotics, antivirals, and antifungals. Unfortunately, these medications often have harmful side-effects.

Boosting Immune Function

Boosting immune function with nutrients is often very helpful in ridding the body of infectious organisms. Vitamin C is a potent antibacterial, antiviral, and antifungal. In one study, carotenoids were shown to enhance immune function, as measured by increase in the number of white blood cells, by 37% in 20 days. Garlic has also shown itself to be a very powerful antimicrobial agent.

Many studies have shown that polyphenols and flavonoids have antibacterial, antifungal, and antiviral activity. They have also been shown to reverse the accumulation of amyloid beta. Both green tea and pomegranate polyphenols are protective against amyloid beta.

Fats and Inflammation

One factor governing the tendency to become inflammed is the balance between omega-3 fats and omega-6 fats. Too many omega-6 fats predispose the immune system to the inflammatory response even when it is unnecessary.

One type of omega-6 fat is the cornerstone of the inflammatory response. That fat, arachidonic acid (or AA), triggers what is called the arachidonic acid cascade which results in inflammation and also increases the risk of clotting which can lead to stroke.

Unfortunately, arachidonic acid is particularly abundant in foods derived from animals like chickens and cattle that are fed corn and other grains.

Omega-3 fats are required for photosynthesis and are found in leafy greens and in the bodies of animals which have been eating green foods. The eight members of the omega-3 family, especially EPA, counteract the inflammatory effects of arachidonic acid.

Unfortunately, the modern diet can contain anywhere from ten to one hundred times more omega-6 than omega-3. This can be associated with a tendency to exist in a perpetual inflammatory state leading to the cumulative death of brain cells over a lifetime.

While inflammation is triggered by omega-6 fats it is resolved by omega-3 fats. Cessation of inflammation is accomplished by molecules called resolvins and protectins, made from the omega-3 fat DHA.

Lack of omega-3 fats can thus trigger a double whamy increasing the proclivity to become inflammed and, when lacking, leading to an inability to resolve inflammation.

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Malnutrition

Brednesen uses the Greek word "trophic" which means "feeding" to describe this component of Alzheimer's. The brain cells are among the most energy demanding cells in the human body. They require a *consistent* and *adequate* supply of good nutrition for a long and healthy life and brain cells are not replaced as easily when destroyed as many other cells in the human body. Certain nutrient deficiencies are both common and devastating in Alzheimer's.

BDNF

The health of neurons is supported by a substance called brain derived neurotrophic factor (or BDNF for short). This substance "feeds the nerves" and is powerfully stimulated by exercise. This explains why exercise is so beneficial for neurodegenerative conditions including Alzheimer's and Parkinson's disease.

Aerobic exercise improves blood flow in the brain and has been shown to actually reverse age-related shrinkage in the memory centers of the brain.

Panza and associates write, "Our findings suggest that exercise training may delay the decline in cognitive function that occurs in individuals who are at risk for or have AD (Alzheimer's disease), with aerobic exercise probably having the most favorable effect."

BDNF is also promoted by omega-3 fats and B complex vitamins, especially vitamin B3 or niacinamide. Omega-3 fats have been described as "fertilizer for brain cells."

It was once thought that brain cells were not produced later in life. It is now known that substances such as those mentioned above can trigger neurogenesis or the production of new brain cells. Neurogenesis can be blocked by inflammation and reactivated when the inflammation is blocked or resolved.

Homocysteine

One of the more common abnormalities associated with nutrient deficiency found in Alzheimer's is elevated homocysteine. This molecule is terribly destructive to brain cells. It is a byproduct of the metabolism of methionine, a protein found in large quantities in meat.

Methionine is not properly processed when deficiency of several B vitamins exists. Deficiencies of betaine, folate, vitamin B6, and B12 can all contribute to accumulations of homocysteine.

Deficiencies of these nutrients are common due to a number of reasons including the existence of nutritional antagonists to them and genetic defects that can alter the utilization of these vitamins.

A vegan diet can be effective against accumulation of homocysteine since methionine, its precursor, is most abundant in meats.

Vitamin D3

Another critical deficiency is that of vitamin D3 which plays critical roles in maintaining brain health. Vitamin D3 boosts immune competence by promoting the formation of cathelicidin, an antimicrobial peptide. Playing a role in regulating about 900 genes vitamin D3 also reduces inflammation, regulates blood sugar, and most importantly, plays a role in creating and maintainig brain synapses.

Hormones

Imbalances or deficiencies of a number of hormones including estrogen, progesterone, testosterone, thyroid and adrenal hormones can damage brain cells.

Research on the NeoLife tre-en-en concentrate long ago demonstrated that nutrition is necessary for proper glandular function and that nutrition of tissues is often mediated and regulated by the endocrine glands. These glands need not only vitamins and minerals to function properly, but also lipids and sterols.

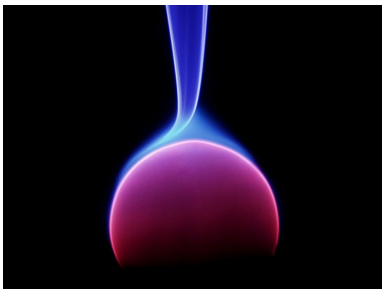
If medical support for endocrine glands is necessary, bioidentical hormones are generally preferable.

Minerals

Several minerals play a key role in brain health. Magnesium, selenium, and zinc are particularly important.

Magnesium is at the heart of energy production in the mitochondria. Failure of energy production in the these energy factories in brain cells is a hallmark of dementia.

Selenium is a critical component of glutathione, the body's most important antioxidant. Selenium is essential



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for coping with mercury toxicity as well as other toxins. Mercury is one of the most damaging molecules to brain cells.

Zinc deficiency and copper excess has been noted by some in Alzheimer's. The excess copper leaches into drinking water from copper tubing which carries the water into homes.

Copper and zinc are competitive nutrients, so high copper can result in low zinc. Zinc is a critical component of the detoxification machinery of the body including both glutathione and also metalothionein.

Zinc is also essential for the production of insulin, its storage, and its release. Low levels are also associated with auto-antibodies which can be a major source of inflammation.

Zinc deficiency is common. It is estimated that one quarter of the world's population is zinc deficient. Deficiency is particularly common among the elderly.

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SUMMARY

Inflammation and malnutrition of brain cells are two key problem areas that need to be addressed in the prevention and treatment of Alzheimers. Supplementation needs to address both nutrients which feed and build healthy neurons, but also the issue of inflammation and infectious agents must be addressed.

There are two other key areas which will be addressed in a later newsletter. Alzheimer's is impacted by glycotoxicity or poor insulin functioning and erratic blood sugars. Toxic exposures such as mercury and aluminum can also play a role. Failure to address all the issues associated with Alzheimer's can result in a poor treatment outcome.

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