NEURODEGENERATIVE DISEASES AND ASPARTAME

By

A thesis submitted by Spice Williams-Crosby
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ABSTRACT

In the United States today, there is an ever-increasing number of Americans that are afflicted with neurodegenerative diseases. Because the development of these diseases remains idiopathic, we must consider that diet and environmental factors play a major role. Due to the limited information on excitatory amino acid buildup in the body and the negative and degenerative effect it has on the human nervous system, medical professionals are limited when it comes to proper diagnosis. Over the past four decades, more and more people have unknowingly consumed excitotoxins that have been proven to cause brain damage, perhaps even brain lesions and tumors. There is now enough epidemiological evidence to point a finger to an environmental etiology for neurodegenerative diseases such as Alzheimer’s disease, Parkinson disease, Multiple Sclerosis (MS), and amyotrophic lateral sclerosis (ALS). That environmental factor is Aspartame. Aspartame (C₁₄H₁₈N₂O₅) is a compound of three components: aspartic acid, phenylalanine, and methanol. Aspartame causes neurons to die and mimics degenerative brain diseases.
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INTRODUCTION

In 1964, under the aegis of G.D. Searle and company, a group that included Dr. Robert Mazur, Dr. James Schlatter, Dr. Arthur Goldkemp, and the Imperial Chemical Company was formed to work on an ulcer drug that would act as an inhibitor to the gastrointestinal secretory, Gastrin (Stegink, 1984; Faber, 1989). While determining the strength or biological activity of that substance, an intermediate chemical was synthesized. That new chemical, aspartylphenylalanine-methyl-ester, was given the name Aspartame.

![Chemical structure of Aspartame]

*Figure 1. The chemical structure of Aspartame - Aspartame consists of three components: aspartic acid (a nonessential amino acid), phenylalanine (an essential amino acid), and methyl ester which is metabolized to free methyl alcohol, or methanol.*

In December of 1965, Dr. Schlatter was working with Aspartame during the process known as recrystallization, a procedure for purifying compounds. Dr. Schlatter was recrystallizing Aspartame from ethanol, when the mixture spilled onto the outside of the flask. Some of the powder got onto his fingers. He licked his fingers in order to pick up a piece of paper and suddenly became aware of a very strong, sweet taste. His discovery of that powder was reported
in 1966 with no mention of the sweet taste (Furia, 1972). It was Dr. Mazur who reported this discovery of an artificial sweetener in the Journal of the American Chemical Society in 1969 (Mazur, 1969).

Dr. Harry Waisman, a biochemist, was a Professor of Pediatrics and the Director of the University of Wisconsin's Joseph P. Kennedy Jr. Memorial Laboratory of Mental Retardation Research when he was contracted by G.D. Searle to conduct a study of the effects of Aspartame on primates. The study began on January 15, 1970 and was terminated on April 25, 1971 after Dr. Waisman’s death in March 1971. The study involved seven infant monkeys who were given Aspartame with milk. The monkeys were divided into three groups. The low dose group was given 1.0 g/kg of Aspartame in their milk. A medium dose group, 3.0 g/kg, and a high dose group 4-6 g/kg were also fed the same milk laced with Aspartame and administered orally however, the high dose group did not consume intended levels of Aspartame during the study. This was thought to be due to the overt sweetness of the Aspartame (200 times greater than sugar). Thus, researchers involved in this study concluded, the high-dose group actually ingested approximately as much Aspartame as the medium-dose group. Because of Dr. Waisman’s death early on, the low-dose group of monkeys were pulled from this study at about 200 days prior to when brain seizures commenced for the medium and high-dose groups (Stoddard, 1995; Merrill, 1977; Graves, 1984; Congressional Record, 1985; Gross, 1976).

All medium and high dose monkeys showed increased phenylalanine levels in their blood. All medium and high dose monkeys exhibited brain seizures, starting about seven months into the experiment. The study reports "All animals in the medium and high dosage groups exhibited seizure activity. Seizures were observed for the first time following 218 days of treatment. The seizures were of the grand mal type. One monkey, m38, of the high dose group,
died after 300 days of treatment. The cause of death was not determined” (Rao, McConnell, & Waisman, p. 9). After 300 days, one monkey died and five others had grand mal seizures. These results were never shown to the Food and Drug Administration (FDA) when G.D. Searle submitted its findings. In fact, G.D. Searle denied any involvement whatsoever in the study (Stoddard, 1995).

In 1970, Neuroscientist and researcher, Dr. John W. Olney found that oral intake of aspartic acid caused brain damage in mice, and he informed G.D. Searle of his findings (Olney, 1970). He revealed in his findings that the transport of excitotoxins across the blood brain barrier and within the cerebral spinal fluid (CSF) caused several reactions to occur. 1.) The excitotoxins stimulate the nerves to fire excessively. 2.) The normal enzyme actions required to offset the induced, repeated firing of these neurons are negated by the phenylalanine and aspartic acid. 3.) The energy system for the required enzyme reactions becomes compromised from depleted intracellular ATP stores. 4.) The presence of formaldehyde alters intracellular calcium (Ca+) uptake. 5.) Damage to cellular mitochondria, destruction of the cellular wall, and the subsequent release of free radicals potentiates oxidative stress and neurodegeneration (Olney, 1970).

Dr. Olney also expressed that these toxic by-products instigate secondary damage by increasing capillary permeability, which continues to destroy the surrounding nerve and glial cells. This expedites enzyme reactions, and promotes DNA structural defects (Olney, 1970; Bowen & Evangelista, 2002).

Dr. Olney reported that cellular death occurs over 1 to 12 hours. This does not include the long-term or cumulative effects of other metabolites. With each test he conducted, he discovered that the dead cells leave behind lesions. His evidence showed that the following
disease states could be clinically identified by their corresponding anatomic nerve fiber, or nerve bundle damage:

1) Aqueduct of Sylvius - Hydrocephalus
2) White matter bundles - Multiple Sclerosis (MS)
3) Pyramids/Basal Ganglia - Parkinson's Disease
4) Lateral corticospinal tracts of spinal cord and bulbar nuclei - Amyotrophic lateral sclerosis (Lou Gehrig's)
5) Destruction of hypothalamic regions - Neuro-endocrine disorders, obesity, psychogenic disorders (behavior, anger) malfunction of autonomic nervous system, and immune suppression. (Bowen and Evangelista, 2002)

G.D. Searle responded by hiring Dr. Ann Reynolds, a researcher who had done research for the Glutamate (MSG) Association, to confirm Dr. Olney’s tests. Dr. Reynolds confirmed Aspartame was, in fact, a neurotoxin in infant mice (Reynolds, 1971). Aspartame, a neurotoxin, excites the neurons to death and hence the name, excitotoxins, given this title by Dr. John Olney (Whetsell, 1993).

By March of 1973, G.D. Searle’s petition for approval to market Aspartame as a sweetening agent was published in the Federal Register to the FDA. Martha M. Freeman, M.D., of the FDA Division of Metabolic and Endocrine Drug Products, addressed the credibility of the information submitted by G.D. Searle in their petition to approve Aspartame in an FDA memorandum dated September 12, 1973 (Freeman, 1973). Her complaint was that all of G.D. Searle’s studies had been single-dose studies. Dr. Freeman pointed out, as early as 1973, the inadequacy of single-dose tests of Aspartame as compared to multiple dose studies. Since then,
the NutraSweet® Company has continued to publish single-dose studies when most of the experimental studies to determine physical harm work in a dose-dependent fashion.

Due to the uncertainty of the regulatory future on Aspartame, construction of a large Aspartame manufacturing plant in Augusta, Georgia was halted and G.D. Searle commissioned Ajinomoto in Singapore, the inventor and main producer of the food additive monosodium glutamate, to mass-produce Aspartame in commercial quantities (Farber, 1989).

On July 26, 1974, FDA commissioner Alexander Schmidt approved the use of Aspartame in dry foods only. It was not approved for baking goods, cooking, or carbonated beverages (Farber 1989; Federal Register 1974). Despite the fact that FDA scientists found serious discrepancies in all 13 tests related to genetic and neuron damage submitted by G.D. Searle, the sweetener was approved and the agency made public, for the first time, data supporting the food-additive decision.

Following that decision, Dr. John Olney and consumer interest attorney, James Turner, author of a 1970 book about food additives, objected to the decision because Dr. Olney and his team had linked Aspartame to brain lesions in mice. Dr. Olney and James Turner filed a complaint at the FDA objecting to the approval and were able to hold it off the market until 1981. They were particularly worried about Aspartame's effects on children (Graves, 1984; Congressional Record, 1985; Federal Register, 1975; Olney, 1987).

Dr. Alexander Schmidt, in 1975, appointed a special Task Force headed by Philip Brodsky, FDA's Lead Investigator and assisted by FDA Toxicologist, Dr. Adrian Gross, to look at 25 key studies for the food additive Aspartame. All of the studies, whether conducted at G.D. Searle or Hazleton Laboratories, were under the supervision of the Pathology-Toxicology
Department at G.D. Searle (Gross, 1987). FDA Toxicologist and Task Force member, Dr. Andrian Gross stated:

They [G.D. Searle] lied and they didn't submit the real nature of their observations because had they done that it is more than likely that a great number of these studies would have been rejected simply for adequacy. What Searle did, they took great pains to camouflage these shortcomings of the study. As I say filter and just present to the FDA what they wished the FDA to know and they did other terrible things for instance animals would develop tumors while they were under study. Well they would remove these tumors from the animals (Congressional Record, p. S10826-S10827).

Phillip Brodsky stated that he had never seen anything as bad as G. D. Searle's studies (Graves, 1984; Congressional Record, 1985).

When G.D. Searle technicians were caught removing the brain tumors from the rats, they claimed that the rats couldn't breathe well. Dr. Adrian Gross, gave several reasons why Searle's misconduct invalidated their experiments and stated, "It is highly unlikely that the FDA investigative teams found all of the problems with G. D. Searle's studies. G. D. Searle seemed so intent on covering up their misconduct, that it is quite likely that they were able to hide many of the problems from the FDA." (Congressional Record, p. S10826-S10827) According to Dr. Russell Blaylock, no independent studies have been done to examine this vital issue. (Blaylock, 2003).

On December 5, 1975, the FDA reversed their original decision and put a hold on their approval of Aspartame due to the preliminary findings of Dr. Olney’s research as presented to
the FDA Task Force. The Public Board of Inquiry was also put on hold (Federal Register, 1975; Mullarkey, 1994).

Still unable to achieve an approval status for Aspartame, G.D. Searle met with the FDA on August 4, 1976 for consent to continue testing by hiring a private agency, Universities Associated for Research and Education in Pathology (UAREP). (Graves, 1984; Congressional Record, 1985).

G.D. Searle had invested 19.7 million dollars in an incomplete production facility and 9.2 million dollars in Aspartame inventory (Farber, 1989). Donald Rumsfeld, a former member of the U.S. Congress and the Chief of Staff during the Gerald Ford Administration, was hired as president of G.D. Searle in 1977. Attorney James Turner believes that G.D. Searle hired Rumsfeld to handle the Aspartame approval difficulties as a "legal problem rather than a scientific problem" (Gordon, 1987, p. 497 as cited in US Senate, 1987). Perhaps, G.D. Searle needed someone of political stature that could help them reap the harvest of their investments.

In August 1977, the Bressler report was released. This FDA audit of studies (E5, E77/78, E89) was performed by a team of scientists led by Dr. Jerome Bressler and written by Dr. Bressler. Jerome Bressler said that G.D. Searle’s scientists’ “studies on Aspartame were so bad FDA removed 20% of the worst of his report when retyped.” Some of the flaws in the three studies found by the Bressler-led FDA Task Force included missing raw data, errors and discrepancies in available data, exclusions of animals, organ masses and enlarged and atrophied organs. An undiagnosed uterine polyp increased the incidence to 15 percent of the Aspartame-dosed animals and other multiple discrepancies (Roberts, 1990). For each of the major discrepancies found by the Task Force, the FDA Bureau of Foods minimized the problem (Gordon, 1987). Dr Jacqueline Verrett, the senior scientist of the FDA Bureau of Foods review
team created to review the Bressler Report said, "It was pretty obvious that somewhere along the line, the bureau officials were working up to a whitewash" (Gordon, 1987, p. 497 as cited in US Senate, 1987). In 1987, Verrett testified before the US Senate stating that the experiments conducted by Searle were a "disaster." She stated that her team was instructed not to comment on or be concerned with the overall validity of the studies (Gold, 1995).

On September 30, 1980, the FDA Public Board of Inquiry comprised of Dr. Walle J. H. Hauta, M.D., Ph.D. Chairman; Dr. Peter W. Lampert, M.D. member; and Dr. Vernon R. Young, Ph.D. member, voted unanimously to reject the use of Aspartame until additional studies on Aspartame's potential to cause brain tumors, neurodegenerative diseases and gastrointestinal diseases could be done (Brannigan 1983). The Board of Inquiry found that Aspartame becomes a deadly poison at 86 degrees Fahrenheit, Aspartame converting to formaldehyde above 86 degrees Fahrenheit, and then to formic acid, and finally to diketopiperazine (DKP), a known brain carcinogen (Martini, 1995).

In Docket No. 75P-0355, the Department of Health and Human Services of the Food and Drug Administration reported the Public Board of Inquiry’s decision on Aspartame:

On the basis of the conclusion concerning Issue Number 2, the Board concludes that approval of Aspartame for use in foods should be withheld at least until the question concerning its possible oncogenic potential has been resolved by further experiments. The Board has not been presented with proof of reasonable certainty that Aspartame is safe for use as a food additive under its intended conditions of use. The foregoing constitutes the Board's findings of fact and conclusions of law. Therefore, it is ORDERED that: 1. Approval of the food additive petition for Aspartame (FAP 3A2885) be and it is hereby withdrawn. 2.
The stay of the effectiveness of the regulation for Aspartame, 21 CFR 172.804, is hereby vacated and the regulation revoked (Decision of the Public Board of Inquiry Docket No. 75P-0355, 1980, p.49).

The day after Ronald Reagan took office as U.S. President in 1981, G.D. Searle reapplied for the approval of Aspartame, submitting several new studies along with their application. It wasn’t long before President Reagan replaced Jere E. Goyan, Ph.D., who was at that time the FDA Commissioner (Gordon 1987; US Senate, 1987). Dr. Goyan was the first pharmacist to serve as Commissioner of Food and Drugs and served in that position from October 1979 to January 1981. Before Goyan was replaced by Regan appointee Dr. Arthur Hall Hayes Jr., he had set up a five-member "commissioner's team" of scientists with no prior involvement in the Aspartame issue to review the board's ruling. On May 18, 1981, one month after the appointment of Dr. Hayes, three scientists of the 5-member panel sent a letter to the panel lawyer, Joseph Levitt. Those three scientists were Satva Dubey (FDA Chief of Statistical Evaluation Branch), Douglas Park (Staff Science Advisor), and Robert Condon (Veterinary Medicine). In the letter, they made their concerns very clear about the use of Aspartame and claimed brain tumor data was so "worrisome" in one particular study that Mr. Levitt could not recommend the Aspartame be approved (Gordon 1987; US Senate, 1987, p. 495).

Searle petitioned for FDA approval again in 1982 to use the sweetener in diet soft drinks and children's vitamins, claiming that the sodas were cooler than 86 degrees (Gordon, 1987; Farber 1989) and the approval was granted. Within weeks, Dr. Arthur Hull Hayes, Jr. resigned from his position, and became a consultant to Burson-Marsteller public relations firm representing the NutraSweet Co. (Evangelista, 2004).
In 1983, despite the questions and revolving door issues, the FDA was satisfied in supporting Aspartame safety, with the exception of people with the rare disease phenylketonuria, and Aspartame was approved. However, what is becoming clear is that Aspartame is an excitotoxins and a neurotoxin (Whetsell, 1993).

**Excitotoxins and the Brain**

The brain, weighing only three pounds, is made up of 60% fat, due to myelin, and has large concentrations of amino acids. These are carefully regulated because so many amino acids serve as neurotransmitters or transmitter precursors. Each amino acid performs a specific duty, and without careful control of these substances, our brains would not be able communicate properly with our bodies. Neurotransmitters are chemicals that allow the movement of information from one neuron, across the synaptic gap, to an adjacent neuron. Glutamate and aspartate, which are neurotransmitters, are electrically active and process information to be transmitted to specific neurons.

There are billions of neurons in the human brain and they all have specific jobs. Some are involved with thinking, learning, and memory. Others are responsible for receiving sensory information. Still others communicate with muscles, stimulating them into action.

Each neuron consists of a cell body, an axon, and many dendrites. The cell body contains a nucleus, which controls all of the cell's activities, and several other organelles that perform specific functions. Two types of processes extend from the cell body. The axon, which is much narrower than the width of a human hair, and transmits messages to other neurons. Messages may sometimes travel over very long distances. Dendrites receive messages from the axons of other nerve cells. Each nerve cell is connected to thousands of other nerve cells through its axon and dendrites. Additionally, neurons are surrounded by glia cells that support, protect,
and nourish them. Several processes that involve communication, metabolism, and repair, all have to work smoothly together for neurons to survive and stay healthy.

In addition to being electrically active, neurons constantly synthesize neurotransmitters. Aspartate and glutamate are important neurotransmitters that allow neurons to communicate between each other. Normally, any excess aspartate and glutamate in the extracellular fluid is pumped back in the glial cells surrounding the neurons. However, when particular types of neurons are exposed to excessive amounts of aspartate and glutamate, they are overstimulated and the cells die (Coyle, 1981).

In 1968, Dr. Olney, working out of the Department of Psychiatry at Washington University in St. Louis, repeated a study by Dr. Lucas and Newhouse, using the same animal model and the same doses of monosodium glutamate (MSG) and aspartate, one of the main ingredients in NutraSweet (Aspartame). What Dr. Olney found was that not only did MSG and aspartate cause severe damage to the neurons in the hypothalamus, but it also caused widespread destruction of neurons in other areas of the brain adjacent to the ventricular system, called the circumventricular organs (Olney, 1969).

Figure 2. Circumventricular Organs of the Brain. These areas lack a blood-brain barrier. Illustration by Dr. Russell L. Blaylock.
Because the hypothalamus plays such an important role in controlling so many functions, this discovery by Dr. Olney is particularly important. Since the FDA approved Aspartame, the mounting evidence of this type of destruction to neurons by aspartate is being demonstrated in over 92 symptoms, including death, registered at the FDA. (Department of Health and Human Services, 1995).

The wiring of the hypothalamus is some of the most complex in the nervous system, with vital connections to the pituitary gland, the limbic system, the hippocampus, the striatum and the brain stem. The pituitary, the master gland, is only about the size of a white navy bean, and yet it is responsible for controlling some of the most vital hormonal systems in the body, including those of the endocrine glands. It also controls the adrenals, the thyroid, and the reproductive organs by releasing small amounts of its controlling hormones into the blood stream, sending messages to other endocrine organs to regulate secretion of their hormones. The hypothalamus, no larger than a fingernail, works with the body’s pituitary gland to help regulate emotions, autonomic control, parasympathetic and sympathetic responses, hunger satiety, immunity, memory input, and anger control. If these vital brain functions experience any disruption, it can result in anything from minor behavioral problems or endocrine malfunctions to major disruptions in sexual function, obesity, immune suppression, and endocrine gland failure.

It is now known that the hypothalamus is associated with neurological diseases caused by injuries or assaults that create lesions by the di-peptide of phenylalanine and aspartate, known as Aspartame (Blaylock, 2000). Among the many neurons in the hypothalamus, called nuclei, the arcuate or curved nucleus is consistently the most sensitive to Aspartame toxicity. This nucleus regulates growth hormone secretions with the help of the pituitary, and with its association with other nuclei, such as the supraoptic nucleus and paraventricular nucleus. It has been
demonstrated many times in laboratory studies that these excitotoxins cause shrinkage of the pituitary, thyroid, adrenals and gonads in animals exposed to high concentrations of Aspartame (Blaylock, 2000).

The hypothalamus is one of the areas of the brain that is not protected by the blood-brain barrier and extremely fragile to excitotoxins. Aspartate is a major neurotransmitter in the hypothalamus and therefore excess concentrations of it will affect all of the various nuclei in the hypothalamus.

In order to understand this destructive process, it is important to understand how excitatory amino acids were discovered and how they cause the neurons to fire spontaneously and repeatedly. In the early part of 1950, Dr. T. Hayaski, a neuroscientist, was experimenting on a dog by injecting monosodium glutamate into the gray matter of a dog’s brain. The dog collapsed in its cage and immediately began to convulse. Dr. T. Hayaski concluded that the glutamate was triggering the dog’s brain cells to become overexcited and fire uncontrollably (Blaylock, 1997). In 1959, in a different lab, two other researchers, Dr. A. Van Harreveld and Dr. M. Mendelson placed glutamate and aspartate on the muscle tissue of invertebrate crustaceans and they noted that the muscle tissue contracted violently (Van Harreveld & Medelson, 1959). They recognized these amino acids as being part of a new functional category of molecules and gave them the name of “excitatory amino acids” because they caused nerve cells to become excited (Blaylock, 1997). Since then, over seventy excitatory aminos acids have been discovered. It was not until 1973 that these excitotoxins would be demonstrated to be neurotransmitters.
Neurons communicate with each other across a tiny fissure known as the synaptic cleft. The electrical impulse is carried from the axon terminal of the pre-synaptic cell to the receptors on the dendrites of the postsynaptic cell. However, they never physically touch one another.
Neurotransmitters are endogenous chemicals and are manufactured in the neuron’s cell body. The transmission of information from one neuron to another depends on the ability of the information to traverse the synapse between the terminal end of one neuron and the receptor end of an adjacent neuron. The transfer is accomplished by neurotransmitters. The neurotransmitter molecule attaches to a receptor on the membrane of the dendrite and causes the molecule in the membrane to change. This is likened to a lock and key system. The receptor on the membrane is the lock and the neurotransmitter is the key. As you know, not any key can open any lock. It must be a specific key that will fit perfectly. When this happens, it opens a microscopic channel within the membrane and lets sodium and/or calcium spew inside the axon, which in turn triggers the cell to fire a transmitting signal down its axon fiber.

*Figure 3. When a nerve impulse passes the axon terminal, its synaptic vesicles release their stored chemicals into the synaptic cleft. These diffuse through the cleft to reach the membrane of the next neuron, stimulating the latter. This causes the nerve impulse to be transmitted to the next neuron.*
It is now well known that two of the most common neurotransmitting chemicals, glutamate and aspartate, found normally in the brain and spinal cord, will become neurotoxic to the neurons containing glutamate receptors and to the nerves connected to these neurons when their concentrations rise above a critical level (Blaylock, 1999). This means that not only will the neurotoxic levels kill the selected neurons, but also kill any neurons that happen to be connected to it, even if that neuron uses another type of receptor. For this reason, the nervous system keeps a tight control on the concentration of these two amino acids in the extracellular space. This is done by a system designed to remove any excess glutamate from this surrounding fluid. A special pump system is set in place to transfer the excess glutamate back into surrounding glial cells that supply the neurons with energy. If this pump fails, the destruction is inevitable.

This pump system requires an immense amount of cellular energy that is supplied by an energy carrier known as adenosine triphosphate (ATP). ATP is a molecule that is the immediate
source of energy for all cellular activity, including muscle contraction. It is an organic compound composed of adenine, ribose, and three phosphate groups. This is a very unstable molecule primarily because the phosphate groups contain negative electrical charges that repel each other. However, when the phosphate groups break free, energy is released. This tri-molecule is the spark plug that transports chemical energy within cells for metabolism, and is also involved in the activation of amino acids, a necessary step in the synthesis of protein. When ATP loses one of its phosphate groups, and this happens after the process of hydrolysis instigated by the enzyme ATPase, it is brought down to a di-molecule called, adenosine diphosphate (ADP). The muscle contraction is powered by the breakdown of these two molecules, ATP and ADP. If the ADP loses a phosphate and becomes adenosine monophosphate (AMP) and runs out of energy, then once again, destruction is inevitable. Eventually, through a donation of phosphate from creatine, ATP will be restored.

This glutamate pumping system is likened to a boat in the water with a boat crew on board. If the boat springs a leak, then the boat crew becomes the bucket brigade, scooping up water as it is filling up the sinking boat. If the crew is tired and runs out of energy, the boat will fill up with water and sink. The same thing happens when energy production is reduced in the brain. If the ATP is not restored to the neurons, as mentioned in the previous paragraph, then they will die or be excited to death, thus, the term, excitotoxins. Excitotoxins are biochemical substances, usually amino acids, amino acid analogs, or amino acid derivatives, that can react with specialized neuronal receptors, such as glutamate receptors, in the brain or spinal cord in such a way as to cause injury or death to a wide variety of neurons.

Studies have shown within fifteen to thirty minutes of highly concentrated dosages of excitotoxins (MSG, glutamate, Aspartame), suspended in tissue culture, the degeneration of the
organelles within the cell and clumping of the chromatin in the nucleus is visible under a microscope. Within three hours, not only have the neurons died, but the body’s defense mechanism has begun the process of hauling away debris. This in turn puts enormous stress on the body’s systems (Choi, 1990).

Figure 5. When a neuron is exposed to a massive dose of an excitotoxin, the cell immediately begins to swell and dies within one hour. Within two hours, the macrophages begin to clear the remains. When a lower dose is administered, nothing happens until the second hour. This delayed death of the neurons is characteristic of excitotoxins. Illustration by Dr. Russell L. Blaylock.

However, with exposure to lower doses of excitotoxins, neurons during the first 15 to 30 minutes appear to be perfectly normal and unharmed. It is not until the second hour when the cells begin to commit apoptosis. Figure 5 represents the progression over the course of two hours from the start of exposure (Coyle, 1981).

What these studies demonstrate is that two different reactions create cellular destruction: acute and delayed. The acute reaction that happened within the first hour mimicked the result of
a massive influx of sodium to the inside of the neuron. Having a rapid movement of sodium into the cell causes it to swell due to osmotic movement of water into the cell; as it swells out, it bursts and dies. Sodium enters the cell by a selective channel or pore that is controlled by special triggering chemicals. Exogenous glutamate isolates acts as a trigger to open the sodium channel on the cell’s membrane. No matter what concentration of excitotoxins was added to a culture of sensitive neurons, the cells would die during the critical two-hour period (Rothman, 1985; Lucas & Newhouse 1957).

There was no effect on the delayed reaction when removing the sodium. After two hours the neurons still died. At the time, the scientists were considering another channel that might explain the delayed reaction. The study was repeated and that time the scientists removed calcium from the tissue medium. They waited the allotted two hours with no cellular death, then 24 hours and still no cellular death. Calcium appeared to be implicated in the delayed response. Putting it all together, they realized that glutamate opens a special channel designed to allow calcium to enter the neuron, and it was calcium that triggered the cell to die (Blaylock, 1997).

Neurons contain calcium channels that regulate the movement of calcium into the cell. These are important to creating a normal environment inside each neuron and playing a vital role in the activation of neurons and the transmission of their impulses. The calcium channel, once it has been stimulated, will open for no more than a fraction of a second, and only then are minute amounts of calcium allowed to enter the neuron. There is a special protective pump set in place in case too much calcium enters the neuron. This special calcium pump drives the excess calcium back out of the neuron; some of the calcium is also captured and stored within the endoplasmic reticulum of the cell.
Asparate and all excitotoxins appear to work by opening the calcium channels of specific receptors. When these neurotransmitters are allowed to come into contact with the receptor in too high a concentration or for too long a period of time, the calcium channel is forced to stay open (Blaylock, 2000). As the calcium pours into the cell, the cell will explode and die. Just like the bucket brigade uses up its ATP when the boat is beginning to fill up and sink, so too is the calcium pump in dire need of ATP for energy to help siphon out all the excess calcium (Blaylock, 1997).

When dealing with glutamate receptors, it gets a bit complicated. Glutamate is the key and the glutamate receptor on the membrane is the lock, but it is now believed that there are more than twenty sub-types of glutamate receptors on the cell membrane (Watkin & Evans, 1981). It was discovered that a substance that was being used by scientists, called N-methyl-D-aspartate or NMDA, a glutamate analogue, stimulates only certain classes of glutamate receptor and not others. Another substance, called quisqualate, was found to stimulate a completely different set of glutamate receptors, and then a third receptor sub-type was found that responds only to the chemical kainite. All three sub-types of glutamate receptors on the nerve cell membranes can be stimulated with glutamate and/or aspartate (Monaghan, Bridges & Cotman, 1989). NMDA acts as the gatekeeper of the calcium channel on the cell membrane and regulates the entry of calcium into the neuron (Watkins & Evans, 1981). Both glutamate and aspartate can open this calcium channel. Therefore, unlike other lock and key neurons and neurotransmitters, with the NMDA receptor more than one key is required.

The zinc receptor, magnesium receptor, and glycine receptor are the other locks on the membrane. Zinc locks the door and closes the calcium channel tight. Magnesium also locks the door, but there is a notable difference between the two. When zinc locks the door on the calcium
channel, it remains bound even if the neuron has been fired, whereas when magnesium locks the calcium channel, it is automatically released when the neuron fires. Glycine is another amino acid necessary for the calcium channel to open. Scientists found that when glycine is removed from a culture of nerve cells, no concentration of glutamate would make the nerve cell fire. However, when glycine is added to a culture of nerve cells, the neurons become much more sensitive to the excitotoxins and, if they are not protected or rescued, they will eventually be destroyed (Choi, 1989).

Once glutamate or aspartate come into contact with the receptor, they slide into the lock, the glutamate receptor, like a perfect fitting key. Then glycine is inserted into its lock close by. When normal levels of magnesium and low levels of zinc are present near the neurons, the channel will then open wide, and calcium and sodium will pour into the neurons, causing it to fire (Kleckner & Dingledine, 1988).

During the opening of the calcium channel in NMDA and glutamate receptors, the excess intracellular calcium also activates nitric oxide synthase (NOS), which generates excessive amounts of nitric oxide (NO). The NO then reacts with superoxide to form peroxynitrite radical, which is a very powerful reactive nitrogen species (RNS). This RNS passes through the mitochondrial membrane with great speed and has been shown to be especially damaging to mitochondrial enzymes and mitochondrial DNA (mtDNA) (Christopherson & Bredt, 1997).

Neurons communicate with other neurons. The nervous system functions by passing information from neuron to neuron. This neuron-to-neuron communication has a language and it is made up of various chemicals that are specific chemicals used to send out special messages around the inside of the cell.
As calcium enters the cell, it activates a mediator called protein kinase C. This enzyme mediates the phosphorylation of certain cellular proteins and is in charge of such important functions as cell growth, ion channel activity, secretion, and the mechanisms by which a presynaptic neuron influences the activity of a nearby postsynaptic neuron, known as a synaptic transmission. Protein kinase C causes more calcium to be released from a special calcium storage site within the cell, called the endoplasmic reticulum. Because of this, more calcium pours into the cytoplasm and, at times, can alter the membrane of the cell causing the calcium channel to be inactivated. When this happens, calcium continues to pour into the cytoplasm of the cells, which in turn, sends signals to another enzyme called, phospholipase C. This enzyme is responsible for breaking down some of the fatty acids within the plasma phospholipid membrane and compartmental membranes. Excess calcium within the cell is also destructive to the cell's mitochondria, structures that are involved in the cell's ability to produce ATP. Mitochondria soak up excess calcium until they swell up and stop functioning. If enough mitochondria are damaged, nerve cells degenerates (Nairn, Hemmings, & Greengard, 1985).

In the process of breaking down this phospholipid membrane, arachidonic acid is released. Once in the interior of the cell, arachidonic acid can cause great harm to the cell, especially if it is in high concentration (Farooqui & Horrocks, 1998)

As arachidonic acid is released in the interior of the cell, it becomes a target for two other enzymes, lipoxygenase and cyclooxygenase (COX). These two enzymes start digesting the arachidonic acid and thus the destruction begins. A series of reactions takes place that produces a number of chemicals that trigger prostaglandin synthesis, thus creating free radical formation and rapid cell death. This cascade of destruction starts because of a deregulation of calcium,
instigating the cell to swell and explode. A by-product of this reaction is the creation of free radicals (Blaylock, 1997).

In order to understand the extent of damage that takes place within the neuron when this cascade of destruction occurs, it is important to understand what a free radical is. Atoms are most stable in the ground state (the state of least possible energy in a physical system). An atom is considered “grounded” when every electron in its outermost shell has a complementary electron that spins in the opposite direction. By definition, a free radical is any atom with at least one unpaired electron in its outermost shell, that is capable of independent existence. A free radical is easily formed when a covalent bond between atoms is broken and one electron remains with each newly formed atom. Free radicals play a central role in almost every injury and disease known to man, from cancer to neurodegenerative diseases (Stadtman, 1992).

Free radicals are not only produced during disease or injury, but also are inadvertently produced when energy is utilized in the cells during metabolism. Free radicals involving oxygen are referred to as reactive oxygen species (ROS). Some oxygen molecules that have become free radicals are unstable, highly reactive, and react with the plasma phospholipid and organelle membranes, weakening the structure of the cell and disrupting the cell’s function. Once these free radicals are released, they react indiscriminately with many molecules destroying everything, even the genes (Packer & Colman, 1999). Each of our cells suffers over 10,000 hits per day from free radical molecules (Cherniske, 2003).
Figure 6. Shows how calcium activates destruction reaction from within the neuron by triggering prostaglandin synthesis and free radical formation. Illustration by Dr. Russell L. Blaylock, M.D.

Not all neurons die because of excitotoxins. Excitotoxins are very selective in attaching themselves to specific neurons. Their targeting is based on specific receptors on some neurons for glutamate and not others. Almost all excitotoxins, such as aspartate, attach themselves to the glutamate receptors on the membranes and stimulate the neurons (Choi, 1988).

As a neurotransmitter, glutamate is found in about 50% of the forebrain synapses of all mammalian brains. The fact that these receptors are concentrated in specific brain areas that are affected by Alzheimer’s disease, Huntington’s disease, and Amyotrophic Lateral Sclerosis (ALS), suggests that these excitotoxins play a role in these diseases (Maragos, 1987; Spencer, 1987; Plaitakis, 1990).

A broad range of chronic neurodegenerative diseases, such as Alzheimer’s disease, Parkinson's disease, ALS, multiple sclerosis (MS), and dementia are now believed to be caused by the excitotoxic action of glutamate and aspartate. What is true about the characteristics of all these neurodegenerative diseases is that they all develop in normal brains and slowly progress into death. Each disease appears to have a specialized group of cells that are affected by excitotoxins.
It was first believed that these specific neurons lived a shorter life than normal neurons, and that their accelerated aging process was built into their genetic coding. W.R. Gowers, a famous neurologist at the turn of the century, first popularized this theory calling the process “abiotrophy.” However, as science evolved, so did our understanding of how and when neuron death occurs. It is understood today that as the neurons are being destroyed and meeting their deaths because of excitoxins or genetic factors, the symptoms do not appear until much later in life. As scientists observe in Parkinson’s disease or Alzheimer’s disease, the symptoms do not manifest themselves until over 80 to 90% of the specific neurons have died (Calne, Michael & Zigmond, 1991). These neurons did not just die all at once. Dr. Russell Blaylock refers to this degrading process as the “Creeping Death.”

Why does this happen to certain neurons and not to others? Some scientists believe that these specific neurons begin to slowly die due to an autoimmune condition wherein the immune system begins by attacking the nervous system. However, the results of extensive studies to make that connection to the neurodegenerative diseases are not that convincing (Blaylock, 1997). The most plausible connection, that makes scientific sense, outside of genetics, is that toxins in the environment are causing neurodegenerative diseases (Kurland, 1988).

From 1940 to 1980, epidemiologists studied the Chamorros Indians, natives of the Mariana Islands in Guam that were dying from a mysterious disease. The natives began to waste away and became too weak to stand or even swallow their food. Eventually, the doctors realized that this disorder resembled ALS. What brought them to the hypothesis that these diseases would have an environmental connection was the fact that these native Indians had fifty to hundred times higher incidents of ALS than developed countries, suggesting clear that something was attacking them from their own environment (Kurland, 1988).
What was affecting them was a neurotoxin found within the food they were consuming. The natives ate a large amount of a plant called cyca circinalis, or cycad, from the false sago palm. Cycad has a toxic compound called β-N-methylamino-L-alanine (L-BMAA) which has been found to cause seizures in mice. One researcher, Dr. George Spencer, found another toxic compound, called β-oxallylamino-L-alanine (L-BOAA), that caused sudden onset of weakness and paralysis in the legs. While continuing to research, Dr. Spencer came across a report about a single monkey fed a concentrated solution of L-BMAA for several weeks (Kurland, 1988). The monkey developed the same disease the natives were dying of in Guam. When Dr. Spencer necropsied the monkey, he found that it had the identical pathological changes in its spinal cord and brain as did the natives. Dr. Spencer repeated the same experiment again, only this time on thirteen monkeys. After two to twelve weeks of consuming a concentrated solution of L-BMAA, the same monkeys exhibited signs of ALS and Parkinson’s disease. They developed signs of severe weakness in their limbs, a shuffling gait, and a blank stare with a mask-like expression on their face (Spencer, Nunn & Hugon, 1987).

Everyone has a different sensitivity to toxins. Some respond immediately, others experience no symptoms at all until it is too late, and some remain resistant to certain toxins with no side effects. Resistance can depend on the protective mechanisms within a person’s blood brain barrier (BBB), the ionic and glutamate pumps, and the free radical scavengers. Many immigrants from Guam migrated to the United States and while they appeared to be normal, within the following thirty years they developed ALS. The question is did those toxins reside quietly in their neurons and become active in killing the same neurons in their spinal cord decades later?
It is possible that the natives migrated from the island where they were consuming massive amounts of food with high levels of neurotoxins, the level of exposure to these neurotoxins was only up to 50% of target cell deaths – below the threshold for neurological symptoms. Degenerative neurological diseases do not appear until 80% to 90% of the specific cells in the brain are dead. Once 80% to 90% of the central nervous system (CVS) motor neurons are completely killed, then all the signs of these neurodegenerative diseases will appear. It is obvious, this kind of destruction does not happen overnight. Once these immigrants were in the United States, it is possible that they began to consume American foods and drinks that are high in monosodium glutamate, drink sodas with Aspartame and use NutraSweet® in their coffee. With the addition of these neurotoxins added to their diet, the 50% levels within their brains rose to beyond 80% to 90%. At this stage, many developed ALS, ten to fifteen times the United States rate (Blaylock, 1997).
ALS is a rapidly progressive and inevitably fatal neurological disease that destroys the neurons responsible for controlling voluntary muscles. The word amyotrophic means "without muscle nourishment," referring to the loss of signals the nerves normally send to the muscles. Lateral means "to the side" and refers to the location of the damage in the spinal cord. Sclerosis means "hardened" and refers to the hardened nature of the spinal cord in advanced ALS. It is often called a motor neuron disease, in reference to the cells that are lost in this disorder.

The muscle-controlling nerve cells, or motor neurons, are divided into two types: the upper and the lower. The upper motor neurons are located in the upper part of the brain and exert some control over the lower motor neurons, which are in the brainstem and the spinal cord. With ALS, both the upper motor neurons and the lower motor neurons degenerate or die, ceasing to send any messages to the muscles. As the muscles gradually weaken and waste away, they also begin to twitch. Eventually, the ability of the brain to be able to control or start voluntary movement is lost. Individuals with ALS lose their strength and the ability to move their arms, legs and body. As the nerve cells or neurons break down, so do the muscles in the diaphragm and chest wall, causing the individual to lose the ability to breathe without support from a ventilator.

The lower motor neurons are directly attached to muscles through axons. Bundles (nerves) of these axons leave the spinal cord and extend out to the muscles. The function of lower motor neurons is to send "go" signals to muscles. When these cells gradually die, as in individuals with ALS, muscles become progressively weaker and eventually they become
paralyzed. The lower motor neurons control most of the body are in the spinal cord. Those that control the muscles of speaking, swallowing and facial expression are bulbar motor neurons located in the brainstem.

![Diagram of motor neuron pathways](image)

**Figure 7.** In ALS, upper and lower motor neurons degenerate. Upper motor neurons normally send signals to lower motor neurons, which send signals to muscles. Illustration from MD’s ALS Division Publication.

Conventional medicine does not understand the cause of ALS and scientists do not know why this disease strikes some individuals and not others. ALS does not affect a person's ability to see, smell, taste, hear, or recognize touch, and it does not usually impair a person’s thinking or other cognitive abilities, although recent studies have shown a small percentage of patients experience problems with memory or decision-making. There are also signs that some may even develop a form of dementia (Anitei, 2006).

The cell’s energy supply in the form of ATP is normally produced in the cells via the mitochondria in aerobic cell respiration. This is a metabolic process involving oxygen in the breakdown of glucose. During this breakdown process, free radicals are inadvertently
produced, including two important free radicals, superoxide radical and hydroxyl radical (Eisen, 2000). Of these two, hydroxyl radical is the most potent. The body usually produces these free radicals in minute quantities, however if nothing is done to squelch them, they will accumulate in high concentrations and begin to destroy cells.

The enzyme superoxide dismutase (SOD), a free radical scavenger or antioxidant, catalyzes the dismutation of superoxide into oxygen and hydrogen peroxide. SOD is an extremely important antioxidant defense in nearly all cells exposed to oxygen. Normally, SOD deactivates toxins and free radicals that are occurring within all cells. However, it is believed that in individuals with a rare familial form of ALS, there is a defect of SOD within the motor neurons of the spinal cord. When SOD enzymes are at low levels and are exposed to excitotoxins, massive destruction of the motor cells takes place. Excessive amounts of a particular chemical messenger such as aspartate, a neurotoxin, will damage the neurons. Eventually, damage accumulates due to the inability of cells to repair damage as quickly as it arises.

![Figure 8. Free Radical Damage to Motor Neurons – Illustration originally published in Geriatrics and Aging: Volume 3, Number 9, November 2000, Pages 26, 27.](image)
At John Hopkins University in Baltimore, Dr. Jeffery Rothstein recently found that ALS patients have a deficiency of glutamate transporter proteins. These are specific proteins that transport free glutamate from the fluid around the neurons into surrounding astrocytes, a star-shaped neuralgia cell of nervous tissue. Under normal conditions, free glutamate would be removed immediately. Rothstein's group found deficiencies of a key protein, GLT-1, in the brains and spinal cords of some patients who had died of ALS. GLT-1, or glutamate transporter 1, is a protein whose usual job is to clear away excess glutamate (Rothstein, 1996).

The damage caused by the loss of the glutamate transporter does not happen all at once. Dr. Rothstein found that when he applied a glutamate transport blocker to a spinal cord slice in a culture, glutamate levels rose to high levels that persisted for weeks. The neurons appeared normal within the first few weeks; however, as time progressed, he began to see the motor neurons slowly dying (Rothstein, 2004).
Figure 9. When a neuron is damaged, it can no longer control the muscle, as it should. Illustration from ALS Association.

This data suggest that ALS is a disease wherein the glutamate transporter protein is not present, causing a rise in glutamate and aspartate within the spinal cord. Eventually, high concentrations of glutamate and aspartate destroy the large motor neurons in the spinal cord.
CHAPTER TWO

PARKINSON’S DISEASE

Excitotoxic stimulation due to the ingestion of Aspartame creates powerful insults to the brain, whereas individuals can develop clinical manifestations of Parkinson's disease. The depletion of the neurotransmitter dopamine, resulting from the obliteration of enzyme sites by the flood of these excitotoxins, further complicates this condition.

Parkinson's disease is a complex chronic brain disorder resulting primarily from the progressive death of a specific group of nerve cells in a layer of a region of the substantia nigra. This region is in the midbrain and consists of a layer of large pigmented nerve cells that produce dopamine. Also affected is the basal ganglia, made up of a group of nuclei associated with motor and learning functions in the midbrain.

Figure 10. Basal Ganglia and related structures of the brain. Illustration from About.com – Senior Health

Parkinson's disease is one of a group of motor system disorders which result in the loss of dopamine-producing brain cells. The four primary symptoms of this disease are tremors, rigidity
of the limbs, bradykinesia (extreme slowness in movement), impaired balance and coordination. As the individual’s condition worsens, they may have difficulty walking, talking, or completing other simple tasks. Parkinson’s usually affects people over the age of 50; however, since the approval of Aspartame in cold soft drinks, there has been a remarkable rise in the percentage of individuals who are exhibiting early onset of this disease. In the progression of this disease, the shaking or tremors, which affects the majority of the individuals, may begin to interfere with daily activities. Other symptoms may include depression and other emotional changes, difficulty in swallowing, chewing, speaking, urinary problems or constipation, skin problems and sleep disruptions. There are currently no blood or laboratory tests that have been demonstrated to help in early diagnosis. A neurological examination is all that is available today for individuals seeking help. Another challenge for the individual is finding a doctor that will give them a diagnosis for their symptoms. The disease can be difficult to diagnose accurately and doctors may sometimes request brain scans or laboratory tests in order to rule out other diseases.

There is substantial evidence to show Parkinson’s disease is a disorder whose cause appears to be related to excitotoxicity by Aspartame (Blaylock, 1997; Choi 1992; Kurland, 1988). The combination of aspartate and phenylalanine in Aspartame destroys the cells in the brain pertaining to this disease. These excitotoxins cause these brain cells to generate enormous amounts of free radicals. Aspartic acid is 40% of the Aspartame molecule and can exacerbate these destructive changes in the brains of individual with Parkinson’s disease. The additional toxins created by Aspartame are DKP, aspartate, methanol, formaldehyde and formic acid, all adding to this injury (Bowen, 2000).

Dr. Hyman Jacob Roberts M.D. F.A.C.P., F.C.C.P., director of the Palm Beach Institute for Medical Research has authored 18 texts and has had more than 240 original articles and
letters published, most dealing with diagnostic difficulties, metabolic and neurological problems due to Aspartame poisoning or, as he has coined the term, “Aspartame Disease” (Roberts, 2001). He noted in all of his findings dealing with Parkinson’s disease that there is an alteration of serotonin and dopamine concentrations in the brain by phenylalanine and aspartate. The enzyme, phenylalanine hydroxylase converts phenylalanine to tyrosine and then to dihydroxyphenylalanine (DOPA) and then precursor to dopamine. DOPA can cross the sympathetic neuronal membranes and reach the blood stream. It then becomes a source for the synthesis of tissue catecholamines (dopamine, epinephrine, and norepinephrine), even in the absence of tyrosine hydroxylase, a primary regulator in catecholamine biosynthesis. However, there is a reduction of brain dopamine in the presence of high concentrations of phenylalanine and recent evidence demonstrates oral Aspartame creates formaldehyde as the phenylalanine accumulates within the cells, damaging proteins and destroying the cell’s DNA (Congressional Record-Senate, 1985). In addition, chronic exposure to excess phenylalanine and aspartic acid can decrease the levels of serotonin and other neurotransmitters within several other regions of the brain (Roberts, 1995).

As aspartate overexcites the cortical glutamate cells, it produces parkinsonism a group of nervous disorders similar to Parkinson's disease, marked by muscular rigidity, tremor, and impaired motor control due to the use of certain drugs or frequent exposure to toxic chemicals (Lynch & Guttmann, 2002). The cortical glutamate cells connect to the nigrostriatal neurons lying deep in the brain. Dr. Roberts claims, “It is sort of like lightning hitting the power line outside your house and burning up all of the appliances connected to that line.” The power line represents the cortical glutamate neurons and the appliances, the nigrostriatal system.
Aspartic acid is a recognized source of this damage to the basal ganglia area where Parkinson’s disease degeneration occurs (Bowen, 2002). As in the case with methyl alcohol, the molecular structure of Aspartame makes the aspartic acid damage 5000 times more potent than from free aspartic acid on a milligram per milligram basis. When dopamine, a neurotransmitter necessary to let the brain circuitry function normally, is no longer produced in sufficient amounts in neural tissue, reduction in its concentration within the brain will lead to Parkinson's disease (Bowen, 2000).

Because phenylalanine isolate competes with all other amino acids at the enzyme sites in the brain, it decreases dopamine production making Parkinson symptoms much worse (Bowen, 2000).

The first step in dopamine synthesis in the brain is to have the amino acid tyrosine decarboxylated to tyramine, usually replacing a carboxyl group with hydrogen. Phenylalanine isolate also competitively inhibits the active site on the decarboxylase enzyme (Bowen, 2005). When the tyrosine is not decarboxylated to tyramine the dopamine levels in the brain plummet considerably. Chronic exposure to excess phenylalanine can decrease the levels of serotonin and other neurotransmitters within several regions of the brain (Wurtman, 1987).

The methyl alcohol derived from Aspartame also plays a role in Parkinson’s disease. It has been reported that methyl alcohol appears to cause Parkinson’s through postsynaptic dysfunction by interfering with dopamine reuptake at nerve terminals (Indakoetxea, Lopez de Munain, Marti-Masso, & Linazasoro, 1990). Insufficient levels of dopamine from the neurons of the substantia nigra synapsing on neurons in the striatum are believed to be responsible for the primary symptoms of Parkinson's. Individuals with Parkinson’s disease use L-Dopa to try to
increase the dopamine levels; however, the use of Aspartame can completely defeat this therapeutic endeavor (Gold, 2002).
CHAPTER THREE

ALZHEIMER’S DISEASE

While conventional medicine has dedicated substantial efforts to study the cause and cure of Alzheimer’s disease and on the formation of amyloid plaques and neurofibrillary tangles that are thought to contribute to the degradation of the neurons in the brain and the symptoms of Alzheimer's disease, Aspartame is not on the list to investigate. In an individual with Alzheimer's disease, there is an accumulation of amyloid plaques between neurons in the brain. Amyloid is a general term for protein fragments that the body produces normally. Beta-amyloid found in senile plaques (or neuritic plaques) is a fragment of a protein that is snipped from another protein called amyloid precursor protein (APP). In a healthy brain, these protein fragments are broken down and eliminated. In Alzheimer's disease, the fragments accumulate to form hard, insoluble plaques (Blaylock, 1997).

Perhaps the accumulation of this abnormal protein is just a result of this disease rather than the cause. Something else is injuring the neurons causing them to acquire amyloid protein (Blaylock, 1997).
With age, the brain normally shrinks due to the death of brain cells. There is also loss of the myelin (fatty insulation) surrounding the fiber pathway within the white matter of the brain. However, the brain does not lose neurological function just because humans age. The brain is capable of functioning neurologically until we die (Duara, 1984). What scientists do know and see in an aging brain is the presence and progressive accumulation of an “age pigment” called lipofuscin. This is a yellow-brown pigment known to collect in neurons of the elderly. However, the accumulation of lipofuscin does not appear to affect mental function.

The cause of Alzheimer’s can be attributed to many events in an individual’s life, including a severe blow to the head, high fevers, or a chronic subdural hematoma (a large collection of blood in the brain). However, most cases of Alzheimer’s present as a long, gradual, and silent loss of neurons over many years, rather than a massive loss over a short period of time.

The hippocampus of the temporal lobe is the area of the brain that is responsible for new memories, and in Alzheimer’s patients, it is the area that shows the most extensive damage. Researchers are not sure whether the mechanism that retrieves new memories breaks down or
rather the inability to store the new memories as they are created is the problem. Eventually, long-term memories will begin to fade along with whatever new memories are made.

Neuritic or senile plaques and neurofibrillary tangles are microscopic bodies found throughout the brains of patients with dementia, chronic dementia, and Alzheimer’s disease. However, these two microscopic injuries or inclusions have also been found in the brains of normal elderly persons. There is growing evidence that these lesions are not the cause of Alzheimer’s disease, but rather the corollary of it (Blaylock, 1997). These lesions develop because of the death of neurons, but do not cause the neurons to actually die (Trojanowski, 1999).

The neurons where these neurofibrillary tangles are located are neurons with glutamate receptors. The frontal and parietal cortex and the hippocampus of the temporal lobes are heavily concentrated with these neurofibrillary (age) tangles and plaques. These are the areas where most of the damage and neuron deaths occur. Viewing these neurofibrillary tangles under the electron microscope, scientists see a mass of twisted fibrils found within the dying neuron. They appear as clusters of paired strands twisted upon each other. These are called, paired helical filaments. Glutamate and aspartate are found in the highest concentration of any other amino acids within these neurofibrillary tangles (Blaylock, 2006). The number of neurofibrillary tangles determines the degree of dementia.

In 1985, two scientists, Umberto De Boni and D.R.C. McLachlan, were working with spinal cord cells that were exposed to excitotoxins in tissue culture (DeBoni & Crapper-McLachlan, 1978), and discovered that prolonged exposure to glutamate or aspartate resulted in the formation of paired helical filaments identical to those seen in the brains with Alzheimer’s disease (DeBoni & McLachlan, 1985).
Senile or neuritic plaque is a darkly staining buildup of abnormal brain cell fragments located outside the neuron and in the same areas as the neurofibrillary tangles. What the two scientists found in the core of these plaques was the protein beta-amyloid not usually found in the brain. Although neuroscientists believe that Alzheimer’s disease is a disorder resulting from the accumulation of this abnormal protein in the brain cells, they can not explain why it only accumulates in certain neurons that contain glutamate receptors and not others.

One indicator of Alzheimer's disease is the accumulation of amyloid plaques between nerve cells in the brain. Because the beta-amyloid stimulates an abnormal flow of calcium into the interior of the neuron, it makes a glutamate-sensitive neuron even more sensitive to excitotoxins (aspartate and glutamate), which allows the neuron to be excited to death. Therefore, these findings show that high concentrations of glutamate and aspartate in neuritic plaque are causing these neurons to die (Blaylock, 1997).

There are specific types of proteins found in high concentrations in the brains of individuals with Alzheimer’s disease. The immunological staining test that is done to determine the degree of concentration of these proteins is ALZ-50. Exposing cultures of normal neurons from the hippocampus to high concentrations of glutamate below that which can kill neurons showed a marked increased in the modification of the color of these neurons. Apparently, concentrations of glutamate and aspartate can also increase the immunoreactive staining, which lead researchers to believe that lower concentrations of food excitotoxins, such as Aspartame, can result in the same specific immunoreactive stain changes seen in Alzheimer’s disease (Blaylock, 1997; Mattson, 1990).

Wanting to retest the results, Umberto De Boni and D.R.C. McLachlan then added glutamate-blocking drugs to the culture medium and the results showed the effects caused by the
glutamate and aspartate were completely blocked, suggesting that the excitotoxin was causing the destruction. These researchers showed that by exposing neurons to glutamate or aspartate they could induce paired helical filaments almost identical to those seen in individuals suffering with naturally occurring Alzheimer’s disease. The destruction was dose-dependent, showing that the higher the level of excitotoxins, the greater the damage (Sindou, 1992). Researchers also noted that the beta-amyloid acted through all three subtypes of glutamate receptors, NMDA, quisqualate, and kainite (Blaylock, 1997).

These findings proved that glutamate and aspartate can induce the same immunoreactive proteins found in Alzheimer’s disease, and once the neurons begin to destruct, they create abnormal proteins in the form of beta-amyloid. The effect is dose-dependent; the amount of excitotoxins determines the level of beta-amyloid protein present. This in turn, can further enhance the toxicity of glutamate and aspartate. The more plaques that are formed, such as beta-amyloid, the more sensitive surviving neurons are to the excitotoxins.

What this all means is that if an individual unknowingly has the beginnings of Alzheimer’s disease and consumes Aspartame found in diet soda, sugar-free candies, artificial sweetener, medication, supplementation, or any other products, they have subjected themselves to further exposure of excitotoxins that will accelerate the process that leads to full-blown Alzheimer’s disease. Below, shown in Table 1, you see the progress of 129 Aspartame consumers with what was diagnosed as Aspartame-associated memory loss.
Table 1

FDA Data on 129 consumers with Aspartame-associated memory loss

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<tr>
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Note. Dr. Linda Tollefson (FDA's Assistant Commissioner for Science) provided information on 129 consumers who complained to the FDA about memory loss while using Aspartame products. Chart Illustration from Aspartame Disease: An Ignored Epidemic -- by Dr. HJ Roberts, MD, FACP, FCCP.

One of the most important advancements in medical technology has been the positron emission tomography (PET). It is the fastest-growing and accepted nuclear medicine tool in medicine today and is noninvasive. Its scanning technique utilizes small amounts of radioactive positrons (positively charged particles) to visualize body function and metabolism. Physicians first used PET to obtain information about brain function, and to study brain activity in various neurological diseases and disorders including stroke, epilepsy, Alzheimer's disease, Parkinson's disease, and Huntington's disease. Doctors are able to evaluate patients for cancers of the head
and neck, lymph system, skin, lungs, colon, breast, and esophagus. PET can also evaluate heart muscle function in patients with coronary artery disease or cardiomyopathy.

Because of PET, scientists can actually watch the brain function and metabolize glucose for energy. With all this new technology, doctors have confirmed their beliefs that Alzheimer’s disease does not happen overnight as a result of sudden loss of massive amounts of neurons, but rather as a creeping death of the brain over decades (Blaylock, 1997). PET has allowed the scientists to rule out certain preconceptions of how Alzheimer’s disease is caused. It was once believed that infectious malformed proteins, prions, could infect the brain and cause a slow die-off of cells that would lead to Alzheimer’s disease (Whitehouse, 1986). However, there seems to be a problem with this hypothesis. Prions tend to affect widespread areas of the brain rather than selected sites and they found symptoms not related to Alzheimer’s disease. The theory of abiotrophy is not convincing because only very specific brain cells are seen dying and other cells adjacent to these dying cells are completely healthy. Something is targeting the neurons, and that something can distinguish these neurons from others. Moreover, that something may be an environmental neurotoxin causing these neurons to die.

Excitotoxins, such as aspartate and glutamate, excite the brain rather than calm it down (Blaylock, 1997; 2005). They stimulate the formation of free radicals within exposed neurons that, in turn, can trigger the release of even higher levels of glutamate within the brain. This results in even greater damage and shows us that once this cascade of damage begins, it becomes self-generating (Breitner, 1994). It is important to keep in mind that most studies that are done to understand how excitotoxins (glutamate and aspartate) affect neurons in the brain and their toxic effects are executed by using very high doses. The reason for this is that technology today to
detect low level damage requires special equipment and researchers may not have the specialized
tools to observe the effect of prolonged exposure to lower doses. Nevertheless, they do know
that neurons exposed to low dosages of excitotoxins in a dose-dependent manner in rat
hippocampal and septal cell cultures produced identical changes in the dendrites of neurons seen
in an individual with Alzheimer’s disease (Mattson, 1992). Unlike most animal studies, where
 glutamate and aspartate are used over short terms to produce results of excitotoxin damage,
human exposure may persist over decades, even a lifetime (Mattson, Cheng, Davis, Bryant,
Lieberburg, & Rydel, 1992). People consume foods containing glutamate and aspartate without
any knowledge of its ability to kill.

Glutamate and aspartate are commonly used as metabolic fuels and as neurotransmitters,
however, when the concentration of these excitotoxins elevates to a crisis level and are not
carefully regulated, toxic amounts will build up and destroy specific neurons. To prevent
 glutamate accumulation from happening, the brain has a pump system that pumps excess
 glutamate found in the extracellular fluid around the neurons into the surrounding glial cells. As
it enters the glial cells, there are special enzymes that deactivate the glutamate from producing
any further damage. When this protective system fails due to the lack of ATP, the glutamate
begins to accumulate and stimulate the receptors on the surface of the cell membrane, which
allows calcium to pour into the cell damaging the mitochondria (Blaylock, 1997).

When the brain begins to deplete its ATP the body experiences hypoglycemia or low
blood sugar. The brain uses more glucose in twenty minutes of deep concentration than the body
does in one hour of physical activity and consumes over 25% of all the glucose used in the body.
Under normal conditions, the brain absorbs twice as much glucose as it uses. This offers a large
measure of protection under conditions where the brain’s energy needs are greatly increased, such as with seizures and during the early stages of brain injury or when excitotoxins deplete the brain of ATP. Unfortunately, it cannot store this energy and when the brain becomes hypoglycemic, it begins to fail rapidly. When there is a lack of sugar to the brain, then designated parts of the brain begin to die. These same areas of the brain are also destroyed when large dosages of excitotoxins are introduced.

![Image](image_url)

**Figure 12. Areas of the brain affected by Alzheimer’s and other dementias. These areas of the brain are most sensitive to excitotoxins damage. Illustration from Nucleus Medical Art.**

The brain damage caused by severe hypoglycemia in the presence of Aspartame is a result of the failure of these protective mechanisms and not from a lack of fuel to the brain cells themselves (Lindvall, 1988). Scientists have also noted the same damage to the brain if oxygen is removed. It appears that what ever the cause of the energy failure, the result is identical. Glutamate accumulates in the brain in high levels and destroys glutamate–sensitive neurons (Benveniste, 1984). However, the reversal of this situation can actually protect the brain and herein lies a huge misunderstanding.

Aspartame and MSG are flavor enhancements that are often used to prepare foods that have few or close to zero amounts of carbohydrates. If an individual is consuming Aspartame
and/or glutamate-laden foods, and other foods containing carbohydrates, the fuel from the carbohydrates will protect the neurons from hypoglycemic destruction. However, most individuals using Aspartame as an artificial sweetener are primarily concerned about not consuming carbohydrates. A diet soda or NutraSweet® /Equal® packet used in sweetening coffee or other consumable products are usually void of carbohydrates or have very low levels to assist with feeding the brain the necessary fuel it needs to function. Therefore, there is no protection to the neurons to help them with the needed energy to stay afloat as they begin to swell up with calcium and begin to burst.

While glucose can reduce the amount of neuron damage caused by high doses of glutamate and aspartate, the protection is not complete. Cellular damage still occurs, but to a much lesser degree. Chronic exposure to high levels of glutamate or aspartate will cause less brain injury when an individual is consuming adequate levels of carbohydrates; nevertheless, accumulative damage over many years may be substantial.

Figure 13. When there is enough energy or ATP within the neuron, they are resistant to excitotoxins. However, when the energy or ATP runs out, the neurons become vulnerable at even low dosages. Illustration by Dr. Russell L. Blaylock, M.D.
There are many deleterious consequences to a decreased ATP production including increased free radical production and oxidative stress. Cytochrome oxidase is an oxidizing enzyme found in the mitochondria (Parker, 1990). It is extremely important in cellular respiration as an agent of electron transfer from certain cytochrome molecules to oxygen molecules. Individuals with Alzheimer’s disease have neurons that are defective in the delivery of glucose to the brain. Recently, it has been demonstrated that there is a 50% reduction of cytochrome oxidase in platelets of patients with Alzheimer's disease. The deficiency of this key energy-metabolizing enzyme could reduce energy stores and could contribute to brain dysfunction and neurodegenerative processes associated with Alzheimer’s disease (Mutisya, Bowling & Beal, 1994). In Parkinson’s disease, an enzyme that is predominately deficient in the mitochondria is Complex I (Schapira, Cooper, Dexter, Clark, Jennery & Marshen, 1990). There are a number of studies that show a decrease in complex I activity in peripheral tissues affected by Parkinson’s disease (Schapira, et al. 1990). Mitochondrial ATP production is a foundation for health and it is necessary for physical strength, stamina and consciousness. Because the mitochondria produce most of the currency used by the body, cells with a high metabolic rate, such as heart muscle cells, may contain many thousands of mitochondria while other cells may contain only dozens. Even subtle deficits in mitochondrial function can cause weakness, fatigue, and cognitive difficulties. Excitotoxins that create neurotoxicity can strongly interfere with mitochondrial function and are potent poisons (Cooper & Schapira, 1997).

The pyruvate decarboxylation reaction links glycolysis and the citric acid cycle. This reaction is the conversion of pyruvate into acetyl CoA. The pyruvate decarboxylation reaction is catalyzed by the pyruvate dehydrogenase complex. In 1985, Dr. Kwan-Fu Rex Sheu and his
coworkers produced evidence that the pyruvate dehydrogenase complex is inhibited in calcium-loaded cerebrocortical mitochondria. Impairment of mitochondrial function happens because of calcium-loading and is one of the significant events that leads to neuronal death after an ischemic insult. Pyruvate dehydrogenase is an important metabolic enzyme found to be deficient with Alzheimer’s disease. Once again, without ATP to bail the calcium out of the swelling neurons due to aspartate and other excitotoxins holding the calcium channel open, it is inevitable that the neuron will be excited, expand and explode (Lai, DiLorenzo, Sheu & Rex, 1988).
CHAPTER FOUR

MULTIPLE SCLEROSIS

The question begs an answer as to why only certain neurons suffer because of these enzyme deficiencies and why others are spared? The hippocampus contains one of the highest concentrations of glutamate receptors in the entire brain and is the most severely damaged in Alzheimer’s disease, whereas there are fewer glutamate sensitive neurons in other areas of the brain. However, it is the overstimulation by glutamate, aspartate, and other excitotoxins that cause the energy deficient neurons to be damaged in the first place.

Aspartame is made up of 10% methanol or wood alcohol. This deadly poison is gradually released in the small intestine when the methyl group of Aspartame encounters the enzyme chymotrypsin, a pancreatic digestive enzyme that catalyzes the hydrolysis of certain proteins in the small intestine into polypeptides and singular free form amino acids. Free methanol is created from Aspartame when it is heated above 86 degrees Fahrenheit or 30 degrees Celsius and its absorption is expedited when ingested. This will occur within a human body that has a temperature above 86 degrees Fahrenheit, or when products containing Aspartame are not refrigerated properly or used in cooking.

Once methanol has been absorbed, it breaks down into formic acid. In nature, formic acid is found in the stings and bites of many insects, including bees and ants. Formic acid is also a precursor of formaldehyde, which is a neurotoxin (DHHS, 1993; Liesivuori, 1991). The U.S. Environmental Protection Agency (EPA) assessment of methanol states that exposure of humans to methanol by inhalation or ingestion may result in visual disturbances, such as blurred or
dimness of vision, leading to blindness. Neurological damage, specifically permanent motor
dysfunction, may also result. It is considered a cumulative poison due to the low rate of excretion
once it is absorbed (U.S. Environmental Protection Agency, 2000). Both formic acid and
formaldehyde are neurotoxic metabolites (Osterloh & Holmes, 1995).

Low-level exposure to methanol has been shown to cause headaches, dizziness, nausea,
tinnitus, gastrointestinal disturbances, weakness, vertigo, chills, memory lapses, numbness,
shooting pains, behavioral disturbances, neuritis, misty vision, vision tunneling, blurring of
vision, conjunctivitis, insomnia, vision loss, depression, heart problems, diseases of the heart
muscle, and pancreatic inflammation. It is important to understand that extremely low-level
methanol and formaldehyde exposure mimics multiple sclerosis (MS). Dr. Woodrow C. Monte
wrote: "Methanol, one of the breakdown products of Aspartame, has no therapeutic properties
and is considered only as a toxicant. The ingestion of two teaspoons is considered lethal in
humans" (Monte, 1984, p. 44).

The chronic intake of free methanol in significant amounts account for some of the
symptoms associated with neurodegenerative diseases. In 1975, Dr. Herbert S. Posner, who is
associated with the National Institute of Environmental Health Sciences, wrote a review entitled,
“Biohazards of Methanol in proposed New Uses” six years before the FDA approved Aspartame.
He stressed the failure to recognize the delayed and irreversible effects on the nervous system of
methanol at widely varying levels of exposure and at rather low levels. (Posner, 1975; Roberts,
2000). The intake of methyl alcohol from natural sources averages less than 10 mg daily;
Aspartame beverages, however, contain about 55 mg methanol per liter and nearly twice as much
in some carbonated orange sodas in order to preserve the taste. The Methyl ester imparts
sweetness to Aspartame (Monte, 1984). Individuals who drink five liters a day therefore can be ingesting over 400 mg of methanol.

Table 2

One (1) 12oz. can of diet soda contains close to 200 mg of Aspartame (Murray, 2005).

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<thead>
<tr>
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<tr>
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<tr>
<td>Aspartic Acid</td>
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<tr>
<td>Methanol</td>
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The insulating envelope of myelin that surrounds the core of a nerve fiber or axon and facilitates the transmission of nerve impulses is called the myelin sheath. The loss of the myelin sheath on the nerve fibers characteristic of MS is due to the death of the oligodendroglia cells at the site of the lesions or plaques. Oligodendroglia cells are part of the supportive tissue of the nervous system or neuroglia, consisting of cells similar to but smaller than astrocytes, found in the central nervous system and associated with the formation of myelin.

Figure 14. Areas of damaged myelin are known as plaques. These plaques, or sites of damage, can cause MS symptoms.
Studies have shown that excessive exposure to excitotoxins, like Aspartame, at the site of the lesions can result in the death of these important cells (Blaylock, 2004). These excitotoxins are secreted from microglia immune cells in the central nervous system. This process not only destroys the myelin-producing cells it also breaks down the blood-brain barrier (BBB), allowing excitotoxins in the blood stream to enter the site of damage. The BBB is semi-permeable to allow some materials to cross its wall of capillaries but prevents others. In most parts of the body, the smallest blood vessels and capillaries, are lined with endothelial cells. Endothelial tissue has small spaces between each individual cell so substances can move readily between the inside and the outside of the vessel. However, in the brain, the endothelial cells fit tightly together and substances cannot pass into the bloodstream. With the BBB damaged, as in MS, these excitotoxins can freely enter the site of injury, greatly magnifying the damage (Blaylock, 2004).

A condition called benign MS is diagnosed after a person experiences one or two symptoms with complete recovery. This form of MS does not worsen with time and there is no permanent disability. Benign MS can only be identified when there is minimal disability ten to fifteen years after onset. Benign MS tends to be associated with less severe symptoms at onset. However, continued consumption of Aspartame can convert this benign condition into full-blown, clinical MS (Blaylock, 2004). Methanol is an axon poison and when combined with the toxicity of the aspartate, adds up to considerable brain toxicity. Once MS becomes full-blown, further consumption of excitotoxins can magnify the toxicity, increase the symptoms, and may lead eventually to death (Martini, 2007).
Glia cells or astrocytes form a layer around brain blood vessels and are important in the development of the BBB. Astrocytes, a sub-type of the glia cells in the brain, regulate the internal environment of the brain, especially the fluid surrounding neurons and their synapses, and provide nutrition to nerve cells. Glia have important developmental roles such as producing molecules that modify the growth of axons and dendrites. They are also active participants in the hippocampus and cerebellum in synaptic transmission. They regulate clearance of neurotransmitters from the synaptic cleft and release ATP which modulates presynaptic function (Evangelista & Bowen, 2002). The BBB has several important functions, among them are protecting the brain from foreign substances in the blood that may injure the brain, protecting the brain from hormones and neurotransmitters that exist in the rest of the body, and maintaining a constant environment for the brain.

Several areas of the BBB are weak, allowing substances to cross into the circumventricular organs in the brain somewhat freely. The brain is then able to monitor the composition of the blood as it passes through the pineal, neurohypophysis, area postrema, subfornical organ and the median eminence (Smith, 2000). In general, amino acids are carefully regulated because some also serve as neurotransmitters or transmitter precursors; without strict control of these substances, our brains would be transmitting extraneous impulses.

Chronic elevations of blood excitotoxins can seep through the normal BBB when high concentrations are maintained over a long period of time. This, naturally, would occur when individuals daily consume foods containing excitotoxins such as Aspartame. In nature, aspartate levels are not normally elevated on a daily basis. Sustained elevations of these excitotoxins are attributed to the modern diet in humans who also concentrate MSG in their blood five times
higher than mice from a comparable dose, and maintain the higher blood level longer than mice (Blaylock, 1997). In fact, humans concentrate MSG in their blood to a greater degree than any other known animal, including monkeys. And children are four times more sensitive to a given MSG dose than adults (South, 2003).

Scientists have used excitotoxins to make lesions in the brains of experimental animals because they have the ability to destroy neurons while leaving the fibers of passage alone (Winn, 1990). Recently, findings show that excitotoxins act at different sites within the central nervous system (CNS) not only destroying the neurons, but also stripping the myelin from fibers, and compromising the integrity of the BBB. Excitotoxic lesions of the lateral hypothalamus have shown to produce local demyelination, which is characteristic of MS (Brace, Latimer & Winn, 1997).
CHAPTER FIVE
SIEZURES

In 1987, H.A. Tilson, a neurotoxicologist at the Laboratory of Behavioral and Neurological Toxicology within the National Institute of Environmental Health Sciences in North Carolina, found phenylalanine, a major constituent of Aspartame, a possible cause for an amino acid imbalance resulting in behavioral alterations. He noted that shortly after approval for usage in humans, several reports of Aspartame-associated neurological effects were reported, including headaches, dizziness, and mood alterations (Massachusetts Medical Society, 1984). In the same year, Dr. Wurtman and Dr. Maher noted that the disproportionate elevation of brain phenylalanine concentrations after Aspartame or phenylalanine used in animal studies diminished the dopamine released from the brain (Wurtman & Maher, 1985). They attempted to compare the doses of Aspartame producing neurochemical effects in rodents and humans. Their data shows that 15-20 mg/kg administered to rodents diminishes catecholamine release, which are associated with lower seizure thresholds in genetically epilepsy-prone rats and suggested that exposure to large amounts of Aspartame may ultimately decrease brain levels of norepinephrine and adversely affect some people predisposed to seizures (Tilson, 1985).

Seizures are caused by abnormal electrical discharges from brain cells, often in the cerebral cortex. Their occurrences are not considered a distinct disease. Normally, nerve transmission in the brain occurs in an orderly way, allowing a smooth flow of electrical activity. A seizure occurs when neurons generate uncoordinated electrical discharges that spread throughout the brain. This can occur with both normal and abnormal nerve cells. However, in some cases, overactivity of excitatory neurotransmitters or underactivity of inhibitory
neurotransmitters may lead to seizure activity by allowing an uncoordinated flow of electrical activity in the brain (Neurological Channel, 2007).

A seizure reflects the results of too much excitation, too little inhibition or neurons that are too sensitive to the neurotransmitters. During a seizure, certain cells begin to fire repeatedly and spread this behavior to other cells. A normal brain responds with enough inhibitory neurotransmitters to stop the spread. However, if a group of neurons "runs away," firing repeatedly, and the brain cannot inhibit them, a seizure results. Seizures are often self-limiting as the renegade neurons exhaust themselves and eventually stop firing. The period after the seizure may reflect lower than normal activity among the neurons. How and why these "renegade" neurons cause a seizures is still a matter of investigation.

Interestingly, certain areas of the brain are more likely than others to be the source of a seizure. These areas include the motor cortex, (responsible for the initiation of body movement) and the temporal lobes, which includes the hippocampus and involves memory. The reason for this probability may be that nerve cells in these areas are particularly sensitive to certain situations that can provoke abnormal electrical transmission, such as changes in brain biochemistry and communication between brain cells. These basic functions of the neurons that become altered and abnormal can produce seizures or prolonged seizures that will cause injury to the brain. Seizures that last longer than 20 to 30 minutes can permanently damage the brain’s neurons.

Recently, scientists have discovered a link between inflammation of the neurons and various disease conditions that man has been facing (Wyss-Coray & Mucke, 2002). Aspartame is an excitotoxin that creates an enormous inflammation in its repetitive action. Dr. Soffritti and his
research team at The European Foundation of Oncology and Environmental Sciences "B. Ramazzini" in Bologna, Italy stated clearly in their recent study that, "inflammation was observed in both animals who were treated with Aspartame as well as in the control group" (Soffritti, 2005, p. 16). What was recently discovered was how radiation creates inflammation in the brain, which leads to Alzheimer’s. Most children who have radiation therapy to eradicate brain tumors may come through the treatment with the tumor destroyed, but will go on to develop learning and memory problems similar to those seen in Alzheimer's patients. The decline of brain cells begins many months or years after the treatment and is currently irreversible. Scientists are starting to understand why. Theo D. Palmer of the Department of Neurosurgery at Stanford University stated, “It's very sad because these children survive the cancer but later on develop cognitive problems and often end up in special education or are institutionalized” (Winstead, 2003, on-line article).

In an effort to understand why radiation to the brain causes cognitive decline, Dr. Palmer and two colleagues conducted experiments in rats and learned that inflammation caused by radiation blocks the production of new neurons in the hippocampus, which usually generates thousands of neurons every day. This region is critical for learning and creating new memories. Individuals who continue to consume Aspartame are creating the same kind of inflammation to their neurons in the hippocampus and possibly destroying their chances in the future of maintaining memory function. They found that rats exposed to radiation stopped producing neurons (Winstead, 2003). Part of the reason they stopped was inflammation, and Swedish researchers working independently made the same discovery concurrently.
“Both studies found that new neurons are very sensitive to inflammation,” says Olle Lindvall, of Lund University Hospital in Sweden, who expressed surprise at how detrimental inflammation is to the production of new neurons. The studies suggest a link between inflammation and the suppression of new neurons that may contribute to cognitive decline in people with brain diseases (Winstead, 2003). If the neurons are being constantly excited by excitotoxins, then they will become inflamed and new neurons will not be generated.

Another study done on type I diabetes mellitus (TIDM) shows us that it is not an autoimmune disease, but one of a neuron inflammation to the islet cell (Dosch, 2006). Individuals who are constantly worrying about their weight will fall victim to the high pressured ads on television, internet, or magazines today about Aspartame being safe and sound for them, especially if they have diabetes.

In Canada, Scientists cured twenty-one mice with diabetes over night. It appears the nervous system and inflammation play an enormous key role in diabetes. When diabetic mice were injected with a substance to counteract the effect of malfunctioning pain neurons in the pancreas to restore proper function they became healthy virtually overnight (Dosch, 2006).

When inflammation occurs it contributes to the eventual death of insulin-producing islet cells in the pancreas. Dr. Hans Michael Dosch, an immunologist at the hospital and a leader of the studies, had concluded in a 2000 paper that there were surprising similarities between diabetes and multiple sclerosis (Dosch & Becker, 2000). He noted that there was an "enormous" number of nerves around the beta-cells and pain neurons primarily used to signal the brain that tissue has been damaged. It turns out the nerves secrete neuropeptides that are instrumental in the proper functioning of the islets. Further study by the team, which also involved the University of
Calgary and the Jackson Laboratory in Maine, found the nerves in diabetic mice were releasing too little of the neuropeptides, resulting in a "vicious cycle" of stress on the islets (Dosch, 2006).

Dr. Russell Blaylock wrote, "With the public concern over childhood obesity and diabetes, few are being told of the overwhelming evidence that early exposure to excitotoxins as found in Aspartame consistently produce gross obesity and insulin resistant diabetes, just as we are seeing in our youth" (Martini, 2005). All these connections are showing us that the axon of the neuron reaches out to many other areas of the body to mimic and create diseases that are associated with these neurons being destroyed by neurotoxins that are exciting neurons to death.

![Diagram of a neuron with labels for dendrites, nucleus, cell body, axon, synaptic knob, and receptor site.](image)

**Figure 15.** Axons are in effect the primary transmission lines of the nervous system and as bundles they help make up nerves. The longest axons in the human body, for example, are those of the sciatic nerve, which run from the base of the spine to the big toe of each foot. *Illustration from Bipolar Disorders: A Guide to Helping Children by Mitzi Waltz.*

Excitotoxins lead to neurological inflammation. Glutamate and aspartate are the most abundant neurotransmitters in the CNS (Yasko, 2003). They are also the most toxic, and because of this, they must be highly regulated.
When Protein kinase C activates phospholipase A2 (PLA2) within the neuron membrane, it brings about the release of arachidonic acid into the cytoplasm. PLA2 is a principal phospholipase that cleaves off fatty acids, primarily arachidonic acid. After arachidonic acid is acted on by lipoxygenase and COX, which produce a series of potentially destructive eicosanoids, it is the cyclooxygenase 2 (COX II) enzyme that brings about the accumulation of prostaglandin E$_2$ (PGE2) and Prostaglandin D$_2$ (PGD2). Both PGE2 and PGD2 are pro-inflammatory molecules (Nairn, 1985). Interestingly, only glutamatergic or glutamate receptor-related signal neurons contain COX II enzymes, which are located on distal dendrites and are concentrated in dendritic spines (Blaylock, 1997).

It is the accumulation of inflammatory eicosanoids that leads to the production of free radicals, including the destructive hydroxyl radical. As free radical production accelerates they interact with the neuron’s numerous membrane structures, which including the nuclear membrane, inner and outer mitochondrial membranes and plasma membranes (Gough, Kyriakides & Hechtman, 2006). Once this cascade of destruction begins, a chain reaction within

\textbf{Figure 16. Dendrite spines are any of various outgrowths of certain nerve-cell dendrites, ranging in shape from small knobs to thornlike processes that are preferential sites of synaptic axodendritic contact.}
the membrane’s polyunsaturated fatty acids is initiated. This process is called lipid peroxidation (LPO). There is a close relationship between excitotoxicity and free radical generation. Free radicals speed up the release of glutamate in the brain, and excitotoxins trigger the production of large number of free radicals creating positive-feedback (Blaylock, 1997).
CHAPTER SIX

HUNTINGTON’S CHOREA

Huntington’s Chorea is essentially a disease of the nervous system. The English word "chorea" itself comes from the Greek word choreia, which means "dance." The name chorea is given to this disease because those affected by it experience jerking movements of the face, arms, and eventually, the entire body. This is then followed by a relentless deterioration of the neurological function. Patients eventually wind up bedridden.

Huntington’s Chorea’s most marked and characteristic feature is a violent spasm affecting the voluntary muscles. There is no sense of loss or of consciousness while these contractions are acting out, as there is in epilepsy. The upper extremities are usually the first affected. All the voluntary muscles are likely to be affected, those of the face rarely being exempted. The patient will experience a great deal of difficulty in performing the simplest of tasks, such as sticking out his/hers tongue out or trying to walk or eat. The body is in constant perpetual motion.

While autopsying Huntington’s Chorea patients, an examination of a cross-section of their brains showed characteristic changes in the internal structure. The characteristic changes were found in each hemisphere of the brain, and in the middle of the four basal ganglia. This area is partly responsible for body movement and coordination and known as the caudate nucleus (so named because it looks like a tail). It was originally thought to primarily be involved with control of voluntary movement; however, it is now known to be an important part of the brain's learning and memory system. The paired nuclei lying adjacent to the frontal horn of the ventricles were badly shrunken from degeneration. These nuclei are called the basal ganglion or
striatum. The striatum includes three structures: globus pallidus, putamen, and caudate nucleus. It is part of the brain involved with regulating the intensity of coordinated muscle activity such as movement, balance, and walking. There is a clear connection between nerve damage and the presence of excitotoxic amino acids when found within these regions (Blaylock, 1997).

The striatum plays a vital role in individual movements, such as swinging the arms, and complex patterns of arms and leg movements that are automatic. These movements are already preprogrammed within the striatum so that you do not have to figure out how to perform them each time they are needed. Examination of the striatum of a typical Huntington’s patient under the microscope shows that not all of the neurons in the striatum are destroyed. Rather, the most severe loss is to the small and intermediate sized neurons, with almost complete sparing of the larger neurons. Some neurons are lost in the frontal cortex as well, and the damage is very specific to some neurons while sparing others that are close by.

Scientists have been unable to produce an exact model of Huntington’s disease, however, they have come very close by using only one class of chemical to produce the selective damage that is seen in these brains. These chemicals are excitotoxins (Mclin, Thompason & Steward, 2006).

Scientists have shown that neurotoxins produce neurodegenerative diseases and that there is a recurring pattern seen with excitotoxin accumulation (National Institute of Neurological Disorders and Stroke, 2005). They see the same characteristics of energy deficiency in the brain, the mechanisms in place to protect the neurons are impaired, low magnesium, and high calcium accumulation due to the disruption of regulatory control in the neurons involved (Siesjo, Bengtsson, Grampp & Theander, 1989).
Figure 17. In photo A, the cross-section shows a normal brain. Photo B shows the typical findings in a case of Huntington’s disease with shrunken caudate nucleus and the adjacent enlarged ventricles.

It is important to recognize the difference between natural dietary amino acids, and pharmaceutically produced amino acid isolates. Dietary amino acids are absorbed from the gut by an organized digestive process involving acid and alkaline digestive juices that the body uses to break down the long poly-peptide chains of amino acids from food stuff and then absorb them as a singular free form amino acid. This allows a slower release into the blood stream and they are always accompanied in a balanced mixture of other amino acids which are in the proper enzyme-regulated proportion for use by the body. Since they are in competition with one another for the enzyme sites, the body ensures that no one amino acid dominates the others.

However, amino acid isolates that have been artificially separated from the rest of the amino acid chain and reconfigured, as in the case of Aspartame, are now part of a man-made compound without the proper enzyme-regulated proportion for use by the body. Aspartame is then added to foods during the manufacturing process. These amino acids are isolated, as single
or dipeptide molecules. This is very different from the 80 to 300 amino acid chains that form natural proteins from dietary sources. The isolates of Aspartame are then incorporated into a compound containing free methanol, aspartate and phenylalanine, which easily break down into formaldehyde, formic acid, and DKP inside the human body (Barua & Bal, 1995).

One can of diet soda pop yields about as much phenylalanine as a large bowl of beans. However, beans are natural to the body and not pharmaceutically produced (Bowen & Evangelista, 2002).
CONCLUSION

Yes, it is true that Aspartame is the most tested product in the world; however, it is not true that it is safe. Does "most tested" imply safe for human consumption? More importantly, what were the results of these studies and how was Aspartame approved? An in-depth look at the history of Aspartame approval reveals a trail of suspicious methods and possible collusion between the FDA and the G. D. Searle Company. Upon closer examination, the available research revealed that the manufacturers, G.D. Searle and Monsanto, along with the FDA are manipulating the public into believing that Aspartame is safe and allowing it to be used in everything from children’s vitamins to vaccines.

Aspartame is a neurotoxin. It is also known as NutraSweet, Equal, Spoonful, and Equal-Measure. It is made up of aspartic acid, phenylalanine, and methanol. Since it was discovered by accident in 1965 by James Schlatter, a chemist of G.D. Searle Company while testing an anti-ulcer drug, it has been a part of one of the biggest controversies in FDA history.

Despite the numerous studies presented to the FDA since 1964, there has been no prevailing evidence that establishes the safety of Aspartame especially in everyday consumption. However, Aspartame and its amino acid isolates have been implicated as one of the causes of neurodegenerative diseases such as ALS, Alzheimer’s, Parkinson’s, MS and seizures. Yet, Dr. Arthur Hall Hayes Jr. overturned the board of inquiry’s decision and approved Aspartame for dry goods in 1981 and carbonated beverages in 1983 even though Dr. John W. Olney found that oral intake of all excitotoxic amino acids cause brain damage in mice and informed G.D. Searle that aspartic acid caused holes in the brains of mice.
In a prepared statement by Dr. John W. Olney, M.D. titled, “Aspartame Board of Inquiry,” Dr. Olney showed how aspartate and several other structural analogs of excitotoxic amino acids have well-established brain damaging and neuroendocrine disruptive effect when administered systemically to various animals species. Animals of any age are vulnerable, however, young animals are more vulnerable at lower doses than adults are (Olney, 1980).

Brain damage occurs with oral administration of aspartate and glutamate because neurotoxins have free access from blood to certain brain regions that lack BBB. Because humans develop much higher glutamate and aspartate plasma levels from a given intake load than do experimental animals, humans are at a much higher risk for either brain damage or neuroendocrine degeneration than animals.

In 1974, Dr. Olney demonstrated to G.D. Searle that oral administration of Aspartame to infant mice results in lesions in the Circumventricular Organs (CVO) regions of the brain with minimal loading doses required to destroy the central neurons. Searle claimed that Aspartame does not damage monkey brains based on their own experiments. However, because all the animals Searle tested developed brain tumors, convulsions and died, the tumors were removed from the animals and were kept under lock-and-key for close to a year before the FDA would be allowed to view them (Evangelista, Sweet Misery, 2005).

Due to Searle’s persistent tendency to avoid performing experiments of appropriate design to clarify the safety of Aspartame, Searle failed to establish the safety of their product and then decided to do the unacceptable-experiment on humans without sufficient knowledge and comprehension of the chemical they would be ingesting.
Section 1 of the Nuremberg Code begins with the following two sentences: "The voluntary consent of the human subject is absolutely essential. This means that the person involved should have legal capacity to give consent; should be so situated as to be able to exercise free power of choice, without the intervention of any element of force, fraud, deceit, duress, over-reaching, or other ulterior form of constraint or coercion; and should have sufficient knowledge and comprehension of the elements of the subject matter involved, as to enable him to make an understanding and enlightened decision" (Nuremberg Code, 1949).

In 1906 the Pure Food and Drugs Act was established as part of our government protection plan. The FDA’s mission is to protect the safety and wholesomeness of food. The agency's scientists test samples to see if any substances, such as pesticide residues, toxins, poisons and bio-hazardous chemicals are present in unacceptable amounts. If contaminants are identified, FDA takes corrective action. The FDA knows more about the toxicity of Aspartame than any other agency and yet has been a co-conspirator with the manufacturers of Aspartame to hide the truth about the irreparable damage this man-made chemical has and will create within the human body.

For sixteen years, Dr. Adrian Gross and Dr. Jacqueline Verrett, toxicologists working for the FDA, vehemently objected to Aspartame approval. The travesty lies not just in the fact that Aspartame is not safe but also that the manufacturer filtered out what they didn't want the agency to see. All the studies showing the convulsions, seizures, brain tumors, and neurodegenerative destruction were mysteriously made to look innocuous (Olney, 1996).

On January 10, 1977, a 33-page letter was written by FDA Chief Counsel Richard Merrill recommending to U.S. Attorney Sam Skinner that a grand jury investigate G.D. Searle for
apparent violations of the Federal Food, Drug and Cosmetic Act, 21 U.S.C.331(e) and Act 18 USC 1001, for "their willful and knowing failure to make reports to the Food and Drug Administration required by the Act 21, U.S.C. 355 (i) and for concealing material facts and making false statements in reports of animal studies conducted to establish the safety of Aspartame.” This action did not prove promising (Martini, 2007). It was Jerome Bressler of the FDA who wrote the Bressler Report on Aspartame safety in regards to G.D. Searle’s testing. Bressler claimed the FDA removed 20% of the written work because of how poorly the testing was done (Bressler, 1977).

Up until 1981, Aspartame wasn't fully approved because the FDA knew it caused brain tumors and seizures in lab animals (Gross, 1987). It wasn't approved until President Ronald Reagan got into office when it was obvious that a political tug a war was going on between the head commissioner and G.D. Searle. Soon the manipulation, lies and deceit worked in G.D. Searle’s favor and Aspartame found its way into America's unsuspecting stomachs. Interestingly, when the FDA Commissioner, Jere E. Goyan, Ph.D. wouldn't approve of Aspartame, President Ronald Reagan wrote an executive order making the FDA Commissioner powerless to oppose Aspartame (Gordon, 1987). He was ultimately fired, and Dr. Arthur Hull Hayes, Jr. was appointed in his place. Even then, there was so much residual opposition to Aspartame that Dr. Hayes had no choice but to set up a Board of Inquiry that ironically ended up advising him "not to approve Aspartame." Dr. Hayes overruled his own board, approved the use of Aspartame in carbonated beverages, and then curiously left his appointed position for greener pastures as a consultant with a subsidiary of the G.D. Searle Company, manufacturers of Aspartame!
On June 5, 2004, former president Ronald Reagan died. He had been suffering with Alzheimer's disease for 10 years. Maureen Reagan, his daughter died of brain cancer in 2001. The question posed was Aspartame a part of their daily diet, and if so could it have been the factor that created these diseases?

As Dr. Russell Blaylock states, “Although excitotoxins are widely distributed in our food supply, we may not be able to depend upon the Food and Drug Administration (FDA) to protect us from these toxic, excitatory amino acids” (Blaylock, 1994)

Those most susceptible to the deleterious effects of excitotoxins are the very young and the very old. The blood-brain barrier (BBB) excludes many of these substances because it is a built-in protective mechanism for the central nervous system. However, the BBB is not fully developed in the very young, and it is easily damaged by excitotoxins that cause brain insults in older people.

In 1984, Norma Vera, a medical secretary, was placed for a period at the offices of G.D. Searle International, Inc in Coral Gables, Florida to work with Dr. Miguel Ortega in assisting him in translating Aspartame studies done in Mexico, Argentina, and Guatemala. The human experiments were carried out in six villages and then the field notes from the studies were sent to Mexico for compilation, printing and binding. The villages were broken up into three groups, consisting of fifteen to twenty subjects, in a combination of males and female subjects with the majority being very young people. The test subjects were told that the Aspartame that they would be taking in was a derivative of the papaya fruit (Affidavit of Norma Vera, 2004).
It was not long before the translation of these studies, from Spanish to English, revealed that Aspartame was hazardous to the health of the people being tested. It became Dr. Ortega’s mission to insure that Aspartame was never approved for human consumption. The study results were obvious and chilling. In all six study groups of those given Aspartame, 70% to 80% developed astrocytomas. Ms. Vera stated, “Aspartame usage seemed to cause the brain fluid to thicken and to slow down or completely stop the transmission of signals between brain cells and resulted in Alzheimer’s-like problems.” Twenty-five percent to 30% of the groups developed behavioral problems that caused them to become irritable and argumentative, and 10% to 20% of the females in the groups had spontaneous bleeding episodes (Affidavit of Norma Vera, 2004).

Approximately 90% of the subjects experienced either grand mal or petite-mal seizures and 90 – 95% experienced headaches or migraines within a very short time after ingesting Aspartame. The group experienced tremors in muscles, vertigo, disorientation, and loss of balance and loss of memory. One woman began bleeding profusely, had a miscarriage, and was eventually removed from the study (Affidavit of Norma Vera, 2004).

In 1985, Dr. Ortega, who was adamant about Aspartame never being approved, was directed by G.D. Searle to destroy all his records. Dr. Ortega refused to do so and as of today, no one has been able to contact or locate him. It is as if he has vanished off the face of the earth.

Aspartame is not only a deadly substance but the forces behind its approval process have demonstrated to be equally as deadly. Animals and humans alike were subjected to a multitude of “guinea pig” tests as they were unknowingly used in experiments. Aspartame should have never been approved based on the information submitted to the FDA, including the information that was not submitted. It is a fact that politics and those with 'hidden agendas' illegally affected
the regulatory processes, misused and abused their authority, to bring to market this toxic food additive.

To coin a phrase, “The truth is out there!” However, it has been difficult to come by. Countless victims, including investigators, lawyers, doctors, lay people, teachers, educators, regulators, and whistleblowers, have managed to piece together this highly complex puzzle and now see this issue for what it is. The evidence is overwhelming. Nevertheless, many physicians are afraid of the truth for it would go against their belief in their own system. When a patient comes to them with any of the symptoms listed on the FDA’s adverse reaction list and they are not questioned to whether they are users of Aspartame, that neglected action should be considered medical negligence. The same understanding holds true to the layperson that has a responsibility to do their own research and self-testing.

To get to the truth, one would have to punch holes into the world with a propensity for creating a false reality of health. There are large government health associations and many other so-called health groups that have received substantial money, contributions, or stipends from the manufacturers of Aspartame in regards to promoting their product. The very people who are supposed to be helping the public are willingly accepting underhanded bribes, in the name of financial support for their cause, for their own voracious greed and because of this diabolical system, the truth is screaming to be set free.

In 1993 the FDA approved Aspartame as an ingredient in numerous food items and removed all restrictions from Aspartame allowing it to be used in everything, including all heated and baked goods.
Concerns about Aspartame frequently revolve around symptoms and health conditions that are allegedly caused by this artificial sweetener. In February of 1994, the U.S. Department of Health and Human Services released the listing of adverse reactions from Aspartame reported to the FDA (DHHS, 1994). Aspartame in the food supply accounted for more than 75% of all adverse reactions reported to the FDA's Adverse Reaction Monitoring System (ARMS) to adverse reactions to this substance in the food supply from 1981 to 1995 (Food Chemical News, 1995). In 1995, FDA Epidemiology Branch Chief Thomas Wilcox reported that there are over one hundred thousand complaints now registered at the FDA and 92 different symptoms, including death; health conditions including Parkinsons, ALS, seizures Alzheimer’s and MS have been reported by physicians and consumers to the FDA. (Department of Health & Human Services, 1993).

However, the FDA now has plans to discourage or even misdirect complaints about Aspartame to the suicide hotline. Apparently, they don’t have the manpower to handle all the calls. The saddest fact about all of this is that most victims have not taken the time to educate themselves about the misinformation disseminated out by the companies manufacturing this poison. In December 1992, G.D. Searle's patent extensions on Aspartame expired, allowing other companies to produce Aspartame as well.

As Dr. James Bowen told the FDA many years ago: "the only responsible action would be to immediately take Aspartame off the market, fully disclose its toxicities, offer full compensation to the injured, public and criminally prosecute anyone who participated in the fraudulent placement of Aspartame on the marketplace. That includes those who work so
diligently to keep it on the market as well." The FDA still finds no reason to alter its previous conclusion that aspartame is safe as a general-purpose sweetener in food.
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cori@soundandfury.tv


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