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Alzheimer's Part 2

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What is Alzheimer's?

In 1992 Shahrooz Rabizadeh conducted a neurological experiment which changed the understanding of how nerve receptors function. A ligand is a small molecule which attaches to another molecule which is usually larger. Ligands in the brain attach to receptors and change the way cells function.

Prior to this experiment is was assumed that ligands could be harmful or beneficial, but that they could not be considered essential for health of cells. In this experiment Rabizadeh found that if a key ligand was missing brain cells would commit suicide. When the ligand was present it would completely block cell suicide.

Subsequent research found 21 receptors that functioned in this fashion. They were named "dependence receptors." When not bound to their partners, they induce the deterioration or death of the cell.

Imagine a lemming running toward a cliff which is certain death. When it gets bound by a lemming trap, it can not commit suicide. Or imagine a house rigged with explosives. They are configured to only go off when the key is removed from the front door. This is the nature of the dependence receptor.

Alzheimer's is characterized by the accumulation of amyloid beta which can be likened to a bandage. When this substance attaches to neurons it covers the dependence receptors and prevents the partner ligands from attaching to the cells. The cells begin to shrivel up and die leading to Alzheimer's or dementia.

The first letter in this series described how numerous factors impact a substance called amyloid precursor protein (or APP). These factors determine whether APP splits to form amyloid beta which blocks the dependence receptor from receiving its appropriate ligand or whether it forms CTFa which promotes the health and growth of neurons.

APP is a switch. It is switched on or off by trophic or anti-trophic factors. Trophic factors build up or nourish brain cells. They are the construction crew. Anti-trophic factors are the demolition crew. They can create an unfavorable environment initiating downsizing of the brain in unfavorable conditions.

We saw that malnutrition, inflammation, and infections can send signals to split APP so that amyloid beta is produced. In this newsletter we shall explore how toxicity can contribute to the development of Alzheimer's.

An eye test which can detect Alzheimer's before symptoms appear has recently been announced. This may provide a warnig to at risk individuals giving them time to address these issues.

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Alois Alzheimer: By uncredited - http://neurophilosophy.wordpress.com/2006/11/03/100-years-of-alzheimers-disease/, Public Domain, https://commons.wikimedia.org/w/index.php?curid=11653065

Glycotoxicity

Glycotoxicity or sugar toxicity can be a contributor to Alzheimer's. Dale Brednesen describes glycotoxicity as a combination of malnutrition and inflammation. This is true, but sugar in excess also has toxic properties which we shall discuss.

The malnutrition associated with Alzheimer's is not primarily starva-

tion in the sense of reduced calorie intake, but rather the unavailability of glucose for utilization by brain cells.

Type 3 Diabetes

Abnormalities of sugar metabolism are so common and so profound in Alzheimer's that Suzanne de la Monte and Jack Wands published an article in 2008 suggesting that Alzheimer's is Type 3 diabetes.

It is known that brain cells produce their own insulin. It is also known that in the Alzheimer's brain cells are often resistant to insulin signaling. This locks glucose out of the neurons depriving the brain cells of energy which can lead to cell death.

Even though the brain is a relatively small organ in the body it can use up to 20 percent of the body's energy production. Two-thirds of this energy is used to help nerve cells "fire" or send impulses. One third is used for cell health maintenance, housekeeping and defense. Failure of this housekeeping and defense function results in cell death.

Brain cells can be nourished and energized by either glucose or ketones. When physician Mary Newport's husband was diagnosed with Alzheiemer's, she reached the conclusion that perhaps supplementing with ketones would compensate for the failure of glucose utilization which is so common in Alzheimer's. When she augmented her husband's diet with ketones in the form of coconut oil and medium chain triglycerides, the improvement was remarkable. The result was her book on the use of ketones to augment the treatment of Alzheimer's entitled *What If There Was a Cure?*.

Cells downregulate response to insulin when there is an excessive supply. Thus a diet high in sugars may contribute to insulin resistance in the brain. It has also been suggested that excessive food intake or consumption of junk fats may contribute to insulin resistance in the brain.

AGE's

The acronym AGE's refers to advanced glycation endproducts. The abbreviation is appropriate because AGE's accelerate the aging process of both body and mind.

AGE's are also known as glycotoxins. These are combinations of sugars and proteins resulting from excess accumulation of sugars or from cooking foods with dry heat, high temperatures, or for prolonged periods of time. Dietary AGE formation can be increased anywhere from 10 to 100-fold above foods in the raw state by cooking in this way.

Cooking foods at a low temperature and in a moist environment, as in a crockpot, dramatically reduces the production of AGE's in foods.

Considerable quantities of dietary AGE's are produced when cereal grains and made into flakes or crackers. Meats produce tremendous quantities of AGE's when they are cooked with dry heat at high temperatures. Foods will often turn brown or black when these toxic compounds are produced.

In the study by Uribarri and associated bacon had far more AGE's than any other food tested coming in at 91,577 units of the measure they were using. This was 5-10 times most other meats and up to 100 times many vegetables. AGE's can be absorbed in the digestive process triggering inflammation and oxidative damage.

AGE's can also be produced spontaneously within the body when blood sugar levels are elevated. Diabetics manufacture a great many AGE's which are responsible for contributing to heart disease, cataracts, kidney problems and other health issues associated with diabetes.

Vitek and associates argued in 1994 that AGE's promote amyloid beta accumulation contributing the the development of Alzheimer's. The mechanism involved is the generation of free radicals.

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Bacon: By Renee Comet (Photographer) - This image was released by the National Cancer Institute, an agency part of the National Institutes of Health, with the ID 2686 (image) (next)., Public Domain, https://commons.wikimedia.org/w/index.php?curid=24036670

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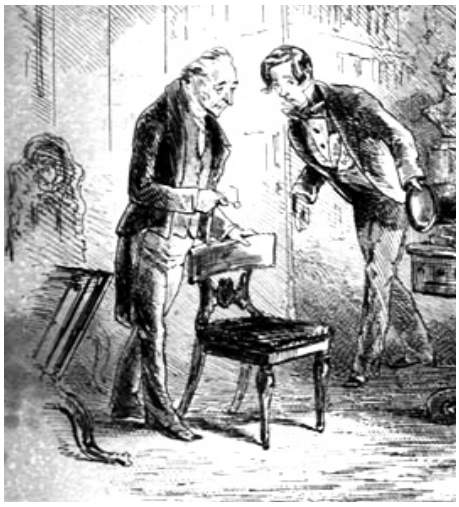
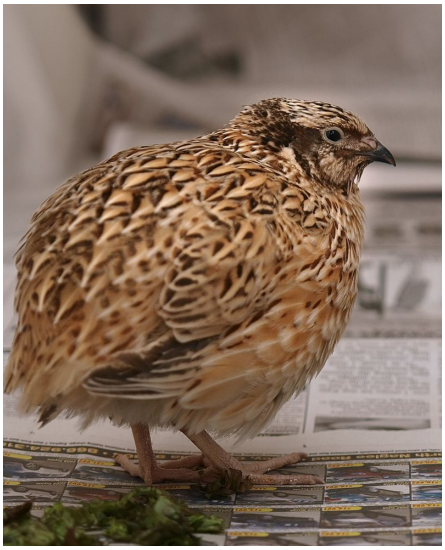
Toxin Exposure

Several toxic substances have been discussed as contributors to the Alzheimer epidemic. Bradnesen found





that many of these individuals did not have the typical APOE4 genetic marker associated with other types of Alzheimer's. These individuals tended to be younger and more difficult to treat. Exposure to mold toxins, mercury, and aluminum have been commonly discussed as toxic contributors to Alzheimer's.

Aluminum

Yumoto and associates identified the presence of aluminum in the senile plaques of patients with Alzheimer's in 2009. The aluminum was surrounded by amyloid beta. This suggests that perhaps the aluminum was being treated like a microbe.

Christopher Exley, a world leader in aluminum research, suggests that aluminum may potentiate oxidative damage in the brain by its interaction with iron.

In 2009 Walton and Wang found that aluminum upregulates amyloid precursor protein (APP) at levels achieved by chronic ingestion. They wrote, "This study shows *aluminum routinely derived from chronic oral ingestion, that gradually accumulates in brain regions important for memory-processing*, is sufficient to increase APP levels in neural cells of those regions. Aluminum may thus launch the cascade that results in the formation of amyloid plaques in human brain."

The use of aluminum as an adjuvant in vaccines has raised concerns on the part of many. Christopher Exley believes that the only acutely toxic form of aluminum is that which is injected into the bloodstream as an adjuvant in vaccines. This aluminum exists in the form of nanoparticles with different properties that the aluminum humans are typically exposed to. Other aluminum exposures lead to a gradual buildup of aluminum in the brain.

Christopher Shaw found that injected aluminum caused the degeneration and death of nerve tissue. He also found accumulation of "phosphorylated tau protein, a pathological hallmark of various neurological diseases, including Alzheimer's disease and frontotemporal dementia."

A wide variety of nutrients have been shown helpful in reducing aluminum toxicity. One paper identifies compounds which have been tested, "Specifically, supplementation with silica, taurine, vitamin E, quercetin, selenium, turmeric/curcumin and acetyl carnitine can provide protection." All of these nutrients and probably many others are most protective when used prior to exposure to aluminum.

Mercury

An excellent video entitled "How Mercury Causes Brain Neuron Degeneration" is available online from the University of Calgary. It clearly describes the extreme neurotoxicity of mercury. No other metal had the damaging effects upon neurons that mercury did in this study.

Mercury exposure results from the use of silver-mercury amalgams in dentistry and from eating large fish like tuna which tend to carry heavy loads of mercury.

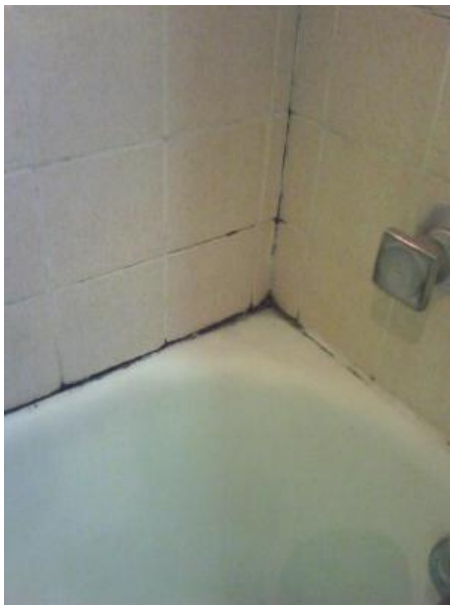
Selenium is a key nutrient for coping with mercury toxicity. In 1972 a study was conducted in which researchers fed Japanese quail mercury contaminated tuna which should have killed the birds. They were damaged by the same amount of mercury administered without the tuna. It turned out that the tuna was high in selenium which was protecting the birds. Selenium is a key protective nutrient against mercury. Selenium itself can be toxic in excess, however.

Mycotoxins

Brednesen classifies molds as dementogens. He estimates that they have damaged at least 500,000 people. The worst offenders are *Stachybotrys*, *Aspergillus*, *Penicillium*, and *Chaetomium*.

Dr. Ritchie Shoemaker has done more research on treating mold toxicity than most physicians. His research is summarized in his books including *Surviving Mold: Life in the Era of Dangerous Buildings*. Molds can grow anywhere that moisture accumulates. Shoemaker describes a syndrome called CIRS or chronic inflammatory response syndrome which can be triggered by mold toxins.

About one in four individuals in the United States has a genetic makeup which tends to cause immune activation when exposed to mold. The immune system in these individuals does not identify the problem, however. The immune system is chroni-



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cally activated making them sick, but antibodies are not formed to attack the toxins.

The prolonged immune activation in these individuals results in widespread fatigue and pain with numerous symptoms like asthma, nosebleeds, rashes, headaches, and cognitive decline.

Shoemaker found that those with the genetic markers HLA-DR and HLA-DQ were most susceptible to CIRS. Urine testing for mold toxins is available.

Bradnesen often finds infections in the sinuses or mouth which affect the brain. The infections in the sinuses are often mold related.

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CONCLUSION

Brednesen lists Alzheimer's as the third leading cause of death in the United States. He identifies 36 different factors which can contribute to Alzheimer's by increasing the production of amyloid beta. This set of papers is only a summary of his pioneering work with some of my own observations included.

Brednesen finds that Alzheimer's is associated with inflammation, blood sugar issues, malnutrition, toxic exposures, circulatory issues and trauma. If his concept is correct, and he is one of the few people who has had any success in helping people diagnosed with Alzheimer's, then any lifestyle changes and supplement programs that address the appropriate problems should be helpful in slowing the progress of the disease and even reversing it in the early stages.

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