ALZHEIMER’S DISEASE

A simple man’s treatment alternative

Laure-édith Zoko
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Professor Danil Hammoudi
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Alzheimer’s disease is the term that is used to describe a disorder by a certain brain changes. This disorder is a brain disease that leads to the lost of mental and physical functions. With this disease, the patient ordinarily becomes unable to care for his or herself and depends on other to manage and supervise the simplest of mental and physical activities. This disorder is unknown it affect a small but significant percentage of older Americans. A very small minority of Alzheimer’s patients is under 50 years of age. But most of the people mostly get Alzheimer’s disease are at the age of 60. Alzheimer’s disease does not affect the lifespan of the patient. The gender, ethnicity, and genetics are very confusing to researcher. A researcher feels that anyone could get Alzheimer’s disease. Many researchers found that in some patient, they don’t have any family history or genetics or ethnicity in order to get Alzheimer’s disease. But researcher also came up with many drugs to treat Alzheimer’s disease, such as Hydergine, horticulture. The diagnostics of Alzheimer’s disease is still under improvement. This experiment will test Hydergine, horticulture and other sedative drugs in a double blinded theoretical trial.

RISK FACTORS & CAUSES

There are four major risk factors in theoretically three causes of Alzheimer’s disease.

- Possible autosomal transmission; though findings so far lean more toward sporadic gene mutation. (Wikipedia). It has been reported a stable annual incidence of 1.5% in the early 90s, which reason might be the operation of a dominant gene with population allele frequency of 0.13. It is possible that one third of the theoretical predisposition to Alzheimer becomes manifest during the life time of the patient relatives that are at risk.

- Head trauma due to injuries (ie. repeated blows to the head). (Center for Neuro Skills)
  Neuron loss increases with age and is possibly accelerated by head injuries. A person who has for a long received very hard blows to the head or has suffer at least once of a head injury may suffer from a formation of neurofibrillary tangles in the absence of neurotic plaques. This condition is classified amongst the characteristics of Alzheimer’s.

- Some complications from diabetes like stroke, hypertension are possible risk factors for Alzheimer’s. (Luchsinger et al.). Study proves that people with diabetes score lower on some cognitive functions than people without diabetes and that Alzheimer patient exhibit impaired glucose regulation compared with normal subjects.

Tests showed that, early onsets form a certain distribution that is different from that of later onsets. So the causes are maybe different. The known causes for Alzheimer are mostly genetics. The non-genetic cause is aging. Research shows that the brain’s ability for regrowth and reorganization may diminish with age. If head injuries occurs with old age then the consequences becomes more devastating. Other causes of Alzheimer’s disease are the mutation in the Presenilin gene. Presenilin mutations are believed
to cause early onset Alzheimer by triggering increased production of a-beta protein also known as amyloid beta protein. The senile plaques that form in Alzheimer patient brain are deadly and mainly made up of a-beta protein. The fact that the fibroblast cells taken from precenilin mutation produce increase level of a-beta proves the previous statement. Only precenilin gene cause excessive a-beta production. Apolipoprotein E-epsilon better known as ApoE ε-4 is considered a “risk factor gene” for the disease. (National Institutes for Aging). The association between Alzheimer's and head injuries varies with the presence of the ε-4 allele. Head injury with lost consciousness was associated with increased risk of Alzheimer's. There is a little change in the interaction between head injury and ApoE ε-4 when family history is included during a study 1987 to 1995 involving 349, elevated risk was observed among men but not women, and no significant variation in the head injury Alzheimer risk relation by ApoE ε-4 genotype was found.

**DIAGNOSIS**

To diagnose Alzheimer's many methods had been tried. The ones to be studied are:

1. **Start Wells Model**
   With start wells model of distribution of incubation periods for infectious disease, a pattern of Alzheimer's dementia occurrences has identified. Using the patient's age as the incubation period for Alzheimer's, it has been found in 1987 that the distribution case to date fits the start wells logarithmic normal curve. As a result it was said that the estimated incubation for the disease was from 44 to 74 years of age (after birth). So we can say that in general, heredity plays a stronger role in Alzheimer's transmission. For that study, the researchers focus on prenatal experience.

2. **Alz 50 Staining**
   In 1988 researcher reported new data showing that protein A68, a protein which was thought to be present only in the brain of Alzheimer's patient, is also present in the brains of fetuses at 34 weeks of gestation or in infants until 2 years of age. Alz50 Antibody used to detect the presence of A68 in the body of the patient. It also helps detect some amyloid plaques and neurofibrillary tangles in brain tissue from Alzheimer patient by staining those plaques and tangles. It detects about 85.7% of Alzheimer's cases.

3. **A-beta Protein Detector**
   Around the year 1991, researchers found two antibodies that could be used to detect a-beta protein. Those antibodies were Thioflavin and Congo Red. They could detect the disease with about 79% acuity. The a-beta protein has probably a vascular or circulatory origin. Like Alz50, Congo Red and Thioflavin detect by staining.

**SYMPTOMS**

Alzheimer's patients have a healthy appearance for the most part of the illness. Before physical death, they undergo a "psychological death" meaning that they lose all touch with their own personality. Communication is interrupted and the ability to act, think, or remember is reduced. The disease attacks
the nervous system in particular and cholinergic neurons are selectively destroyed. The reason for this loss of cholinergic neurons is not yet clear. Research showed that neurons affected by Alzheimer’s incur a loss of acetylcholine. In 1989, 2.5 millions Americans suffered form Alzheimer’s. Among the people 64 years and older, 7% suffered from the disease. Many of them had visual problems such as glare, clumsiness, and misjudgment of distances, but when tested, had a normal visual activity. Tests by researchers found that M-cells, which mediate low spatial contrast sensitivity, depth perception, motion, and orientation, degenerated in 80% of the clinically diagnosed patients suffering from Alzheimer’s disease.

EXPERIMENT FOR CURE
This experiment is a double blinded placebo on 100 people over a period of 12 months. It is conducted in order to find a possible cure for Alzheimer’s disease. To detect this disease, the Alz50 staining process has been use. The data has been recorded daily. After the application of Alz50 staining process, 86 people were left. 14 people were added in order to be used as constants. These people were healthy and not at risk individuals that would be reference points for the measure of cognitive improvement. 43 of the 86 patients suffered from early onset Alzheimer’s. 43 suffered from later onset Alzheimer’s.

1. **subdivision**
The constants, the early and late onset patients lived separately. The early onset Alzheimer’s patient were divided into two groups. The first group on EDA’s is given a preparation that we call HA (Hydergine and chemically broken down acetylcholine) and are initiated to horticulture. HA is preparation containing 0.35mg of each dihydrogergocornine mesylate, dihydrogergocristine mesylate, dihydrogergocryptine meyslate and 0.15mg of chemically broken down acetylcholine. The preparation is administered intravenously twice daily. The patients in the group EOA (Early onsets A) are during the same time initiated to a daily practice of horticulture.
The second group EOB (early onset B) who are administered the same dosage of HA and instead of horticulture, are given Valerian (a natural sedative) on a dosage of one 600mg capsule one hour before bedtime.
The later onset patients are also divided into two groups. The first group is called the LOA (later onset A). That group is given the same preparation. They too are initiated to horticulture. We will administer HA twice a day. The second group, LOB, is given HA and Valerian on a dosage of one 600mg capsule one hour before bedtime HA weights 1.2mg.
There is a possible risk of chemical interaction causing side effect like migraines, gastrointestinal discomfort, dizziness, and drowsiness, among the group of people using both HA and Valerian.

1. **Hypothesis**
The fact that Alzheimer’s patients are lacking acetylcholine and the two enzymes that break it down plays an important role in making the cure. It is expected that there will be greater recovery in the early onset groups than the late onset groups; the early onset groups being younger than the late onset group. Both horticulture and Valerian are used to calm down the agitated and violent.
2. **Discussion**

96.5% of the non-constant participants regained the will to live longer by the end of the 12-month trial. Cognitive improvement was observed in all the groups. The combination horticulture-HA produced far better results in the LOA than in the EOA group. 51.2% of the LOA group compared 34.9% of the LOB group reported reduced anxiety levels and showed improvement in daily cognitive functioning. 74.4% of the EOB group compared to 39.5% of the EOA group reported reduced anxiety level. Daily cognitive improvements were observed in 41.9% the EOA group 4.5 months after starting the treatment. Daily cognitive improvements were observed in 53.5% of the EOB group 3.8 months after starting the treatment. There was a gradual decrease in violent outbursts and/or otherwise antisocial behaviors. During the horticulture sessions, it was more likely to see the formation of sub-communities in the LOA group than in the EOA. The EOA group members were more likely to exhibit individualism. The latter experienced overall a less significant decrease in antisocial behaviors. The groups on Valerian exhibited a decrease in sporadic sleep patterns. The EOB group reported few to no instances of drowsiness and sleepiness in the morning. 25.6% of the LOB group reported experiencing post-awakening sleepiness during the first quarter of treatment. Functional MRIs of the brain showed an increase in activity in the region encompassing the right and left intraparietal sulci and in the hippocampal region during memory testing 6 months after starting the treatment in the EOB and LOA groups when compared to the observations prior to treatment. No change was observed with the constants. No significant change was observed with the EOA and LOB groups. The functional MRI results at the end of the trial showed an increased in activity in the region encompassing the right and left intraparietal sulci and in the hippocampal region during memory testing.

"Beep... beep... beep... beeeeeeep!" “What’s that noise? Where... But... Man! What a dream! Could it be this easy to help people with Alzheimer’s?”

Alzheimer’s disease is a very complicated pathology. Being linked to both psychology and genetics, it is harder to treat with 100% acuity. Many attempts have been made during the last two decades or so to explain and overcome the disease but none of them are absolute. For those directly or indirectly facing it today, it is important to have a clear and true discussion with the treating physician in order to be exposed to all available treatment alternatives. It is a big hurdle but not an insurmountable hill.
REFERENCES


7. Cover page image:
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