The Deadly Bloom

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Abstract

Alzheimer’s patients are suspected to have an increase level of Beta-methyl-amino-L-alanine (BMAA). BMAA is an environmental toxin produced by cyanobacteria and is linked to algae blooms. Found in sea creatures such as sharks, fish and shellfish that ingest algae, the neurotoxin is believed to trigger the neurodegenerative disease Alzheimer’s. The examiners performed an autopsy and removed the entire bran from forty deceased bodies. The deceased were separated into a control and experimental group. The experimental group consisted of twenty of the deceased who had diagnoses of Alzheimer’s while living and the remaining twenty had not victims of Alzheimer’s disease. The brains were then placed in a cylinder filled with fluid to distinguish the average volume between the control and experimental group. The results came back to the experimental group suffering from brain atrophy and the control group having no brain atrophy. Medical examiners also used an enzyme-linked immonosorbent assay (ELISA) to disclose the presence of the toxin in the brain tissue of Alzheimer’s patients. The ELISA test proved that BMAA was present in eighteen of the brains of the deceased diagnosed with Alzheimer’s while living. The findings also showed that BMAA was present in three of the healthy brains. To further understand the relation between Alzheimer’s and BMAA scientist should further investigate BMAA. More funding will go towards investigation. The safety information for seafood will contain both a safety summary and a complete toxicological profile. Exposure to BMAA should also decrease to reduce the cost of health care cost to Alzheimer’s.

Literature Review

Alzheimer’s disease is an illness that affects the brain and includes memory loss causing individuals to forget about loved ones and interfering with social skills. The disease attacks nerve cells of the brain, which then weakens a person’s ability to recall certain aspects of lives (Alzheimer’s Association, n.d). Having this complex illness interferes with daily routines, eating and taking medications have to be reminded by an aid or a family member. Impaired judgments, personality changes, annoyance are some of the symptoms (Web Md, n.d). Pictures, sticky notes are placed on items for remembrance.

Ranked number sixth leading cause of death in the United States, Alzheimer’s is irreversible (National Institute on Aging, 2016). Early onset Alzheimer’s occurs in people as young as thirty years old. Late onset Alzheimer’s occurs after sixty years old (NIH Senior Heath, 2015). The disease progresses slowly for some and faster for others. Mental abilities become compromised. Conversations are challenging as a result of compromised mental abilities (Alzheimer’s Association, n.d). It is categorized into three stages, mild, moderate and severe Alzheimer’s.

Individuals are diagnosed with the illness during mild Alzheimer’s. Driving and working are capable, however, during this stage a person is experiencing memory lapses. Family members start to take notice of struggles (Alzheimer’s Association, n.d). Close relatives notice difficulties with paying bills handling money and personality changes (National Institute on Aging, 2016). Doctors also detect problems in concentration and memory in this stage (Alzheimer’s Association, n.d).

The second stage is moderate Alzheimer’s. “Damage occurs in areas of the brain that control language, reasoning, conscious and sensory processing” (National Institute on Aging, 2016). Individuals require more help than the previous stage. There is an increase in memory loss, an urgency and uncontrollable time use the bathroom. Remembering names, faces and multitasking worsens. “At this stage people have hallucinations, delusions and paranoia” (National Institute on Aging, 2016). More time is spent with family members and not alone.

The final stage called severe Alzheimer’s is where assistance is needed at all times. Memory is compromised; people do not remember where they live or family member names, most times are spent in bed “Plaques and tangles spread throughout the brain and brain tissue shrinks” (National Institute on Aging). Joining in conversations does not happen and personalities change dramatically (Alzheimer’s Association, n.d).

The ultimate way to diagnose Alzheimer’s is to perform an autopsy. Doctors look at the brain tissues of the deceases to out find whether plaques and tangles exist (NIH Senior Health, 2015). Neurologists can make a diagnosis by asking patients and family members a series of questions about past health; conduct a test to measure memory and a brain scan to look for any abnormalities in the brain (NIH Senior Health, 2015).

A person diagnosed with Alzheimer’s can live between ten or twenty years. Depending on the severity some can remain alive for less than three years (Holland, 2016). The cause of Alzheimer’s is genetic for most and idiopathic for others. . “Late-onset Alzheimer’s arises from a complex series of brain changes that occur over decades” (National Institute on Aging, 2016). Alzheimer’s can be inherited from a parent. A child with one parent carrying the gene has a 50/50 chance of inheriting the mutation and develop early onset Alzheimer’s (National Institute of Aging, n.d).

A major determinant for late onset Alzheimer’s is the apoliprotein E gene (APOE). “APOE is a cholesterol carrier that supports lipid transport and injury repair in the brain” (Nature Reviews Neurology, 2013). Patients who carry copies of APOE e4 have a higher risk of developing Alzheimer’s disease than patients who are carry APOE 3 (UC Irvine Institute n.d). “APOE lipoproteins bind to several cell-surface receptors to deliver lipids and also to hydrophobic amyloid-B peptide which is thought to initiate toxic events that lead to synaptic dysfunction and neurodegeneration in Alzheimer’s disease” (Nature Reviews Neurology, 2013).

Health and lifestyle factors induce the risk of Alzheimer’s. “Researcher suggests that a host of factors beyond genetics may play a role in the development of Alzheimer’s disease” (National Institute on Aging, 2016). Researchers mention that heart disease, stroke, high blood pressure and diabetescould play a role in increasing the risk of developing Alzheimer’s disease (National Institute on Aging, 2016).

Beta-methyl-amino-L-alanine (BMAA) can also increase the risk of Alzheimer’s disease. BMAA is an amino acid produced by cyanobacteria (Ethno Medicine, n.d). The toxin is found in sea creatures such as sharks, fish and shellfish that ingest algae. “BMAA is inserted into human proteins, causing them to misfold replaces amino acid serine in the protein sequence” (Ethno Medicine, n.d). Beta-methyl-amino-L-alanine replaces the amino acid serine. Serine is an amino acid that is important in the functioning of the brain and central nervous system (Vitamins stuff, n.d). Replacing serine causes protein aggregation and apoptosis (Ethno Medicine, n.d). BMAA is a trigger for Alzheimer’s in some people.

An estimated 5.5 million Americans are diagnosed with Alzheimer’s in 2017 (Alzheimer’s Association, n.d). 200,000 are estimated to be under the age of 65 and 5.3 million are 65 and over (Alzheimer’s Association, n.d). Two thirds of Alzheimer’s patients are women (Alzheimer’s Association, n.d). African Americans are twice as likely to have Alzheimer’s as Caucasians (Alzheimer’s Association, n.d). Hispanics are one and one-half as likely to have Alzheimer’s as older Caucasians (Alzheimer’s Association, n.d). “Today, someone in the United States develops Alzheimer’s every 66 seconds” (Alzheimer’s Association, n.d).

Alzheimer’s disease does not have a cure, however, individuals can take medications and participate in non-drug therapy (Alzheimer’s Association, n.d). Cholinesterase and memantine are used to stabilize the symptoms of Alzheimer’s temporarily (Alzheimer’s Association, n.d).

Cholinesterase inhibitors are used to help delay mild to moderate Alzheimer’s (National Institute on Aging, 2016). These medications include donespezil and galantamine. These medicines help reduce the symptoms and prevent them from getting worst for an amount of time (National Institute on Aging, 2016). Although taking inhibitors is helpful, cholinesterase may lose effects as a result of the disease progressing (National Institute on Aging, 2016).

To help moderate to severe Alzheimer’s doctors prescribe Namenda (memantine) (National Institute on Aging, 2016). These medications help maintain functions longer. While this can be helpful for most, medications and non-drug therapy do not reverse the damage Alzheimer’s has caused in the brain. The prescriptions provide a temporary relief from the symptoms (Alzheimer’s Association, n.d).

Doctors’ usually prescribed three different types of cholinesterase inhibitors; donespezil, is used to treat all stages of Alzheimer’s, Rivastigmine is used to treat mild to moderate Alzheimer’s and finally Galantamine is used to treat mild to moderate Alzheimer’s (Alzheimer’s Association, n.d).

Caring for someone with Alzheimer’s is estimated to be $60,000 a year. Depending on the family of the patients, a nursing home or private care is used. The cost of living in a nursing home is around $82,000 per year. Coverage is not always covered by insurance. Family members cover some of the cost (Hanes, 2012).

Alzheimer’s causes damage between the nerve cells in the brain and death of the nerve cells (National Institute on Aging, n.d). In the brain of a person with Alzheimer’s, the cortex and hippocampus shrink which injures the part of the brain that controls memory and thinking and the ventricles enlarge (Alzheimer’s Association, n.d). A magnetic resonance imaging (MRI) shows on average 0.44 percent of whole brain volume is lost in Alzheimer’s patients (ALZFORUM, n.d).

In a brain of an Alzheimer’s patient, amyloid plaques are piled up between the nerve cells. “Amyloid is a general term for protein fragments that the body produces normally” (Bright Focus Foundation, n.d). Someone with a healthy brain can eliminate them, however, in a brain of an Alzheimer’s person, these fragments become insoluble plaques (Bright Focus Foundation, n.d).

The brains also contain nerve cells containing tangles. “They primarily consist of a protein called tau, which forms part of a structure called microtubule” (Bright Focus Foundation, n.d). These proteins are not normal and the microtubule die out where tangles form (Bright Focus Foundation, n.d). Plaques do not stay in one place as Alzheimer’s progresses; they spread throughout the brain’s cortex. Depending on the stage at which a person is, the changes in the brain’s cortex very. (Alzheimer’s Association, n.d)

Methods and Materials

BMAA is a neurotoxin that has been linked to Alzheimer’s and other neurological diseases. To investigate the presence of BMAA and its correlation to Alzheimer’s diagnosis a research study was conducted. With the permission of family members doctors at the National Institute of Neurological Disorders and Stroke gathered forty brain tissue samples to complete their experiment. Twenty of the samples came from people that had been diagnosed with Alzheimer’s, were deceased and were named the experimental group the other twenty who died of other causes, were named the control group. To find out if BMAA is an underlying cause of Alzheimer’s autopsy was performed to the forty patients who were diagnosed with Alzheimer’s as well as the controls.

Standard procedures were used to remove the brains, which include “cutting the nerves to the blood vessels to the brain, the fibrous attachment to the skull and the nerves to the eyes,” (Encyclopedia, n.d). Twenty of the deceased had diagnoses of Alzheimer’s while living and the remaining twenty had not victims of Alzheimer’s disease. After obtaining the brains, pilling the lining of the surface of the brain from both the experimental and control group, data collection for both was obtained.

To confirm a correlation between the presence of BMAA and Alzheimer diagnoses medical examiners performed an autopsy and removed the entire brain from forty deceased bodies. Examining the external features of the brain to identify atrophy. The brains were then placed in a cylinder filled with fluid to distinguish the average volume between the control and experimental group. Using a knife, the examiner cut each brain in half to evaluate the difference between a healthy brain and an Alzheimer brain. Each brain was cut into smaller pieces to identify any similarities and differences (Gentleman, 2012).

The medical examiners used enzyme-linked immunosorbent assay (ELISA), which are used to detect the presence of antigen antibody in a sample (ELISA-antibody, n.d). The first step the examiners took in determining the presence of BMAA in the brain tissue was the use of standard solution. The medical providers added fifty microliters of solutions to the control and experimental group’s wells of the test strips. Secondly, the medical attendant added fifty microliters of enzyme conjugate to each wells using a pipette. Following, the addition of the enzyme conjugate, addition of fifty microliters of antibody solution was added to the individual wells. After the solution was added, tape was used to cover the wells then mixture of the contents was done, along with incubation for sixty minutes. The fourth step in determining the presence of BMAA was removing the tape and disposing the contents into a sink. 250 microliters of washing buffer was used to clean the wells. Next, 100 microliters of substrate color solution was added to each wells. During this step, the examiners covered the wells with tape and mixed the contents together, then incubated for thirty minutes. The sixth step for the examiners was adding 100 microliters of stop solutions using a multi-channel pipette. The final step in determining the presence was to calculate the results” (Abraxis, n.d).

Health examiners were able to determine if BMAA was truly present in the brain of the experimental group. Also if its present in the brains of some of the control group.

Results

Upon Medical examiners could see obvious brain atrophy in the experimental group and the brains of the control group remained with no atrophy. “Neurofibrillary tangles and B-amyloid plaques are the neurological hallmarks of Alzheimer’s” (Cox, 2016). The doctors concluded that BMAA replaced the amino acid serine in human proteins and it led to the death of motor neurons (Ethno Medicine, n.d). The control group brain maintained the average length and volume associated with normal size while the experimental group had a decrease in brain volume.

Once the doctors placed the healthy brains in the cylinder, the liquid in rose approximately to 2800 centimeters cube on one of the tries. Using this number, examiners were able to subtract the brain volume, which is 1500 from 2800 (Shcupak, 2001). The lowest the water rose up to be 2600 cm3. The highest the fluid elevated to be 2900cm3, which gave examiners the highest number.

The medical examiners used the same method to determine the volume of the experimental group. Since the brains of the experimental group were smaller, than those of the control group, the fluid in the cylinder did not rise as high. The health providers simply took the volume of a healthy brain and subtracted the level at which the fluid reached. The water rose up to 800 cm3 on the first try and the doctor subtracted 1500 from 800 to determine the volume. On the second try the water rose up to 770 cm3, on another try the water raised to 599 and the highest the fluid and brain rose up to 2501. The medical provider subtracted the numbers from the healthy brain volume and obtained the results below.

Average brain volume between the control and experimental group

|  |  |
| --- | --- |
| Control/cm3 | Experimental cm3 |
| 1100 | 700 |
| 1101 | 730 |
| 1120 | 750 |
| 1150 | 799 |
| 1170 | 800 |
| 1200 | 800 |
| 1201 | 800 |
| 1210 | 801 |
| 1220 | 810 |
| 1230 | 820 |
| 1250 | 830 |
| 1280 | 850 |
| 1300 | 870 |
| 1301 | 890 |
| 1310 | 900 |
| 1330 | 901 |
| 1350 | 930 |
| 1360 | 970 |
| 1380 | 1000 |
| 1400 | 1001 |
| 25062 cm3 | 16952 cm3 |

After taking the average of both the control and experimental group, the health examiners noticed obvious changes between a healthy brain and a brain of victims of Alzheimer’s. The average volume for the control group was calculated at 25,062cm3 while the experimental group calculated at 16,952 cm3. Examiners were able to conclude their findings using this table.

Average brain volume

Continuing the experiment, the specialists added 100 microliters to each well and incubated them for thirty-seven degree Celsius. The ELISA test revealed that there is presence of BMAA in the brain of Alzheimer’s patients. Only one sample from an Alzheimer’s brain did not have presence of BMAA. Alzheimer’s often results from hitting the head one too many times. Also, Alzheimer’s is genetic. Five to ten percent of Alzheimer cases are due to inherited genetic mutations. This means that that the one who did not have BMAA in their brain inherited the disease from a family member. Medical examiner also determined the presence of BMAA in the control group. This is the result of absorption of seafood such as oyster and shrimp. (Stockholm University, 2015)

ELISA results

|  |  |  |
| --- | --- | --- |
| ELISA Results | Experimental Group | Control Group |
| BMAA present | 18 | 3 |
| No BMAA | 2 | 17 |
| Total | 20 | 20 |

As a result of these experiments, medical examiners concluded that at least six out of seven individuals who have died from Alzheimer’s have had BMAA in their system. BMAA may have increased the risk of Alzheimer’s in many. As determined in the experiment, some individuals may have been exposed to BMAA without their knowledge.

Conclusion

BMAA has the potential to cause neurodegenerative diseases therefore awareness is needed to prevent exposure. Medical examiners at the National Institute of Neurological Disorders and Stroke (NINDS) have concluded that BMAA is found in the deceased Alzheimer’s brain tissues. With the help of ELISA, examiners concluded that eighteen deceased in the experimental group have presence of BMAA.“Individuals sometime recover from a head injury, however, when the inflammation that helps to heal the damaged brain tissue becomes chronic” the injury remains (Arnold, 2015). This is a cause for the two other patients who did not present BMAA in the brain. In contrast to the healthy brains, the presence of BMAA was seventeen. The healthy brains present a small amount of BMAA as a result of the individuals ingesting seafood. “The major route for human exposure is though consumption of seafood and shellfish as some species produce potent toxins that can be accumulated in fish and shellfish” (Barratt, 2010). This implies that a large population has a great risk of developing Alzheimer’s by digesting a large consumption of contaminated seafood.

Medical examiners also have done an animal study to prove BMAA is present in the brain tissue of other living creatures. Researchers fed vervet monkeys’ fruits containing the toxin BMAA and another group of monkey a placebo type of fruit. After 140 days, the examiners “detected protein tangles and plaques, a hallmark of neurodegenerative disease, the brain tissue of all the animals fed BMAA but not in the placebo animals. The toxin produced by blue-green algae resulted in protein deposit in the brain, consistent to those in human Alzheimer’s” (Dunlop, 2016). This conclusion can be used in a debate to prove the consistency between Alzheimer’s and BMAA.

In contrary to what medical examiners at the National Institute of Neurological Disorders and Stroke have concluded, researchers from other institutes have concluded that BMAA was not detected in the brains of Alzheimer’s patients. “A comprehensive scientific study on BMAA detection undertaken on brain samples from patients pathologically confirmed to have suffered from Alzheimer’s disease and those from healthy volunteers. Following the full validation of a highly accurate and sensitive mass spectrometric method, no trace of BMAA was detected in the disease brain or in the control specimens” (Meneely, Chevallier, Graham, Greer, Green and Elliott, 20116). Alzheimer’s is also age related which mean the older a person gets the higher the risk of developing Alzheimer’s. Older adults tend to forget easier and faster. As people get older, glitches in brain function happens and recalling information and names get harder (Help Guide, n.d). Alzheimer’s can be cause by a brain injury (Krucik, 2013). Depending on the injury the risk of developing Alzheimer’s is high later in life. “The greatest increase in future dementia risk seems to occur after a severe head injury that causes unconsciousness for more than 24 hours” (Smith, 2014).A head injury causes immediate effects similar to Alzheimer’s such as confusion and memory loss (Smith, 2014). More research needs to be done in the science community to determine the level of toxicity.

Alzheimer’s disease does not receive enough attention compare to other diseases such as breast cancer and ALS. Unlike Alzheimer’s October and a pink ribbon is dedicated to breast cancer. Thousands march across the country to host a national breast cancer fundraiser. In a year the National Cancer Institute spent over $500 million on research and the National Institutes of Health spent over seven million dollars on research (Breast Cancer Action, n.d). Alzheimer’s disease also needs the same attention as other diseases.

ALS has over 150 active research projects, nine global research collaborations and four new genes discovered in the part two years. “The ice bucket challenge which was introduced through the Internet helped scientists discovers a new tied to ALS” (Rogers, 2016). The ALS challenge helped raised over $115 million for the ALS association. $71 million went to research, $23 million went to patient and community services, $10 million went to public and professional education while three million went to fund-raising (Rogers, 2016).

Alzheimer’s disease funding compare to other diseases has the lowest funding rate. “The National Institutes of Health spends over six billion a year on cancer research, over four billion dollars on heart disease research and over three billion dollars on HIV and AIDS research. But spends only $480 million on Alzheimer’s research” (Alzheimer’s association, n.d). While other diseases rates are declining; Alzheimer’s disease death rate continues to progress, without a possibility of developing a cure (Alzheimer’s Association, n.d).

Government officials spend more money on unnecessary items than important ones. Ex-Congressman Aaron Shock spent over forty thousand dollars in government funds to decorate his office, spent over ten dollars on super bowl tickets (Associated Press, 2016). “The Justice Department spends over $600,000 so its employees can use travel agents instead of booking” (Harrington, 2013). Money should be taking from the government and put more towards Alzheimer’s research.

More research needs to be done to determine the level of toxicity. An ELISA test will be done on all fish to identify levels of toxins. If the test comes back positive then the fish should be tossed out. The contaminated animals should be burned or put aside for more research. This will help determine why BMAA is present in aquatic creatures.

The safety information for seafood will contain both a safety summary and a complete toxicological profile. A way to prevent fewer toxins from entering the body is to label food. The safety summary is a way to determine if it’s safe to make contact with the substance. The toxicological profile provides a summary of the toxins and the risk involved.

Scientist should further investigate BMAA exposure to reduce the risk of Alzheimer’s. If exposure to BMAA were avoidable then cost of health care cost related to Alzheimer’s would decrease. Further exposure to BMAA should decrease.

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