

# An Experimental Design for Mitigating Alzheimer's Disease

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Alzheimer's is an irreversible, progressive brain disease that slowly destroys memory and thinking skills, leading to cognitive impairment that severely affects daily living. Alzheimer's is an extremely destructive disease that slowly erodes away life's essence at our very core. Symptoms usually develop slowly and get worse over time, becoming severe enough to interfere with daily tasks. The greatest known risk factor is increasing age, and the majority of people with Alzheimer's are 65 and older. Other age related disease exists including atherosclerosis and cardiovascular disease, cancer, arthritis, cataracts, osteoporosis, Type 2 diabetes, and hypertension. In my opinion, it is likely that these diseases share similar pathways and causes.

I am not a doctor and have no qualifications for medical research. But that does not prevent me from expressing layman opinions, formulating logical approaches and making analytical judgments. So I will begin this article by making a very important point ***Caveat Emptor*** [Caveat emptor is a Latin term that means "let the buyer beware." Similar to the phrase "sold as is," this term means that the buyer assumes the risk that a product may fail to meet expectations or have defects. In this case the product is the experimental approach.] This paper documents a layman's experimental approach that I would apply towards combating this disease. In solving any problem, especially very serious ones, I tend to take a multi-layered approach. Basically I throw everything at the problem including the kitchen sink. If I were to design an experiment for mitigating Alzheimer's disease, I would incorporate the following four elements: Chelation Therapy, Mitochondria Restoration, a Program of Exercise, and the Cessation of Destructive Habits.

The experiment should be analytical and use an administered cognitive screening test to assess changes in the degree of Mild Cognitive Impairment (MCI) and early dementia in order to evaluate its effectiveness.

## Chelation Therapy

Chelation therapy is a chemical process in which a synthetic amino acid solution-EDTA (ethylene diamine tetra-acetic acid)-is injected into the bloodstream to remove heavy metals and/or minerals from the body. Chelation means "to grab" or "to bind." When EDTA is injected into the veins, it "grabs" heavy metals and minerals such as lead, mercury, copper, iron, arsenic, aluminum, calcium and radioactive isotopes and removes them from the body. Except as a treatment for lead poisoning, chelation therapy is controversial and unproved.<sup>1</sup>

Some health professionals have also used chelation therapy to treat atherosclerosis and/or coronary artery disease, although there is not enough scientific evidence to prove that this treatment is effective. Some people believe that EDTA binds with calcium deposits (the part of plaque that obstructs the flow of blood to the heart) in the arteries, and then EDTA "cleans out" the calcium deposits from the arteries, reducing the risk of heart problems.<sup>1</sup>

Around 20 years ago, I experimented on myself using a form of chelation therapy. As I was beginning to get old (approaching my 50's and also becoming obese), my blood pressure was beginning to rise to unacceptable levels. The blockages that form clogged arteries are composed of calcium. I heard about the possibilities of using chelation therapy to treat this problem and decided to perform an experiment using this approach on myself. EDTA tablets are sold as supplements. I purchased a bottle of EDTA 625 milligram [0.625 gram] tablets. I took one tablet a day for approximately 3 or 4 weeks. Once per week I collected my urine in a quart size jar and put it on a shelf in the garage.



Figure 1 EDTA Tablets

*EDTA can cause abdominal cramps, nausea, vomiting, diarrhea, headache, low blood pressure, skin problems, and fever. It is UNSAFE to use more than 3 grams of EDTA per day, or to take it longer than 5 to 7 days. Too much can cause kidney damage, dangerously low calcium levels, and death.*

The urine in the jar from the first week was dark and cloudy and had a very odious smell. After it sat on the shelf for a couple days, white powder precipitated to the bottom of the jar to a depth of around 1 inch. I believed this white paste was calcium, which had been chelated from my arteries. The urine in the jar from the second week was lighter and after it sat for a couple days contained much less white paste. The urine in the jar from week three was a very light yellow, did not have an odious smell and very little white paste precipitated to the bottom of the jar.

After the experiment my blood pressure returned to normal readings for a period of about 2 years and then began to rise again to unacceptable levels.

During the experiment I noticed the chelation therapy began to affect my dreaming. I experienced dreams that were disjointed and illogical. This was very unusual for me. I also had a sense of impending doom.<sup>1</sup> As a result, I discontinued the experiment and never repeated it again.

Since I conducted this experiment almost 20 years ago, I have observed no negative effects from the experiment over this time period.

Recently, I have begun to question if the experiment might have also caused the flushing of plaque blockages in the brain, which may have accounted for the anomalous dreams, which I experienced at that time.

Alzheimer's disease leads to nerve cell death and tissue loss throughout the brain. Scientists observed the following three effects when analyzing brain tissue under a microscope of individuals that have passed away with Alzheimer's disease.

- \* Alzheimer's tissue has many fewer nerve cells and synapses than a healthy brain.
- \* Plaques, abnormal clusters of protein fragments, build up between nerve cells.
- \* Dead and dying nerve cells contain tangles, which are made up of twisted strands of another protein.

Plaques form when protein pieces called beta-amyloid clump together. Beta-amyloid comes from a larger protein found in the fatty membrane surrounding nerve cells. Beta-amyloid is chemically "sticky" and gradually builds up into plaques.

The chelation experiment, may have not only freed the calcium deposits in my arteries and flushed them from my system but also traveled through the fine blood vessels in my brain and freed similar plaque deposits in my brain. This may have produced the strange disjointed, illogical dreams that I experienced at the time.

About the timeframe of this experiment, I began to ask about early memories.

My earliest memory dates back to when I was 2 years, 4 months old. This memory was very vivid and real. My parents took my younger sister Kathy and I and dropped us off at the orphanage. There were many children and lots of toys. The floors were wooden and you entered the orphanage by walking up outside entrance stairs. The place was run by women in strange clothing [nuns]. It was a friendly place. I tried to tell my sister that we were abandoned. Somehow I knew or sensed what an orphanage was. But she was having so much fun with the toys and the other children, she just couldn't understand. [After all she was only one year old at the time.] I became very frustrated that I could not make my sister understand that our parents had abandoned us. So I did the only thing a two year old could do. I cried and cried. My parents came back and took my sister and I home. And I was glad.

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<sup>1</sup> I will suggest that one of the side effects of this treatment is to unlock a sense of overwhelming doom. One of the coping mechanisms to deal with traumatic events is memory blocking (situation-specific amnesia). This process may be chemically induced in the brain. At a very young age we encounter events that are highly emotional. These are experiences such as abandonment and death. This can instill a strong sense of doom. As a young child's brain ages and matures, these episodes of dread are blocked and buried deeply away. Therefore one trait that might be expected by this chelation therapy is to unleash these deep-seated memories and a sense of doom.

I discussed this event with my mother. At first she denied it. Maybe she was ashamed of it or maybe she just forgot. I told her that it was real and had happened. Several weeks later, she said she couldn't understand how I could have remembered it. She filled me in on the details. At the time, my mom was expecting. She was about to give birth to my younger brother David in January. We lived 30 miles outside Buffalo, New York in the deep countryside. The winters were brutal. She was afraid that she would give birth trapped at home in a major snowstorm. So as her due date approached, she decided to move back into the city and stay with her relatives. But her relatives would take her but not her children. So my parents placed my sister Kathy and I in a Catholic orphanage temporarily until after the birth. After a couple days, my parents were contacted by the nuns in the orphanage who said they could no longer keep us. That her son (me) had cried hysterically without stop day and night for two solid days and they were at their wits' end. So my parents came and retrieved us.

Childhood amnesia, also called infantile amnesia, is the inability of adults to retrieve episodic memories, which are memories of specific events before the age of 3 or 4 years. The use of EDTA may also explain how I remembered an event that occurred when I was very, very young (2 years, 4 months). It may have unlocked and restored one of my earliest memories. Although this is not totally outside the range of normal, it is a very early vivid memory. Our brain stores memories of traumatic events in a special place and this one survived. I also remember fragments of another event that occurred around the same age when I was kicked by a large bull, which sent me into a coma for several hours. It seems like my early years may have been best described as a hard-knock life.

I assert that chelation therapy using EDTA **may** dissolve plaque between the nerve cells within the brain and this **may** halt the progression of Alzheimer's disease. The purpose of this experiment is to test that assumption. So the first element of an experimental design for mitigating Alzheimer's disease is a 4-week flush using chelation therapy. This is a repeat of the experiment that I conducted on myself in which I consumed one 625 milligram EDTA tablet each day for 4 weeks. In order to minimize the possibility of producing dangerously low calcium levels, I would recommend that the individual also receive a calcium citrate supplement daily, offset by around 8 hours from the time they consume the EDTA tablet.

## Mitochondria Restoration

The greatest known risk factor for Alzheimer's disease is aging. In order to reverse the degradation caused by this disease, we need to dial back the aging process. The next two steps, mitochondria restoration and a program of exercise, are focused towards that goal.

If you were to read a textbook definition of the mitochondria, it might read something like this: *Mitochondria are sub-cellular structures, called organelles, which are found in almost all human cells. They are responsible for oxygen-dependent metabolism, which leads to energy production in the form of adenosine triphosphate (ATP). Additionally, they play a role in metabolic processes such as the oxidation of fatty acids; amino acid metabolism; apoptosis or cellular suicide; signal transduction; and autophagy (which generally occurs if there's been any damage or stress to the mitochondria).*

But Nick Lane in his book *Oxygen, The Molecule that made the World*, provides a very interesting perspective. Age-related diseases are degenerative conditions brought about by the combination of mitochondrial leakage, oxidative stress and chronic inflammation. Oxygen is a poison for many

components of our cells. The mitochondria are the transport agent of oxygen within the cell. As we age, the mitochondria begin to leak oxygen. The oxidative stress rises as our mitochondria leak free radicals into the cells. But unlike infections, the new threat caused by aging cannot be eliminated. There is no cure for broken mitochondria. Instead, the chronic inflammatory response is perpetuated indefinitely and contributes to our physical and mental decline. The process is similar to a continuous feedback loop. Mitochondria leakage causes inflammation that leads to the release of more oxygen, as the cells believe they are under attack, which then drives a chronic inflammation cycle.<sup>2</sup>

Alzheimer's disease develops from the formation of tangles and plaques in the brain that form primarily under conditions of oxidative stress. Thus as mitochondria age and become leakers, it leads to oxidative stress and the onset of this disease. So in order to halt age-related diseases, it is important to restore vigor to the mitochondria in order to minimize this leakage.<sup>2</sup>

In 1997, medical testing showed that I have severe asthma. The condition plagued me my entire life beginning at least from my early teens. The asthma dramatically impeded my ability to breath. I used a peak flow meter [a calibrated instrument used to measure lung capacity in monitoring breathing disorders] to measure my lung capacity [peak expiratory flow or PEF] periodically. On a good day my peak flow might reach around 390 liters per minute. But I had many bad days. The normal peak for a healthy individual with my age and height should be around 527. Starting in January 1997, I began to treat the condition using albuterol [a bronchodilator]. Using the albuterol inhaler improved my peak flow substantially. It moved my peak flow to an average of 576, approximately a 50% increase in lung functions.

In 2005, I heard about a dietary supplement, Juvenon, which was designed to restore the functioning of the mitochondria. This peaked my attention. Each tablet of Juvenon contained 1,000 mg of Acetyl L-Carnitine HCl, and 400 mg of Alpha Lipoic Acid. Since Juvenon was formulated to rejuvenate the mitochondria and since the mitochondria controls the movement of oxygen in the body, I thought it might help with my asthma. So I performed a simple experiment. On 22 August 2005, I began taking this supplement. My peak flow went up over 100 points, which I considered to be astonishing. After adding this supplement, my average was 691, an increase of an additional 20% in peak flow. [This corresponds to peak flows better than those achieved by a normal 20-year-old adult.<sup>3</sup>] Some peaks were as high as 830. Also this reaction occurred quickly within minutes after taking the tablet and the effect lasted for about 24 hours. As a result, I have been taking this supplement ever since.

So I considered this experiment to be rather successful. It not only improved my lung function, but also in my opinion accomplished this by rejuvenating my mitochondria, allowing improved transport of oxygen within my body.



Figure 2 Juvenon

In order to slow down and reverse the age degradation in the mitochondria, which leads to the leakage of oxygen and contributes to the progression of Alzheimer's disease, the experiment should incorporate the use of this supplement. So I would recommend the subject be given one tablet of Juvenon each day during the experiment.

## Exercise

Another method of dialing back the aging process is to perform daily exercise.

I recently came across the following article and found it to be of interest:

*If feeling older than you look appeals to you, take a seat while you read this: A recent study found that women who sit longer than 10 hours a day, combined with low physical activity, have cells that are biologically older — eight years older to be exact — than their actual age.*

*The study looked at the lifestyles of 1,500 women, between the ages of 64 and 90, who are part of a Women's Health Initiative (WHI) — a national study on chronic disease and postmenopausal women. Researchers found that women who sat for more than ten hours per day, and exercised less than 40 minutes daily, had shorter telomeres — the caps at the ends of DNA strands which protect chromosomes. Shorter telomeres have been associated with increased risk for disease and poor survival, which is why low activity combined with prolonged sitting can contribute to cell aging. The picture was not as bad for women who incorporated at least 30 minutes of daily moderate to vigorous physical activity, even if they spent most of their day sitting at a desk.<sup>4</sup>*

Well let me inject a few words about this article. DNA (Deoxyribonucleic acid) is a molecule that carries the genetic instructions used in the growth, development, functioning and reproduction of all known living organisms. Essentially they are the blueprints of life. Within the DNA are nucleotide composed of one of four nitrogen-containing nucleobases—either cytosine (C), guanine (G), adenine (A), or thymine (T)—and a sugar called deoxyribose and a phosphate group. The order of building blocks in a strand of DNA makes up a "sequence." We can read a DNA sequence like letters in a book. In fact, we know the sequence of the entire human genome—all 3 billion letters. This DNA structure defines each individual, the color of our hair and eyes and skin etc. Most of the DNA is core and very important. But at the end of the DNA strand is DNA that seems to provide no purpose, it is called non-coding DNA or Junk DNA. But in reality, Junk DNA (telomeres) do serve an important function. The reason is that when the DNA replicates, it loses a little of the strand of junk DNA at its ends. Eventually as we age, this junk DNA gets shorter and shorter and eventually the destruction eats into the core DNA. When it does, it introduces anomalies, age related diseases. So this article is saying that exercise can reduce the loss of the junk DNA and we can live longer healthier lives as a result.

So I consider low impact exercise, 30 minutes of walking each and every day to be an integral part of this experiment. Even if the individual has to use a walker [a two or four-footed frame that elderly or disabled people use as a walking aid] to get about, this element is needed to inhibit and reduce the age related component of cellular degeneration. In my opinion, cellular degeneration is one of the primary causes of Alzheimer's disease.

## Cessation of Destructive Habits

Cease destructive habits that produce extreme oxidative stress to the cells. Primary on this list is smoking. This includes cigarettes, cigars, pipe tobacco, marijuana and other recreational drugs.

*Cigarette smoke is dangerous because it is the most dastardly free-radical generator known. Many chemicals in cigarette smoke, including semiquinones, polyphenols and carbonyl sulphide, react with oxygen to form superoxidate radicals, hydroxyl radicals and hydrogen peroxide, as well as nitric oxide and peroxyxynitrite. A single puff of cigarette smoke is said to contain 1,000,000,000,000,000 free radicals. <sup>2</sup>*

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