Image Awareness Wellness Institute

How Can I Keep My Brain Cells?

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Introduction

Alois Alzheimer was a German psychiatrist and neuropathologist. In 1901 he encountered Auguste Deter, a 51 year old patient with strange behavioral symptoms including short-term memory loss. Alzheimer became obsessed with this patient and when he died in 1906 he had the brain brought to Munich where he was working in the lab of Emil Kraepelin. He identified amyoid plaques and neurofibrillary tangles. In a speech he delivered on November 3, 1906 he presented the first picture of presenile dementia discussing both pathology and clinical symptoms. Kraepelin called the syndrome Alzheimer's disease in a textbook and the name was adopted by the medical community.

There are two key markers which are characteristic of Alzheimer's and which help us understand the nature of the condition. The first is tau protein which has become attached to phosphorus. The second is beta-amyloid accumulation.

Tau Protein

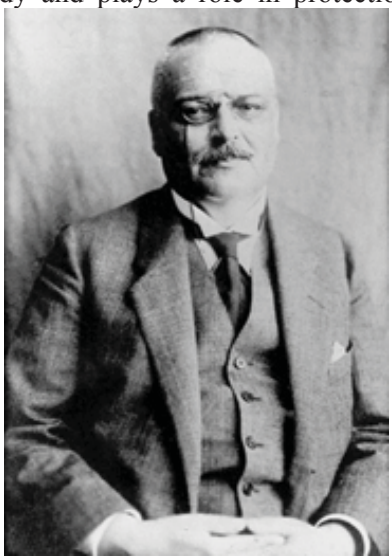
A network of microtubules is critical for the healthy functioning of nerve and brain cells. These tubes carry nutrients into the heart of the cell and waste products out. Tau protein fibrils or segments stabilize and support the microtubules. When tau protein combines with phosphorus it can no longer attach to the microtubules. The microtubules cease functioning resulting in damage to the cells.

When glucose utilization in the cell is compromised levels of an enzyme which decreases glucose metabolism become elevated. This enzyme, called glucose synthase kinase, is though to be most responsible for the attachment of phosphorus to tau protein blocking its normal function.

Failure of microtubule function causes degeneration of the synapses which connect one nerve to another. The practical result is loss of nerve function and potential cell death. Decline of brain function results.

Amyloid Beta (A β42)

Amyloid beta is the main component of deposits found in the brains of those with Alzheimer's disease. Amyloid beta is normally sythesized by the body and plays a role in protection against oxidative stress, regulation of cholesterol transport and anti-microbial activity.



The amyloid beta protein in the brains of those with Alzheimer's does not fold properly and becomes toxic to nerve cells. It is composed of 42 amino acids. Accumulations of this form of amyloid beta trigger oxidative damage, inflammation, toxic damage to nerve connections and tangles. Formation of new nerve connections is hindered and existing connections are damaged leading to decline in brain function.

Production of amyloid beta rises when an individual is awake and falls when sleeping. This is of interest because chronic sleep deprivation is associated with early onset Alzheimer's. Symptoms of Alzheimer's also tend to spike late in the day and may be associated with amyloid beta accumulation through the day.

Amyloid beta also increases the permeability of the blood-brain barrier. Weakening of the blood-brain barrier may compromise the health of brain cells by allowing exposure to harmful substances.

It is of interest that higher cholesterol levels strengthen the blood-brain barrier. Weight loss is a universal finding among those who suffer with Alzheimer's. Decreased cholesterol levels may play a role in damage to brain cells.

References:

https://en.wikipedia.org/wiki/Beta_amyloid

Lorin, Henry, Alzheimer's Solved (Condensed Edition), www.BookSurge.com, 2005, 51-86.

Cole, Greg M., and Frautschy, Sally A., Docosahexaenoic acid protects from amyloid and dendritic pathology in an Alzheimer's disease mouse model, *Nutrition and Health*, 2006; 18, 249-259.

Depiction of neuron mictotubules: https://upload.wikimedia.org/wikipedia/commons/5/51/TANGLES_HIGH.jpg

Alois Alzheimer: https://commons.wikimedia.org/wiki/File:Alzheimer.gif

B Complex

The B vitamins are essential for energy formation in the mitochondria, the energy factory of the cells. Health of the neurons is dependent upon consistent energy production. One of the most promising nutrients for preservation of brain health is vitamin B3.

Vitamin B3 offers special promise in diseases associated with degeneration of the nerves. Researcher Kim Green using a mouse model of Alzheimer's demonstrated that vitamin B3 could decrease this abnormal form of tau accumulation (the attachment to phosphate) and improve learning and memory. Mice treated with nicotinamide had complete recovery from cognitive deficits. The researchers wrote, "In summary, the results presented suggest that nicotinamide has potential as a novel, safe, and inexpensive AD (Alzheimer's Disease) therapy, either alone or in combination with Aβ-lowering therapies."

The researchers suggest that amyloid beta modifies tau protein which in turn mediates decline in brain function. The researchers believe that amyloid beta interferes with the normal degradation abnormal tau protein circumventing a mechanism by which it contributes to dementia.

Reference:

Green, Kim N., et al., Nicotinamide restores cognition in Alzheimer's disease transgenic mice via a mechanism involving sirtuin inhibition and selective reduction of Thr231-phosphotau, *The Journal of Neuroscience*, 5 November 2008, 28(45):11500-11510.

Pomegranate

Pomegranates contain very high levels of antioxidant polyphenolic substances compared to other fruits and vegetables. Two powerful anti-dementia agents, ellagic acid and punicalagin, were identified in pomegranates in one study.

Researchers found that mice genetically engineered to express a protein which leads to Alzheimer's accumulated only half the amyloid beta, a marker for Alzheimer's, when their diets were supplemented with pomegranate juice.

Brain function of the mice was tested by subjecting them to a water maze to find a submerged platform. Mice given the pomegranate juice navigated the maze 35% faster than mice given water and also swam a more direct path to the platform.

Polyphenols in general improve brain function of mice. Supplementation for a period of five months resulted in maximum benefit. Some forms of polyphenols, those with simpler structure, target the brain much better than others. These polyphenols would have an easier time bypassing the blood-brain barrier and preventing inflammation and free radical damage in the brain.

References:

Kwak, H.M., et al., beta-Secretase (BACE 1) inhibitors from pomegranate (Punica granatum) husk, *Arch Pharm Res*, Dec. 2005 28(12):1328-32.

Hartman RE, Shah A, Fagan AM, Schwetye KE, Parsadanian M, Schulman RN, Finn MB, Holtzman DM Pomegranate juice decreases amyloid load and improves behavior in a mouse model of Alzheimer's disease, *Neurobiol Dis*, 2006, 24:506-515.

Fraser, Jessica, October 4, 2006, Pomegranate juice shown to halt Alzheimer's disease progression, http://www.naturalnews.com/020631_pomegranate_juice.html

Wang, Jun, et al., Brain-targeted proanthocyanidin metabolites for Alzheimer's disease treatment, *J. Neurosci*., April 11, 2012; 32(15):5144-5150.

Ketones

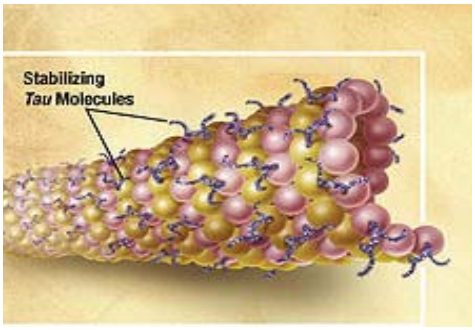
My master's thesis was on the topic of fasting. One of the things I learned in researching this subject was the fact that the body and brain feed on mostly carbohydrate reserves for the first few days of the fast.

By the fourth day of fasting the body switches to utilization of fat as an energy source. The body produces ketones, alternative fuel for brain and nerve cells during the fast. This switch to a fat metabolism suppresses appetite. This is why we read that Jesus did not become hungry until after forty days of fasting. (Matthew 4:2)

Fasting was shown to be a potential treatment for epilepsy in France in 1911. In 1921 Rollin Woodyatt identified three water-soluble compounds produced by the liver during fasting and low carbohydrate diets. These were b-hydroxybutyrate, acetoacetate and acetone. These are collectively known as ketones. In the same year Russel Wilder coined the term "ketogenic diet" and began to use the diet to treat epilepsy.

The ketogenic diet was widely and successfully used to treat many epileptics until the advent of anti-seizure medications in 1938. The diet lapsed into obscurity until resurrected by national media attention in October 1994 when NBC's "Dateline" television program covered the diet.

In the 1960's it was learned that a form of fat known as medium-chain triglycerides (MCTs) produce more ketones that normal dietary fats which are mostly long chains. MCTs are more easily absorbed and more rapidly transported to the liver than other fats. The ability of the brain to fuel its energy requirements with ketones is of great significance as will be discussed shortly.



REFERENCE:

http://www.news-medical.net/health/History-of-the-Ketogenic-Diet.aspx

Prelude: Type 3 Diabetes

The body uses glucose to produce energy. Glucose must be able to enter the cell for this to happen. A transport protein called GLUT (named for "glucose transporter") must carry the glucose into the cell. This is not possible until insulin opens a door that allows GLUT to carry the glucose into the cell. If insulin receptors are not functioning normally, cells can begin to starve. The consequence of total lack of insulin for normal cells is seen in Type 1 diabetes. In type 2 diabetes the insulin does not function properly.

In 2005 Suzanne M. de la Monte, M.D., and her associates discovered that insulin is made in the brain. Prior to this is was thought insulin had its only origin in the pancreas. The researchers found evidence that the brains of those with Alzheimer's disease show both deficiency of insulin and also insulin resistance or failure of insulin to function properly. Researchers coined the term "type 3 diabetes" for this condition. The researchers wrote, "We conclude that the term 'type 3' diabetes accurately reflects the fact that AD (Alzheimer's Disease) represents a form of diabetes that selectively involves the brain and has molecular and biochemical features that overlap with both type 1 diabetes mellitus and T2DM (type 2 diabetes mellitus)."

The brain is only 2 percent of our body weight, but utilizes approximately 20 percent of the calories we consume. The brain normally uses glucose for fuel. Failure of the brain in its ability to utilize its primary fuel has devastating consequences including the cell death and brain shrinkage characteristic of Alzheimer's.

Failure in glucose utilization can result from a nutrient deficient diet or exposure to environmental toxins. Because glucose is converted to energy in the mitochondria type 3 diabetes is a mitochondrial problem.

If a major component of Alzheimer's is failure of insulin production or failure of insulin to function normally in the brain, it only makes sense that provision of an alternative source of energy production would be of benefit to the individual suffering with the disease. Ketone or MCT supplementation offers promise for dementia and neurological conditions for this reason.

The potential value of ketones should not overshadow the fact that utilization of glucose can be improved by a number of means including consumption of complex carbohydrate rather than simple sugars and refined carbohydrates and supplementation with vitamins (especially the B complex) and minerals (especially magnesium and zinc).

REFERENCES:

Steen, Eric, de la Monte, Suzanne M., Impaired insulin and insulin-like growth factor expression and signaling mechanisms in Alzheimer's disease--is this type 3 diabetes? *Journal of Alzheimer's Disease*, 2005; 7:63-80.

de la Monter, Suzanne M., and Wands, Jack R., Alzheimer's disease is type 3 diabetes--Evidence reviewed, *J Diabetes Sci Technol*, November 2008; 2(6):1101-1113.

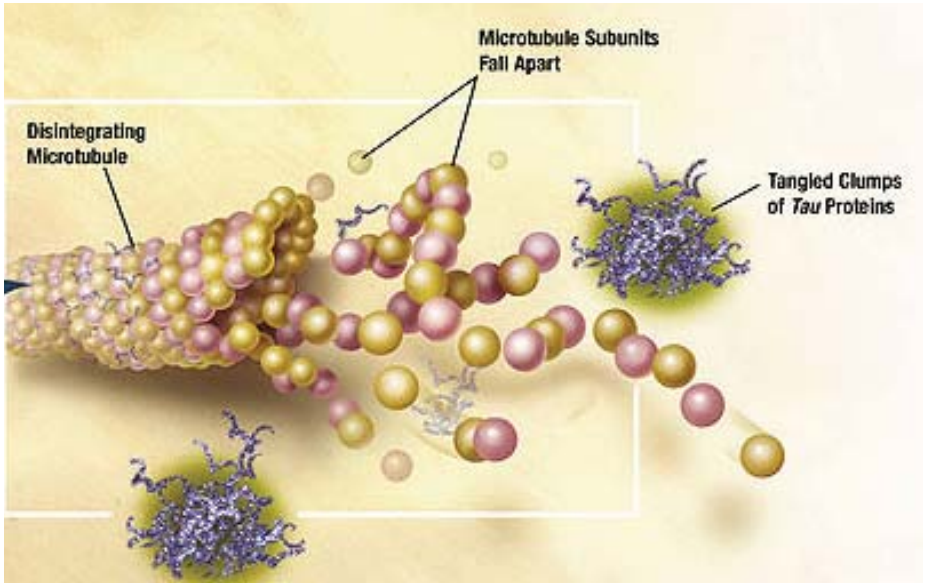
Alzheimer's Disease

Interest in ketones as a contributor to brain health has been reawakened recently by Mary Newport, M.D. Mary's husband, Steve, began to suffer with depression in the year 2000 and was placed on medication. (These medications are known to damage brain cells.)

In 2003 it became obvious Steve's problem was Alzheimer's disease. Despite medication his decline in mental functioning ability was rapid.

By 2008 the situation was desperate. Newport ran across a patent for an experimental drug called AC-1202 (now known as Axona). The drug proved to be a combination of medium chain triglycerides. Half of Alzheimer patients improved with a dose of about 20 grams or four teaspoons of MCT's in the clinical trials. Mary learned that coconut oil was a rich natural source of ketones and medium chain triglycerides.

On May 21, 2008 Mary began to give Steve coconut oil. The dose was two tablespoons. Mary noticed almost



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immediate improvements which improved gradually for an extended period of time. Testing also indicated Steve was thinking better within a matter of days.

MCT's can cause digestive upset and diarrhea. Mary learned that Steve could handle about four teaspoons of MCT oil and three teaspoons of coconut oil a day without problems. After a year she was able to double the levels of the MCT oil.

Newport found that MCT oil caused ketone levels to rise higher while coconut oil kept blood levels elevated for a longer period of time. Steve has continued to improve.

Newport then set about to make the medical community aware of the value of MCTs for neurological disorders. She was denied an exhibit at the International Conference on Alzheimer's Disease. She learned that Accera, the pharmaceutical company which developed Axona was permitted a booth. There was no mention that Axona contained MCT's.

Mary's story was eventually picked up by the popular media and the illustrations of Steve's improved ability to draw a clock received widespread attention both in print media and also on the internet.

REFERENCES:

Newport, Mary T., Alzheimer's Disease What If There Was a Cure? The Story of Ketones, Laguna Beach, CA: Basic Health Publications, 2011.

http://www.coconutketones.com/whatifcure.pdf

Summary

The brain produces insulin and uses glucose as its primary fuel source. Failure in glucose utilization, called by some Type 3 diabetes, can lead to damage and death of brain cells. Medium length fatty acid chains called ketones can serve as an alternative fuel for brain and nerve cells sustaining their health and energy production where glucose utilization fails.

Toxic accumulation of amyloid beta may be a key factor in the development of Alzheimer's disease. Animal studies have shown that simple polyphenols such as those found in pomegranate juice can reduce amyloid beta accumulation by 50% in mouse models of Alzheimer's.

The combination of tau protein with phosphorus is another key marker for Alzheimer's. Supplementation with vitamin B3 in the form of niacinamide reversed this process and restored brain function in mice.

These findings only scratch the surface of the important role nutrition can play in prevention of mental decline. Supplementation may also prevent the progression of dementia and may even reverse the condition to a greater or lesser extent.

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