WHILE SCIENCE SLEEPS

A Sweetener Kills

The introduction of aspartame into the food supply of the United States began in the summer of 1981. Since that time, the incidence of Alzheimer's deaths has increased 100 fold (10,000%). Autism has, with no explanation, increased 25 times (2500%). Autoimmune diseases have reached epidemic proportions, with Lupus (SLE) up 300%, and Multiple Sclerosis, Type II Diabetes and Rheumatoid Arthritis headed out of control. Cancers, the hallmark of formaldehyde exposure, have exploded. Skin cancer has shot up over 400%, liver cancer has tripled, kidney cancer has doubled, and breast cancer is up 50%. The list goes on.....

This 250 page, full color book uses over 100 colorful illustrations, photographs, tables and graphs to explain to the average person the fascinating process by which methanol, a poison hidden in aspartame and some other foods, is converted to formaldehyde at the very locations in the human body where these diseases originate, revealing, for the first time, the exact details of the probable cause of each. It is a cautionary tale of the legacy of the danger of a poisonous food additive and the failure of a government, corrupted by greed, to safeguard the health and welfare of its people.

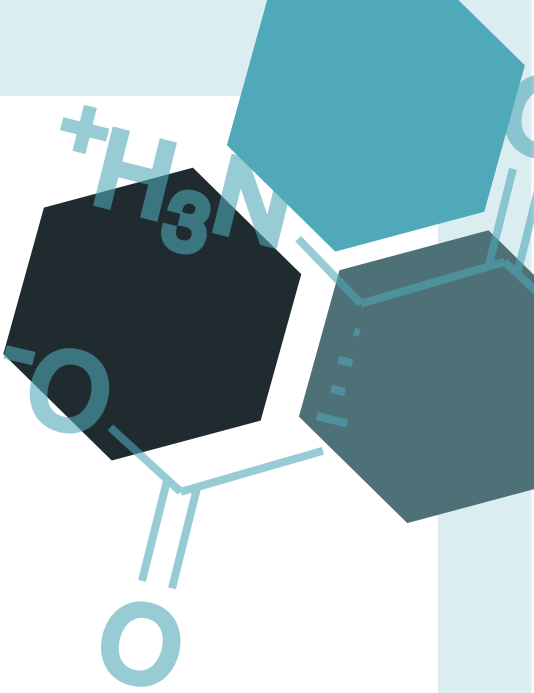
This is a handbook that teaches the tools you will need to protect those you love and inform them about the causes of a number of diseases that have, until now, proven inexplicably elusive to a medical community beholden to Big Pharma ...*While Science Sleeps*.

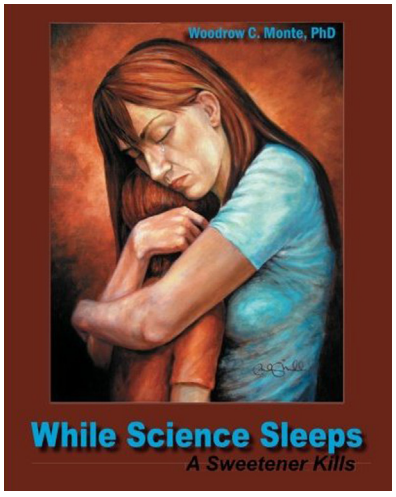
About the Author

Dr. Woodrow C. Monte, Professor Emeritus of Food Science and Nutrition from Arizona State University, has decades of experience in food science and nutrition as a researcher, teacher, inventor, industry consultant and consumer advocate who is committed to food additive safety and the prevention of food borne diseases. For over 30 years he has studied the link between artificial sweeteners and the diseases of civilization including Alzheimer's, Heart Disease, Multiple Sclerosis, numerous forms of cancer, Autism and other Birth Defects.

Dr. Monte's testimony before Congress was instrumental in the prevention of Sulfites from receiving status of US FDA GRAS (Generally Regarded As Safe) and the implementation of mandatory labeling for most foods that contain this dangerous additive.

Through his research, Dr. Monte has been awarded 22 US patents. He has shared his technical expertise during hundreds of television and radio appearances including a special feature on the CBS Evening News with Dan Rather and 60 Minutes. He is the author of numerous scientific publications and the book *While Science Sleeps: A Sweetener Kills.*





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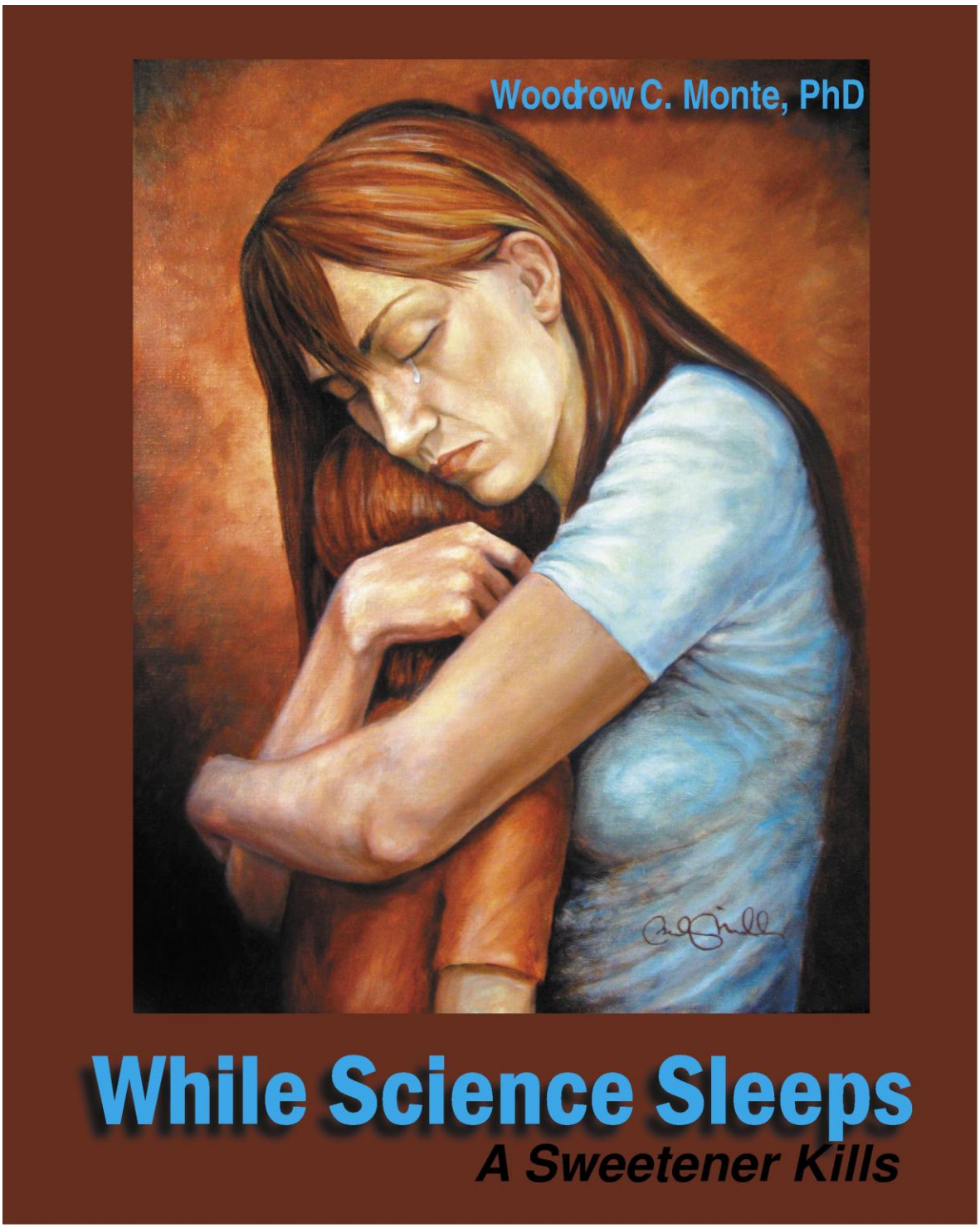

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Chapter 9
Multiple Sclerosis

Nature does not recognize disease; in her eyes, all has purpose, all is equal, and no one natural process is better than another. If you seek the cause of any unknown you are wise not to impede your vision by limiting your course of study. Learn about a natural mystery by taking it in completely; smell and taste it if you can. You must make close acquaintance with what you study before you can hope to see how it fits, discover its origin, and resolve how to make peace with it. View all of life's systems as timely events of a perfect interaction between equals – and you may begin to see their purpose. Let your prejudice be engaged only after the whole truth is plain to you and the course is clear.

We all die. The sentence is imposed at the first beat of our anxious hearts. Few suffer a more prolonged passing than those who succumb to multiple sclerosis (MS). The symptoms, a constant reminder that the brain is losing control of the organism with which it was once master. The young women who are the primary target of this tragic disorder can only cling to hope of a misdiagnosis or the rare outcome of complete remission. We will in this chapter discuss this disease as if the cause has been discovered and relief is on the way. I will be ridiculed for this approach, but then abuse has been the burden of discovery since the beginning of thought.

The Conflagration That Is Multiple Sclerosis

Multiple Sclerosis was on my mind when, at the dusk of a lovely summer day on a flight over California, I witnessed another of Nature's catastrophes – the unforgettable sight of a forest fire engulfing an entire mountainside. Thousands of acres of blackened bare tree trunks were outlined by a thin encircling ring of flame where the damage effectuated. It immediately occurred to me that this was a very powerful descriptor of MS plaque. Plaque is the anatomical equivalent of a slow forest fire, where nerve axons are being slowly stripped bare of their insulation. It is the defining feature for which the disease was first named by the French neurologist Jean-Martin Charcot in 1865.

I was in an introspective mood, and a stout drink or two later I began to daydream. What if, I imagined while reminiscing about the miles of desolation, an alien space ship were to land at the scene of this fire some time after the terrain had gone cold? What if these were brilliant alien scientists and explorers from a planet with little oxygen in the atmosphere and, therefore, no such phenomenon as fire? Surely their curiosity would draw them to this desolate spot that was so different from anything they had ever experienced on their own planet. Their cargo bay would soon be loaded with samples of all manner of charred remnants of plant and animal matter in various degrees of deterioration and decay, along with detailed pictures of the damage done to the terrain and life forms.

As a fellow scientist now caught in such a conundrum myself I imagined their fruitless search for the cause of this blight in the midst of a vibrant living forest, the endless rhetoric that would ensue during their long return flight home, the search for the invisible beasts responsible, the manuscripts of descriptive biology and theoretical speculation written after years of study and debate back on the home planet that had no knowledge of flame. As I write this, lying on my desk, long closed, is a damaged copy of just such a tome, the fourth edition of McAlpine's *Multiple Sclerosis*,[#305] a fifteen pound, thousand page technical masterpiece done in six colors and a print far too small to be easily read by those visually

impaired MS sufferers it purports to describe. The damage to my copy was caused by my ripping it in half to save postage between the two continents I call home. This publication would have much in common with the works produced by my imaginary visiting alien intellectuals. It contains a collection of hard won scientific data, factually correct but in the end, due to just one missing bit of knowledge, providing no help to the suffering and no answer to the most important question: *Why?*

McAlpine's is not listed among the handful of literature that I classify as must reading for someone who wishes to learn about multiple sclerosis. This compendium of the "present knowledge" belabors the trivia of the long history of medicine's failures with MS while incredibly failing to encourage in any way the compelling evidence that support the possibility that an environmental poison might just be its cause. Methanol and formaldehyde do not appear anywhere among its near million words. The bias of the chief editor is expressed in print[#616] in his critical, unreferenced review of an article which I hold in great esteem, authored by a good scientist who steps back and takes a careful, thoughtful, yet critical view of his own discipline and how it connects with reality. His approach is rare, especially among the ranks of the Delphian medical sciences. If you have MS or love someone who does I encourage you to read the brutally honest and thought provoking twenty page review *The Pathogenesis of Multiple Sclerosis Revisited,*[#615] and while you are at it take a look at *What if Multiple Sclerosis isn't an Immunological or a Viral Disease? The Case for a Circulating Toxin.*[#153] These articles will prepare you for what I have to say about the subject.

The hostility between the various factions of those who now study multiple sclerosis may actually be encouraged by the machinations of the major drug companies who sell extremely expensive competing palliatives for the disease, none of which cure it. The rivalry between these companies, who are responsible for directing most of the funding for the search for the "cure" of MS, is inspired by the fact that even the least effective of their concoctions have proven to be billion dollar product lines, due solely to the desperation of MS sufferers and their loved ones. To trust the cure of any disease to a group whose existence depends upon its perpetuation seems counterintuitive, if not outright stupid. (By "group," I mean both the pharmaceutical companies and the various MS societies who encourage and support each other in, to my mind, unproductive ways.)

The heated debate between the various competing groups of scientists studying MS is a debate between those who believe that MS is a defect in the immune system, those who think it is a viral disease, and those very few who believe that the disease is the direct result of a toxic agent from the environment. Though it is well known that animals do not develop MS there is no consensus as to whether animals can be used as experimental models for the disease when artificial methods are used to induce them to lose myelin. These issues have been continually debated for over a hundred years with, up till now, each side presenting only enough evidence to show that they may be on the right track but not enough to assign culpability. Aside from what I will demonstrate to you in this chapter there are few recorded instances where the incidence of MS has actually been manipulated. When Norway was occupied during World War II food was severely restricted for several years and the incidence of MS went down dramatically, unfortunately this has been virtually ignored.[#153]

I am not so very much interested in belaboring every detail of the slow complex conflagration that is multiple sclerosis. My intention here is to give you what you need to know to prevent the fire from reaching the fuel.

Listening to Nature's Whispers

In June of 1550 the Spanish conquistador Cortez kidnapped and eventually killed the Aztec emperor Montezuma in a successful attempt to steal the golden treasures of the Aztec people. From the time of Columbus it was the admitted intent of European invaders to plunder the newly discovered territory of the Americas, no matter what the cost to the "savages." Among other bounty were the golden seeds of a plant unknown to the conquering rabble. This was to be the first of the corn seeds from the Americas to be received enthusiastically by the old world, but to have attached unforeseen dire consequences. The seeds did not come with instructions. Although the Aztec people had much they could teach the Spaniards, their traditions were not heeded. Perhaps these Europeans had such a high opinion of their own civilization that they could not easily accept cultural knowledge from those they so easily conquered. Whatever the case, unimaginable misery was the wage of implementing a new food without sufficient knowledge of its safety. Pellagra, the disease of corn, was to take European scientists over 400 years to rediscover and cost millions of innocent lives to a slow and agonizing death.

Eight thousand years before the Old World finally put an end to their culture, the Aztecs had begun experimenting with native grasses, turning them into something new to nature. They persisted, and after generations of observation and painstaking manipulation, with careful crossing of strains, they produced a new life form that could have never evolved naturally. It was the birth of what appeared on first blush to be a valuable food crop – corn (maize). No record exists of who these individuals were, but without a doubt the work that they performed was a non-invasive genetic manipulation (GM). The most critical way that Aztec science differs from our modern GM is that the Aztecs took the ultimate responsibility for bringing their work to completion. Over a period of years the dark side of their unnatural invention was to present as a plague of biblical proportions. Entire villages of corn eaters would succumb to symptoms of what must then have appeared to be many different diseases. Some individuals would develop devastating and painful skin lesions, while others would lose all semblance of civilization and go mad. Death would finally come to most when the heart would refuse the brain's signal to continue beating.

By observation, Aztec scientists were able to determine that it was the corn that was the cause of these apocalyptic outbreaks and further, and of much greater significance, they observed that when corn was treated overnight with powdered limestone or the ash from a wood fire the suffering would not come. By pure and patient observation of Nature's response to their own unnatural product these ancient people solved the riddle of Pellagra and successfully implemented a reliable prevention. This complex conundrum that we know now was a nutritional deficiency of niacin was laid to rest by observation, intuition and intellect alone.

We have already discussed other ancient cultures that were adept at such research and developed traditional adaptations of the way they prepared food to ultimately prevent premature death and suffering caused by nutritional deficiencies and poisoning from naturally occurring toxic substances. With the exception of the accidental discovery of antibiotics by a university microbiologist and vaccination by a country physician who was really more of a naturalist, our modern medicine men are wretchedly inept at discovering the cause and prevention of disease. If only we could discover what is so very wrong with our scientific methods that makes it impossible for our researchers to tease such revelations from Nature's grasp! We can only guess that part of it may be that present-day medicine appears to have no stomach for the combination of observation, intuition and common sense that ancient peoples would have been forced to employ to come to their cures for food borne diseases. Modern medical researchers, particularly those funded by the pharmaceutical industry, often demean such

observations by using the term "anecdotal" and employing an inflection and tenor as if they were uttering a curse.

My use of observation, intuition and common sense, combined with careful study of the research of many whose narrow perspectives prevented them from seeing the answers they sought, has led me to believe that diseases of civilization (DOC) and, in particular, multiple sclerosis, were originally caused by human meddling with the food supply. In this chapter I will apply both anecdotal observation and the more traditional methods of modern science to point to the cause and prevention of the poisoning that causes MS.

The Scene of the Crime

The brain, along with the rest of the central nervous system, is made up of about 50% neurons (nerve cells) that communicate with other cells of the body via long wire-like strands called axons. Multiple sclerosis results from the progressive deterioration of the protective myelin sheath surrounding the axons. Axons are in many ways similar to a telephone cord, which is made up of a conductive wire surrounded by a protective coating, without which the signal would be interrupted and muted as it travels from the mouthpiece to the phone line. The disappearance of myelin distorts nerve cell communication, leading to a long list of neurological symptoms including loss of sensation, muscle spasms and weakness, fatigue, blindness, and pain. The entire collection of symptoms that constitute multiple sclerosis can be explained by damage to the myelin sheath of specific axons in the brain and upper spinal cord. All the symptomology deriving from the interference to the normal communication between the brain and the rest of the body is due to what is tantamount to short circuits in axons that transmits life sustaining information in both directions.

Specifically, damage is found around the axons that transmit signals to and from the muscles and sensor cells. Even though the impulse that runs down the axon is not identical to the flow of electrons that runs through an analogous electric wire, nevertheless the myelin must be insulated to provide for the security of the messages traveling through it and to prevent the bare axons from touching and allowing signal cross over between nerve cells or, even worse, the breaking of the unprotected axon itself. The one major difference in the appearance of the myelin sheath when compared to the insulation on an electrical wire is that the nerve cell's insulation is applied in numerous evenly spaced lumps, called nodules, that run down the length of the axon, giving a healthy axon the appearance of a strand of beads. Each nodule consists of many layers of myelin wrapped in one continuous band around the axon it protects.

The disease is called multiple sclerosis because usually the myelin sheath can be damaged at multiple sites in the brain. These sites are not haphazard; they have a pattern which is characteristic of the disease and consistent with the usual symptoms. Interestingly, these sites are identical to the sites affected during methanol poisoning.

MS damage to the brain always begins in areas adjacent to blood vessels. Dr. James Dawson at the University of Edinburgh in 1916 performed detailed examinations of the brains of patients who died from MS. His meticulous descriptions of the inflammation and swelling of blood vessels and the damage to the adjacent myelin sheath is valid to this day[#620] and again is startlingly reminiscent of the damage caused by methanol toxicity.[#15] This presentation, consisting of the damaged vessel and many adjacent axons which exhibit complete demylination, is plaque. These plaques radiate out from the vessels around which they are centered. Plaques enlarge three dimensionally over time from the central vein or

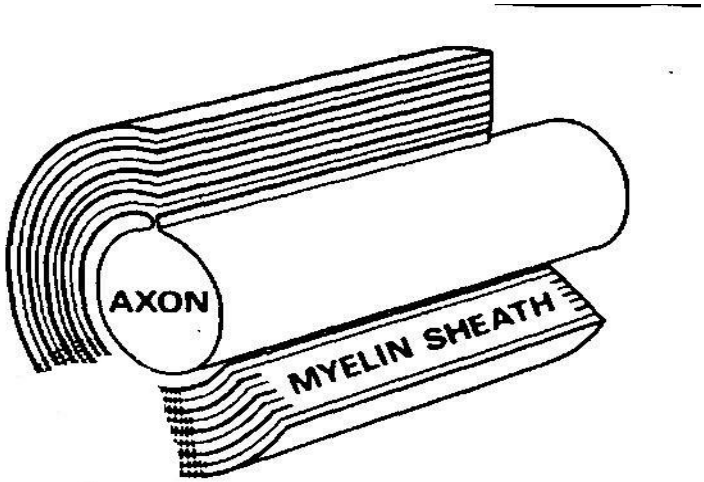
small artery outward until they give the appearance of the growing limb of a tree. The analogy stretches to the bark representing the "active" area of the plaque, where we can find macrophages busy consuming the myelin sheaths of previously healthy axons as the plaque slowly grows with the progression of the disease by what appears so obviously to be diffusion of a deadly poison.

Each human nerve cell has only one axon, some of which can reach over a foot in length. These axons have to be insulated, padded, protected, and fed. Functions that are not provided directly by the neuron (nerve cell) itself, because the distances are so great between the body of the cell and the end of the axon. Rather, the axon is cared for by other cells of the nervous system to be found along its path in the brain and spinal cord. It is the Schwann cell which takes it upon itself to maintain one short segment of axon. To do this it must stretch out a thin layer of its cell membrane and grow that layer around and around the axon many times. These windings are what become the myelin sheath. Thousands of these Schwann cells are spaced at remarkably consistent intervals along the path of the average nerve cell axon, giving the axon the look of a garden hose strung with evenly spaced rolls of paper towels. The Schwann cells remain attached and are physically very close to the axon, providing life support to the living myelin sheath. They can also act to regrow the sheath when it is in need of repair or replacement. The regrowth is slow, but it is precisely what accounts for the periods of remittance which follow the relapses that often occur in MS sufferers.

It is this symbiotic relationship that helps explain the major difference between Alzheimer's disease, which is caused by damage deep inside the axon and is irreversible, and multiple sclerosis, which is a disease of the myelin sheath and is repairable, causing multiple cycles of exacerbation and remission. The modus operandi of formaldehyde during the evolution of MS is identical to that of Alzheimer's – even to the attack on the axons of the nerve cells in close proximity to the small veins and arteries of the circulatory system of the brain. The difference is that during Alzheimer's, the formaldehyde penetrates into the axon of the nerve cell itself, attacking the basic tau protein within. This process eventually kills the entire nerve cell, which cannot be replaced. In multiple sclerosis, it is the protein of the myelin sheath, which is actually part and parcel of the Schwann cell, that is being attacked and consumed by the activated macrophage. The axon of the nerve cell is spared at first, just as what happens in acute methanol poisoning. To put this simply, Alzheimer's is a disease of the nerve cell (neuron), while MS really begins as a disease of the Schwann cell. Eventually as MS progresses the loss of the protective sheath will result in breakage of the axon and repair becomes impossible.

Figure 1 shows a close up diagram of what the layers of myelin look like wrapped around the axon. It reveals what happens as the myelin is devoured by hungry macrophages layer by layer, thus exposing once protected MBP to marauding bands of crazy hawks, leading to its modification by formaldehyde and its eventual consumption, finally exposing the unprotected living axon of the neuron. Some considerable time is required to finally erode away the many layers of myelin. The fascinating thing is that many Schwann cells survive these attacks and can and do regrow their myelin sheath. Although once considered controversial, complete remyelination of demyelinated axons within even large plaques has recently been shown to be commonplace.[#230] This explains the often complete disappearance of MS symptoms during remission in many MS sufferers. In most patients with MS the disease takes an up-and-down course with long periods of remission between attacks. This implies a valiant attempt of the Schwann cells to rebuild missing myelin sheaths consumed by the macrophages.[#151] If we can stop the poisoning early enough – before too many axons break and Schwann cells are killed and the hope for complete recovery is lost – by removing from the environment and food supply the poison that causes it, methanol, then perhaps MS can actually be cured.

Figure 1



The microphotograph of a section of a small vein in Figure 2[#517] shows clearly the plaque formation surrounding the blood vessels in a patient with early MS. The dark spots are macrophages eating away at myelin. Figure 3[#517] shows cross sectional views of the same tissue that can be more easily compared to the figures in the previous chapter of perivascular Alzheimer's plaque. As time progresses and plaques age and become larger, the unprotected thin axons may eventually break from lack of physical protection, cutting completely all communications and leading to the atrophy of muscles and organs and eventually death from lack of functioning of vital tissue.

Figure 2

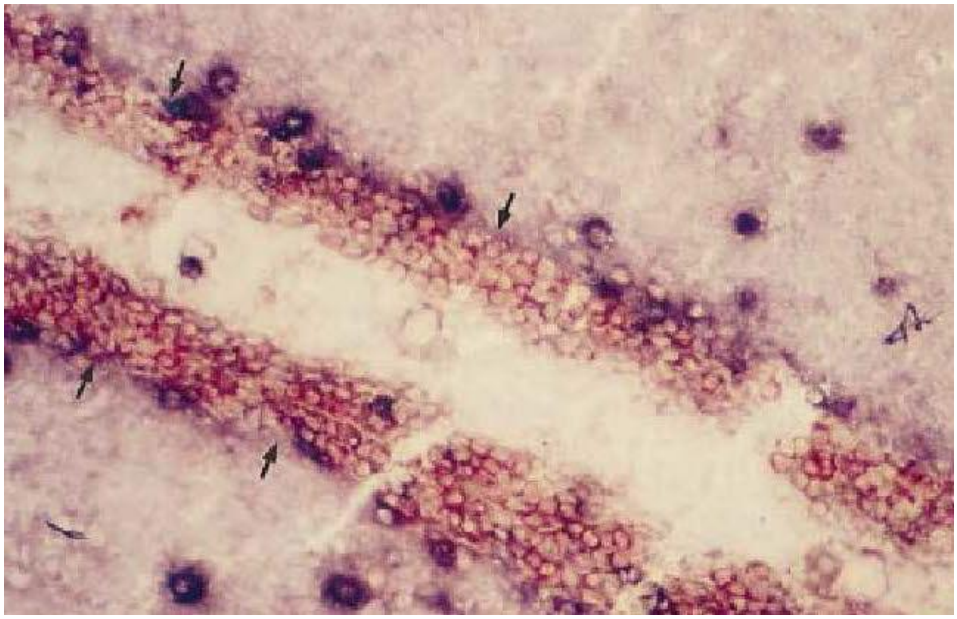
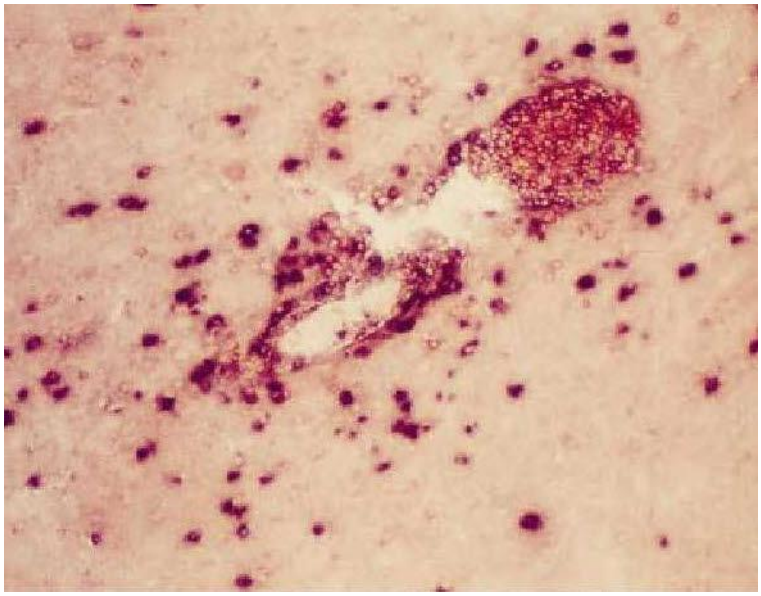


Figure 3



Location, Location, Location

As you now know, human blood vessels are the location of much of the alcohol dehydrogenase class I outside of the liver (extra-hepatic ADH). The intima and media of both the veins and arteries of the brain contain ADH,[#220] which is capable of formaldehyde production from methanol. Once produced, formaldehyde will tirelessly search for a basic molecule with which it can react. The travel distance of formaldehyde within the human body is limited by its extreme reactivity. The formaldehyde hawks find much to keep them busy close to their point of origin and will not wander further than their nearest molecular prey. Yet it must always be remembered that formaldehyde is the smallest of molecules and is capable of going anywhere very quickly if not distracted. Much like a real hawk, formaldehyde that isn't reacting with something is constantly on the move. Once formaldehyde is produced from methanol it diffuses out, mostly in the acid form (crazy hawk), into the watery world of human tissue, hunting for prey.

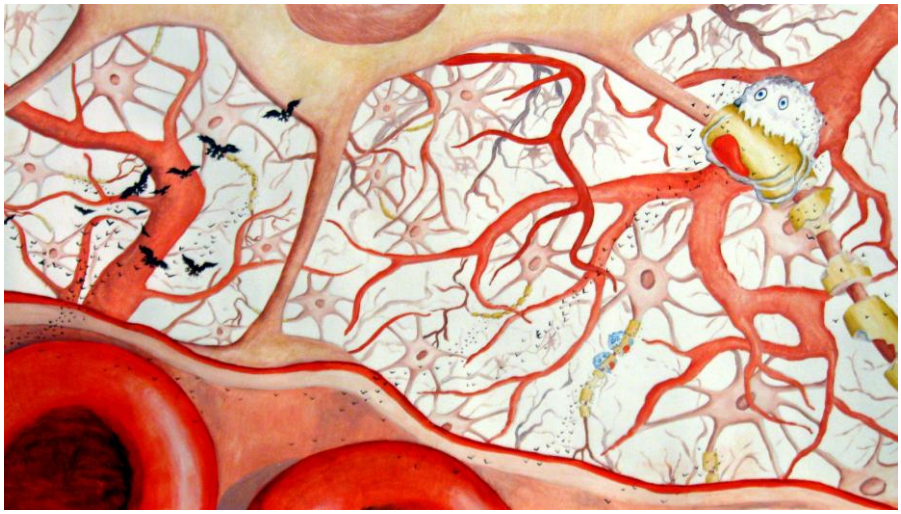
What this all means is that much of the damage from methanol poisoning will be found close to home within tissue surrounding the blood vessels and in close proximity to the ADH sources. Only a handful of diseases are well-known to conform to this "perivascular" modus operandi with most, if not all damage clustered in sites surrounding blood vessels and, in particular, small veins. This anatomical behavior has always been lacking explanation. The two most important of these perivascular diseases are multiple sclerosis[#517] and Alzheimer's. The reason that the vascular sites of ADH formaldehyde production are so critical to the elucidation of both these diseases of the brain is that, other than these sites within the lining of the cerebral veins and arteries, the brain is completely free of alcohol dehydrogenase class I.[#218] In other words, the plaques of MS and the damage of Alzheimer's *only* occur in the *precise areas* where the ADH that converts methanol to formaldehyde is found.

The good news for many, but obviously not all, is that the brain also has very significant,[#327] but genetically serendipitous, distributions[#216] of the good enzyme ADH III, which can destroy

formaldehyde, turning it into the safe formic acid before it gets a chance to attack a basic proteins. Formaldehyde is, therefore, produced only by the ADH I found in the lining of the blood vessels of the brain and not in brain tissue itself.[#528] It is a short journey from the blood vessel, where the formaldehyde is produced within the lining of the vessels themselves, to the locations where MS plaque originates on the myelin sheaths of the numerous axons crisscrossing these vessels. It is noteworthy that some of the very first damage shown in the natural progression of MS is a swelling (edema) and thickening of the lining of these very vessels where the formaldehyde is produced, even before the nearby myelin appears to be affected.[#517] It is again fascinating that this damage is identical to that found in unfortunate individuals who die slowly from acute methanol poisoning.[#15] We can take this as an indication that formaldehyde does some damage to other proteins on its way to finding a more desirable nesting site like myelin basic protein (MBP). In Figures 2 and 3 MS can be seen slowly progressing along the length of these small arteries and veins throughout the brain and spinal cord.[#517]

Please try to imagine flocks of formaldehyde hawk fledglings emerging from these blood vessels into the circuitry of the brain. Imagine wave after wave of hungry hawks looking for protein to attack, having to go further and further as protein landing sites are lost to competitors and then eventually consumed by macrophages, slowly but surely expanding the plaque as the years go by (Figure 7). It is worth noting that a single drop of diet cola sweetened with aspartame contains sufficient methanol to produce well over a hundred trillion of these damaging crazy hawks. What exactly are the crazy hawks attacking?

Figure 7



Details of Plaque Formation

Figure 7 shows us some older MS plaque with almost all of the myelin sheaths having been eaten away by those cute little big eyed macrophages. This forces the crazy hawks, which, if you look closely, are emerging from the blood vessel in the foreground, to fly great distances to find more protein to attack at the war zone at the very edges of the plaque. What are the crazy hawks trying to find, and how do we prove that they are indeed attacking that particular protein? In order for my explanation to go further you need to learn a little something about one additional molecule that will prove to be an extremely important part of our chain of evidence that will establish convincingly that MS is a disease of methanol poisoning. But first a little background.

The Look, Touch, Taste and Smell of Multiple Sclerosis

I lived what seemed a lifetime in Wellington, Colorado in the years I spent earning my master's and doctorate degrees in Food Science and then Nutrition at Colorado State University. It was there that I tasted multiple sclerosis for the first time.

I met her in my Urban Renewal office, where I moonlighted from my research assistantship at the university to earn enough extra money to lead a decent lifestyle in that rough hewn western town on the Wyoming border. She was a striking tall blond woman with the deepest blue eyes and a blistering temper, and we became lovers in very short order. During the length of our relationship I lived with her multiple sclerosis in a more intimate manner than any specialist in the disease usually do. There was always a change in her that I could detect long before her suffering began, not in her mood or her movement, but in her taste and smell. I cannot say it was formaldehyde, but if formaldehyde had a sister it would have been that.

There is much more to multiple sclerosis then we may ever know, and we must be open to accepting evidence from every quarter. I doubt that Nature is an adherent to the scientific method and its null hypothesis.

The Kitchen Autopsy

One can never forget the vision of a scientist wearing a top hat and scullery apron bent over an oversized kitchen sink with his bare fingers thrust deep into the freshly removed brain of one of his MS patients. Jean-Martin Charcot, the discoverer of the disease, was notorious around the saltpeter institute, where he was chief neurologist, to have had subjected the freshly dissected brain of a patient who died from MS to a careful screening by running his fingers through the soft custard like tissue of her untreated brain finding the long perivascular strands of leathery plaque that gave him the idea for the name Sclerosis (French for leather-like). The image of him doing this is reproduced in a book on the history of MS.[#306] One can only imagine how he first encountered this extremely important telltale feature of the disease.

As the MS plaque grows larger, the formaldehyde hydrate produced in the lining of the blood vessels has to travel further and further to find new basic protein to bind to. The lingering of the crazy hawk as it wends its way to the outer reaches of the older plaque provides enough dwell time for some of the formaldehyde to react with deeper tissue within the plaque itself. This reaction of the formaldehyde with the already demyelinated plaque gives it a strikingly hard texture that is different from any other tissue to be found in the brain. This is what formaldehyde hydrate does to tissue and is one of the reasons that it is used for the tanning of leather and to plasticize other substances.[#446] The original physical description of these areas by Charcot in 1846 might have led to a quick resolution of the cause of MS, if only formaldehyde would have been known to science at the time.

Charcot would have no idea what formaldehyde did to tissue, for unfortunately, it was not until 1868, more than twenty years later, that Hoffman was to discover and name the highly reactive substance so easily produced by passing methanol fumes over a platinum wire, (just as in my hand warmer). It wasn't until Ferdinand Blum was hired by the discoverer of formaldehyde to find uses for the extremely dangerous compound that its plasticizing effect on tissue was elucidated and exploited. But at that time it was considered to be a laboratory oddity and not discovered as a natural substance until years later. Much valuable time was lost to ignorance. The trail of the small molecule as the cause of MS grew cold and to this day is rarely revisited.

The fact that brain tissue is never touched with bare hands in the modern histology laboratory, not even after it is soaked in a solution of formaldehyde for days, makes this type of observation unlikely and esthetically barbaric to the contemporary histologist, who rarely removes the latex gloves from his hands. It is a sad reminder of the physical removal of modern medical practitioners from the real world of patients.

The Cause of MS is within the Thickening Blood Vessels

We owe a great deal to Dr. James W. Dawson, the Scottish pathologist who dedicated his professional career to the microscopic study of multiple sclerosis. Prior to his death in 1927 from a lifelong tubercular infection he published a detailed 230 page histological examination of tissue from nine cases of the disease. Dawson's Histology of Disseminated Sclerosis,[#620] published in 1915, applied modern staining techniques that brought color and greater definition to the pathology of the disease. His work reinforced the perivascular nature of MS, along with revealing the details of the considerable thickening and damage to the walls of the blood vessels within the plaque. This well regarded work supplemented the microscopic analysis published by Charcot himself and numerous other scientists over the previous fifty years. Dawson's outstanding critical review of the scientific literature and his own summations give us a clear understanding and insight into the thinking of the medical community as to the progression and causation of MS during the critical early days after the first cases of MS were being reported. The noteworthy scientists of the early Twentieth Century were convinced that MS was caused by a toxic substance that, to their great disappointment, was invisible to them. James sums this up nicely as follows:

> *The supposition of a selective poison acting through the blood-vessels, which has received the support of most recent investigators, is justified as an hypothesis but remains undemonstrated as a fact. …This hypothetical toxin has not been isolated, but it is suggested that it forms in the body. …It is admitted by all, except the supporters of the developmental nature of the process, that the distribution of the plaque areas points to the blood-vessels as the route of conveyance of this agent, and the assumption of an intoxication harmonizes with this relation to the blood-vessels."*[#620]

Though no animal model is reliable for studying either MS or methanol poisoning, when rabbits (one of the few animals used successfully to study some aspects of atherosclerosis in man) were forced to breathe methanol fumes for 8 hours a day for several months, all developed a marked increase in the thickness of their blood vessels similar to what is consistently noted in MS pathology. Dr. Eisenberg's microscopic examination of the rabbits' circulatory tissues attributed the changes to "the actual proliferation of the fixed tissue cells, as seen by the very marked thickening of the adventitia and media of the blood-vessels."[#15] Most telling of all can be found in one of Oluf Roe's ground-breaking articles, published in the early 1940s investigating a massive outbreak of methanol poisoning that killed and blinded numerous of his countrymen. Roe made a profound observation while investigating the optic discs of several of his poisoned patients. These men, one of whose age was only 32 years, had survived between 6 and 12 weeks after their acute methanol poisoning. Dr. Roe, the father of the use of ethanol to treat methanol poisoning, states:

> *There was at the same time marked irregularity of the caliber of the large blood vessels with thickening of their walls. These changes resemble those seen in the blood vessels of the retina in arteriosclerosis."*[#49]

As is often the case it is the earliest of scientific investigators that make the real breakthroughs in discovering the cause of diseases. It is unfortunate that these great minds of the late 19th and early 20th Centuries did not have a way to visualize or detect formaldehyde. Although it was Charcot himself who first noted the thickening and obstruction of the small blood vessels of the MS plaque, it was Rindfleisch in Germany in 1863 who first identified the constant changes of the blood vessels in MS and presented what is today called the "vascular theory of MS." He noted the "enormous thickening" of the vessel lining within the plaque of the MS brain and proposed that whatever was being produced within these vessel walls was the cause of the disease.[#230] What was being produced was formaldehyde.

Symptoms Mean Little unless they are Identical in All Ways…Then They Mean Everything!

We have spent time on the symptoms of MS in Chapter 6, showing how methanol kills. In summary, the symptoms of multiple sclerosis,[#44],[# 83],[# 85],[#169] chronic and acute methanol poisoning,[#13],[#144],[#189] and Aspartame toxicity[#54],[# 58],[# 93],[#181] are in all ways identical. Nothing that happens to the human body from the toxic effect of methanol has not also been expressed during the course of MS...Nothing.[#143],[#144] This generalization extends even to the remarkable ophthalmological conditions common to both: transitory optic neuritis and Retrolaminar demyelinating optic neuropathy with scotoma of the central visual field (which occasionally manifests as unilateral temporary blindness.[#85],[#138],[#163] In fact, these ophthalmological symptoms have been thought for years in their respective literatures to be "tell tale" indications for the differential diagnosis for each of these maladies independently.[#85],[#138],[#148],[#163],[#169] The common symptoms of headache,[#13],[#83],[#181],[#189] nervousness,[#13],[#83],[#181] depression,[#58],[#83],[#189],[#181] memory loss,[#18],[#147],[#85],[#169],[#181] tingling sensations,[#13],[#85],[#168],[#138],[#169] pain in the extremities,[#13],[#85],[#169] optic neuritis,[#85],[#138],[#148],[#163],[#169] bright lights in the visual field,[#139],[#83] seizures,[#21],[#83],[#160] and inability to urinate or to keep from urinating[#139],[#146],[#167] are all shared by each of these conditions and shared yet again by complaints from aspartame poisoning.[#54],[#58],[#93],[#181]

I take these strikingly similar symptom patterns as evidence that these disorders act on identical components of the central nervous system and in the same way. In the early stages of MS, or when a non-lethal dose of methanol has been administered, blindness can occur often in just one eye. Complete recovery is a possibility. The only two afflictions for which such dramatic "remissions" are reported from identical neuromuscular and ophthalmological damage, including "blindness," are relapsing-remitting multiple sclerosis [#85] and methyl alcohol poisoning.[#138],[#163] The pathology of the two maladies is in all ways identical, particularly when it comes to destruction of the myelin sheath with no harm to the axon itself.[#18],[#148],[#176]

It is not unusual for the modern medical practitioner to put little credence in the similarity of symptoms between disorders, and it is indeed true that certain very common symptoms, such as headache and depression, can be caused by all manner of bodily changes. However, the breadth of symptomatological overlap that we are putting forth here between methanol poisoning and MS is far too robust to put to neurological coincidence. In total, they reflect a link that cannot be refuted with any degree of scientific vigor.

Learn a Little About Arginine

There are several important questions that need to be answered when a poison is proposed as the cause of a disease. The first is what is the exact chemical interaction between the suspected etiologic agent and the biological molecule or molecules that initiate the disease process, and how can it be detected? For years the stumbling block of DOC elucidation was the inability to detect formaldehyde. An important

new tool of scientific research, however, has recently made detection possible. To understand how this works we need to understand something about protein.

Our bodies are made of cells that are works of moveable art composed primarily of water and protein molecules. These protein molecules are the major constituents of all earthly life. The original recipe for each of these fascinating structures is protected deep within the nucleus of the cells, never to leave its safe haven in our lifetime. When a new protein is needed for repair or growth, that information can be copied directly out of the recipe book – or chromosome, as it is called – as long as the DNA that makes up the chromosome has not been previously turned off or methylated by formaldehyde (a methyl molecule). Just as your mom would copy a recipe from her tired old cook book onto a slip of paper and hand it to you to for reproduction in your own kitchen, so Nature scribes and passes out of the nucleus such individual recipes to be made into protein to build and repair your body. It is a fascinating structure called a ribosome that has the job of making each protein from the instructions on the recipe slip, putting together the required ingredients.

The ingredients are primarily amino acids. Floating around in the liquid of the cell (cytoplasm) are about 20 of these different amino acids, from which the ribosome can choose. These amino acids constitute the chemical alphabet that Nature links together for performing all of the purposes of living. One of these amino acids is called arginine. This is the most important amino acid for those of us who have an interest in methanol diseases, and it will be the only one that we will take the time to study here. If you are a chemist you know about the structure of the arginine molecule, but what interests us is its behavior. Arginine is one of only three amino acids that are basic in nature and it is the only one that can provide three different nesting sites for our acidic crazy hawk. This means that formaldehyde can react with arginine and methylate it one, two, or occasionally even up to three times. Arginine is considered a formaldehyde capturer.[#225] In fact, it is more than that – it is a trap for formaldehyde, and because of this it can be a tremendously valuable formaldehyde detector, telling us if brain tissue has made contact with formaldehyde. Thus, a molecule invisible for a hundred and fifty years now casts a shadow that can bear witness to its unwanted presence.

But before we can see formaldehyde's shadow, we must have a way to detect arginine as part of a protein. For then we could take protein from the myelin sheaths of MS sufferers and see if the arginine it contains has been modified by formaldehyde (or "methylated"). The good news is that we now have such a technique. The bad news is the evidence has been lost on those sleepy scientists who did not understand its significance. The story is a sad, yet hopeful one.

Myelin Basic Protein (MBP)

Myelin basic protein (MBP) is a major component of the healthy myelin sheath and, in fact, makes up over 35% of its total composition.[#311] For many years this particular protein has been suspected as being the one protein most negatively affected during multiple sclerosis. Because of this it has been extensively studied. We know the exact structure of MBP; it is made up of 170 amino acids, 19 of which are arginine molecules. MBP contains a higher percentage of arginine than any other human protein. This extremely high percentage of arginine is what gives MBP its very basic nature[#41] and from whence it's name is derived. The Crazy Hawk, formaldehyde hydrate, is acidic and acids and bases will interact if given the opportunity. The Crazy Hawk would be naturally attracted to MBP, bonding to its arginine and eventually methylating it and leaving behind an excellent record of its presence as described above.

Even though changes in the MBP composition of the MS brain are considered the most important chemical milestones of the disease's progression, neurologists were surprised to discover that MBP often completely disappears from the center of the MS plaque itself.[#707] Of greater significance was the discovery of an incredible selective loss of this basic protein in otherwise normal appearing tissue up to about a millimeter and a half beyond the edge of these plaques.[#311] If you use your imagination to visualize the flight of the Crazy Hawks outward from their source in the vessels at the center of the plaque, if the formaldehyde modification of MBP would activate macrophages, then its phagocytosis (consumption) could account elegantly for the selective disappearance of MBP from surrounding tissue.

Looking for the Shadow of Formaldehyde

A few years ago while living in a small oceanfront New Zealand village with a population of less than 100 souls, I met a young German couple, both PhD chemists, who rented a home down the street from my cottage. We were quick to be friends and got together from time to time to discuss the chemistry of methanol and formaldehyde. The young woman was kind enough to translate some of the early German work done on methanol toxicity that I had in my almost complete collection of scientific literature on the subject. New Zealand happens to be one of those countries of the world with an extremely high rate of Multiple Sclerosis (the reason for which we will explain later), and so inevitably one day the topic of our discussion turned to MS.

I explained how I believed that the disease could be caused by methanol's transformation within the lining of the veins into formaldehyde, which then modified the basic protein of the myelin sheath, marking it for destruction by activated macrophages. I ended the monologue with my usual complaint that the reason this had not been discovered was the absence of a reliable way to determine that a protein had been modified by the little formaldehyde molecule. Without any hesitation my translator's husband informed me that in fact a new analytical method had been developed that could take large protein molecules, break them up into smaller bits and determine accurately if a change had been made by even a single formaldehyde molecule (one carbon atom) in any of the pieces.[#706] The equipment (an ion trap storage/reflection time-of-flight mass spectrometer) was expensive, and it would not be easy to get time on it, but he had a friend at a German university who had access to one and for such a purpose he was sure he could schedule us the use of it. We immediately began putting together an experimental design that could prove or disprove once and for all if methanol was the cause of MS.

The brain tissue of MS sufferers could be compared to brain tissue from normal individuals. If we could show that the MBP from the normal brains contained arginine with little or no nesting crazy hawks (methylation) and, therefore, prove they had not been attacked by formaldehyde, this would be the first step. The second and most important step would be to test MBP from the areas close to and in the direct path of plaque formation in the MS brain to see if indeed more methylation of the arginine would be found there, giving us conclusive proof of formaldehyde modification. With that kind of evidence, surely the methanol hypothesis would be impossible to ignore.

We would have to apply to tissue banks around the world that stored frozen brain tissue from the cadavers of deceased MS patients and describe to their scientific committees our need for samples of their valuable tissue to perform our experiment. We would then use this fascinating new analysis method to determine if formaldehyde was setting the stage for the damage that became MS. Nothing else would be as effective in determining whether formaldehyde was altering the brains of MS patients as evaluating their brains for methylated arginine. Finally, we had found a way to prove methanol's

formaldehyde as the cause of an important disease of civilization. We parted in a state of great excitement. The real work started the very next day, with both of us hitting the computer and launching an intense review of the literature so that we would be able to write out proposals to the laboratories whose cooperation would be vital for us to carry out our research.

"Woody…They Have Done Our Experiment for Us.… But they Just Don't Get It!"

I will never forget the call I received the very next day. My friend was so excited that he spoke in German, and at first I couldn't understand what he was trying to get across. I only knew it had to do with what we had discussed the previous evening. The good news was that my German friend had found a reference that led him to believe that our experiment had been performed eight years before, in 2002, just as we had designed it. I asked him to email me the reference to the article so that I could read it for myself. The title was indeed titillating: *An Important Role for…Modifications of MBP in the Pathogenesis of MS*.[#224] The bad news turned out to be that those doing the study had not a clue of the scientific significance or impact of what they had discovered. They had found gold and reported the glitter, but they had no use for it. But the results will not be lost on your prepared minds, I promise!

Finding the Shadow of Formaldehyde in the MS Brain: The Smoking Gun (a triple blind study)

Although it is tragic when lives are lost to ignorance, Nature is, by definition, uncompromising and has no inclination to follow the simplistic paradigms of man's seriously flawed scientific didacticisms. The natural world cannot be expected to be completely understood by even the most competent individual investigator if that investigator is only trained in science, but not nature. Thus the results of any scientific study can have many interpretations, the least reliable of which is very often the conclusion of the extremely prejudiced corresponding author of the manuscript in which it is reported. Scientists often only report what they were actually expecting to find, leaving any troublesome "outlying" data and conclusions that may offend their peers in the editorial trash bin. This is why the more removed the scientist is from the interpretation of his own data the better.

A single blind study is a scientific study performed on human subjects that are purposely kept in the dark as to whether or not they are part of the experimental group (usually a group being given a study medication or treatment, or suffering from the disease being studied) or the control group (usually a group being given a placebo or a group that is reflective of the general population). In other words, only the subjects are "blind" and, therefore, unable to purposefully or subconsciously act to skew the results. When the researchers are also kept from knowing this information until after the interpretation of the data, we have what is called a "double blind" study because both the researchers and the subjects are "blind," preventing either party from acting to affect the results. When researchers stumble on a result that they were not expecting and that they cannot explain, I call that a "triple blind" study because all pre-conception, prejudicial design, and ability to skew results has been removed, and the data is pure. Just as a double blind study is better than a single blind study, I consider a triple blind study to bear the most important fruit of all.

The original discoveries of the U-shaped curve that proved the protective effect of low levels of alcohol consumption against various diseases of civilization were all reported reluctantly by researchers who had discovered this association with no prior intent or expectation. These and other such discoveries have been invaluable in the development of my own view of Nature. The collaborative work done on MS brain tissue at the Department of Chemistry at the University of Michigan and the Hospital

for Sick Children in Toronto will probably be the most important of all such studies, and it appears that their reporting was complete and flawless.[#224] They were merely looking for changes – any changes – in the chemical composition of MBP between the brains of normal individuals and those with MS. Of the hundreds of changes they could have found, the only ones worth reporting were the ones that happened to show conclusively that formaldehyde was present in higher concentrations in the brains of those who had died with the disease than in the brains of the control group.

The researchers gathered the MS tissue for their study from brain tissue banks in Canada and Colorado. They carefully matched control cadaver samples by age and sex to normal brain samples obtained from violent or sudden cardiac deaths not involving brain injury. The samples gathered from the brain tissue of MS sufferers had well over twice the number of "Crazy Hawks" attached to the arginine that made up its MBP as the control samples. The results were astounding and went beyond our wildest dreams, for not only was the presence of formaldehyde verified by the large increase in arginine methylation,[#200] but also another completely unsuspected outcome was reported. The phosphorylation of the MBP was "dramatically reduced" by over 90%, going from 60 sites in the normal brain MBP to only 4 in the MS samples. It is well known that formaldehyde inhibits phosphorylation at extremely low concentrations.[#113], [#404]

Do you remember the young black girl whose tragic death gave us the critical number for the minimum lethal dose of methanol in humans? Her dreadful last gasp for breath, which froze in vigor for all eternity, was caused by the ability of formaldehyde to destroy the enzymes in her mitochondria that cause the phosphorylation of ADP. By so doing, it robbed her of her ability to distribute life giving energy from her metabolism. No other phenomenon can explain both of these astounding changes to MBP in the MS brain; it can *only* be explained by the direct intervention of formaldehyde. It is well known that the mitochondria in the axons of multiple sclerosis plaque show significant signs of damage and oxidative stress[#481] similar to that presented during methanol poisoning.[#482]

Let me close this with a paraphrased quote from the authors of this revealing article that will help you tie together the last two chapters.

> *Although we do not have an explanation for the state of hypophosphorylation (reduced phosphorylation) at this time, studies in Alzheimer's disease demonstrated that uncoupling of mitochondrial oxidative phosphorylation resulting in decreased amounts of ATP-activation… and other forms of phosphorylation in the Alzheimer's brain.[#224]*

Tragically the good scientists who performed our experiment for us knew nothing of the ways of the Crazy Hawk. Perhaps if they had, their good work would have led to the end of the mystery of the cause of MS and the beginning of the implementation of a real cure. Maybe now it can.

Evidence That Methanol Causes MS

MS Researchers Can Only Agree on One Cause for Multiple Sclerosis: Smoking

Science has been seeking the cause of multiple sclerosis for 150 years. In the early days of the disease it was repeatedly suggested that significant evidence implicated a small toxic molecule[#153],[#185] – perhaps a solvent.[#74],[#140] Methanol is the smallest of solvents and one whose poisoning symptoms are identical to those of MS.

I will not take the time to go over all of the culprits that have been evaluated as a possible cause for multiple sclerosis. Bacteria, viruses, and most disease-causing agents known to man have at one time

or another come under fruitless scrutiny. To this day, some still believe that MS is caused by a disorder of the immune system itself or by a sexually transmitted entity.[#184] It is important to understand that for very good reasons the original researchers in the field, well into the early 20th Century, all held to the belief that MS was caused by a circulating toxin which they could not detect or identify.[#153] Presently, however, the most regarded theory of causation puts full blame on the darling little macrophage.

The good scientists who held that the cause was a circulating toxin were at great disadvantage. Not only was methanol considered safer than ethanol at the time they were doing their laboratory work, but also formaldehyde had not yet been discovered. Even after the discovery of formaldehyde, researchers remained without any animal test subject for studying methanol poisoning and no way to detect the chemical changes made by formaldehyde to protein. Thus, even if it had occurred to them to do so, it was impossible to test the link between MS and methanol in the laboratory environment. In the article that I mentioned in the previous chapter by the neurologist Dr. Fredrick Wolfgram, he asks his collogues to reevaluate looking for a toxic cause for MS. The very last sentence of his conclusion is noteworthy, valid and insightful to this day: "Do we have any evidence as to whether there is or isn't a low-molecular-weight compound in the brains of MS patients that disrupts myelin? The answer is: 'No,' because no one has ever bothered to look." [#153]

It is of the utmost importance that you understand the significance of the fact that during the last hundred and fifty years the search for a cause of MS has found only one culprit. The screening of literally thousands of viruses, bacteria and toxic chemical substances has to this day elucidated only one universally accepted causative agent for MS: cigarette smoking. Most significant of all, this one etiologic agent also just happens to be an important source of methanol. Tobacco smoke has been shown to cause both new cases of MS[#67-70],[#337],[#338],[#339],[#340],[#71] and to induce the relapse of MS in patients who suffered from the disease but were in remission, transforming a relapsing-remitting clinical course into a much more serious and deadly secondary progressive course.[#69]

Understanding where the methanol comes from in cigarettes requires some knowledge of how tobacco leaves are processed into cigarettes. The leaves of the tobacco plant contain large amounts of pectin; and although most scientists are unaware of this, tobacco leaves are left in barns to ferment for weeks.[#61],[#62],[#66] This fermentation releases some of the available methanol from the pectin into the leaf. Wood is then burned to flue-cure the tobacco. The smoke makes direct contact with the tobacco its methanol-laden smoke soaking into the leaf, as the heat turns the tobacco a brilliant golden color before it is sold to be made into cigarettes.[#65] Additional methanol is generated in the smoldering cigarette itself as the leaf is burned. Consequently, methanol is one of the most abundant poisons found in cigarette smoke.[#63] Methanol has been detected in human breath following smoking,[#64] indicating its absorption by the lungs and presence in the blood.

Looking back to the first discovery of MS it would certainly be difficult to link the disease to cigarette smoke, since the manufactured cigarette was not available in Europe until some time after 1860, when they were first being sold commercially in the United States. More important than this is the reality that many of those who develop MS, even today, do not smoke and in many cases never have. It is clear, therefore, that methanol has more sources than this one but smoking should be considered a reliable source of methanol.

The Etiology of Multiple Sclerosis – Follow the Methanol

Every Disease has a Beginning, a First Performance – Just How Old is MS?

When an individual is suffering from the early stages of MS, it could easily be mistaken for many other diseases; however, the ultimate symptoms and unusual gate of the full-blown disease are unique, unmistakable, and so tragic as to still bring a tear even to my trained eye. Nevertheless, medical texts from the Middle Ages contain no descriptions of any disease which would be recognizable as MS.[#230] The fact that this obvious severe symptomology is completely absent from any historical or even biblical literature is telling. Wolfgram even noted during his plea for the search of a small solvent as the cause of MS that "there is a curious lack of reference to the obvious symptoms of MS in the medical literature prior to the middle of the nineteenth century."[#151] This is all proof that MS is indeed a disease of modern civilized man.

It was the discovery of the controlled use of fire that brought man and methanol into potentially dangerous proximity. In chapter two you learned that the smoke produced from the burning of plant material contains methanol. The impact of this methanol was probably minimal, depending on the relationship between various cultures and how they chose to use fire – and more importantly, its smoke. The smokeless flame of a clean burning wood fire contains no detectable methanol, as the blue flame represents the burning off of methanol. Charcoal is wood that has been heat treated in such a way as to remove all methanol and is safely used by many cultures for indoor cooking with no methanol risk. The cultures that developed a taste for food smoked for preservation purposes were probably the first to feel the sting of methanol disease, in the same way that today individuals who smoke cigarettes laden with methanol notoriously suffer from a higher incidence of all DOC.

It was not until the development of the canning of fruits and vegetables in the early eighteen hundreds that methanol was to begin its slow progression toward becoming a daily component of the diet of the civilized world. The daily consumption of methanol would be an important prerequisite for the continuous, unrelenting demylination that consistently exceeds the ability of the Schwann cell to rebuild itself, eventually leading to the complete removal of axon protection that presents as MS.

The History of Man's Methanol Consumption Corresponds to the History of MS

It is the Frenchman Nicolas Appert, the inventor of canning in 1807, who must take responsibility for beginning a process that was to eventually bring methanol into every civilized household throughout the mechanized world. By following the ebb and flow of the canning industry and the consumption trends of canned fruits and vegetables since those early years we can learn a great deal about the evolution of MS.

The first canning factory was fully operational in England by 1813.[#46] Due to the expense involved in the production of the cans themselves, early canning was undertaken primarily with high value meats, which have no pectin content and, therefore, would not have caused methanol accumulation. Canning of fruits and vegetables, however, quickly followed as the wealthy acquired a taste for these products out of season. Over time canning became more prevalent and less expensive,[#46] and the per capita consumption of canned plant material skyrocketed – as did the incidence of multiple sclerosis. As the canning industry flourished so did the practice of incorporating into recipes the "natural," methanol-laden juices from canned fruits and vegetables rather than discarding them.

Canned fruits and vegetables were, for many years, the major food source of methanol in the human diet. In the years that have passed since caning's humble beginnings, MS has transformed from a medical curiosity to one of the most important and common of the DOC. For 150 of the last 200 years

the very best indicator of incidence of the disease in a population would have been the weight of canned fruits and vegetables consumed by the average citizen of the country being surveyed.

The incidence of MS increased slowly after its symptoms were first put to pen in the diary of the grandson of King George III in 1822. We see no official mention of the disease again until ten years later, when its anatomical details are depicted in a book of medical illustrations published in England by Robert Carswell in 1832. This was a time when canned fruits and vegetables were quite expensive and available particularly to the rich. The first officially documented case of multiple sclerosis was reported nearly 35 years later by Jean-Martin Charcot in a lecture in 1868 (discussed below),[#45] although it is generally agreed that the "first identifiable instance of MS" was that of Augustus d'Este, whose symptoms started between 1822 and 1843.[#45] During the 19th Century MS was considered "quite rare," with Charcot reporting fewer than 40 cases during his long career.[#45] Increasing numbers of cases were reported in the late 19th Century.[#45]

During this same period it was discovered that if wood was heated in a large metal container it would produce a smoke that could be condensed into a dark foul smelling liquid called wood alcohol, the original name for methanol. The use of wood alcohol as a solvent for many industrial uses as a replacement for the much more expensive ethanol began about that time and, in fact, Paris was the first large city that would take advantage of the convenience of this liquid fuel, which was easier to transport than wood and could be used as a cooking fuel. It wasn't long before the rich had methanol burning stoves which, under conditions of poor ventilation, were responsible for exposing kitchen help to methanol fumes. It may not have been a coincidence that the first time multiple sclerosis was to be described in detail in a scientific publication was in 1865 by Jean Charcot of France. His first case was his own kitchen servant, whom he had convinced to donate her body, after her death, to his research.

The Early Years of the Multiple Sclerosis Timeline

- No evidence exists to suggest that MS is an ancient malady.
- MS was unknown until some time after Nicolas Appert of France invented canning in 1807.
- The first identifiable instance of MS was not until around 1822 (Augustus d'Este, Grandson of George III).
- MS was first illustrated in drawings done by Carswell in 1831.
- MS was first described in detail, but as a "rare" disease, in 1865 by Jean Charcot of France.
- MS remained uncommon until the 1890s, when the combination of several factors that increased contact with methanol coincided with the disease becoming common, with variable frequency.
- The beginning of the epidemic of MS over "the last 30 years" coincides with the introduction of Aspartame.

An Explosion of Methanol and Disease: The Turn of the 19th Century

The year 1865 was a noteworthy year for methanol in that it marked the end of the Civil War in the United States. It was during this long war that Union troops were often fed canned fruits and vegetables as sustenance while they were in the field. This was their first introduction to such delicacies, and they

were greatly impressed. When the war was finally over, the soldiers brought home with them a ready appetite and market for the fledgling canning industry.

The invention of automatic machines that could make canned fruits and vegetables faster and cheaper than ever before slowly brought canned methanol-contaminated plant products into every home of the United States. It was at this time that the Ball family developed heat resistant jars that could be used by any homemaker to put away fruits and vegetables from their own gardens. Add to this the discovery in 1895 of a method for making the bad-smelling and -tasting wood alcohol look and taste just like ethanol. Subsequently, this new methanol began to be used as a substitute for ethanol in many industrial applications, such as in solvents used to make fast drying glues for the leather and shoe industry, and as an additive in many cough syrups and foods extracts, such as vanilla. It is easy to see a correlation between the steady acceptance of MS as a "common" disease during the first twenty years of the 20th Century and the steady increase in the methanol in the environment and the diet during the very same period of time. Meanwhile, some of the most intensive testing of the safety of methanol was being conducted in laboratories throughout the world, on every animal except man, and they all proved that methanol was safer than any other alcohol.

Up until the early 1880s the cigarette was a specialty item made by hand, sold for a penny apiece, and very much the stepchild of other tobacco products, such as snuff. But in 1883, an automated cigarette rolling machine, developed by James Bonsack, was put into use, revolutionizing cigarette production. The retail price was cut in half, and volume, which in pre-machine days had never exceeded 500 million, leapt to 10 billion by 1910 in the United States alone. There is no doubt that these few years between the nineteenth and twentieth centuries saw a sudden explosion in the incidence of all of the diseases of civilization, of which MS is a prominent member. This was also a time when the consumption of methanol, not merely from cigarette smoking, but from many different sources, increased dramatically in both the diet and the environment.

Innovations at the Turn of the 19th-20th Century that Increased Contact with Methanol

- Use of methanol for heating in Paris kitchens replaced dangerous wood burning stoves.
- Machine-rolled cigarettes increased smoking by a factor of 20 in less than thirty years.
- Use of methanol as a solvent for fast drying glues in the leather and shoe industries rose dramatically in shoe making centers such as Italy and New York.
- Home canning of fruits and vegetables was becoming extremely popular after the American Civil War; the returning troops loved the idea of eating fruits and vegetables in winter.
- Machines made cans extremely inexpensive and plentiful, and canned fruits and vegetables cheaper than fresh and available all year long.
- Tests on all animals proved methanol was safer to drink than ethanol.
- Methods for purifying and removing bad odor and taste of wood alcohol were developed.
- Methanol was allowed and frequently used in place of ethanol in food extracts, many medicines, and body salves, including witch hazel and other body ointments.

A Food Scientist's Nightmare Called Aspartame

The symptoms of multiple sclerosis, chronic and acute methanol toxicity, and aspartame poisoning are in all ways identical because they all reflect slight variations on the same theme. Nothing happens to the human body from the toxic effect of methanol that is not also expressed during the course of MS – not one thing.

Thirty years ago as Professor and Director of the Food Science and Nutrition Laboratory of Arizona State University I was unwittingly thrust into the aspartame debate with a great deal of publicity that accomplished only two good things. First, it brought to the public's attention the fact that a problem of some kind was associated with the new artificial sweetener. Second, it established me as a contact person with whom the public could share their experiences and information.

The calls from consumers started coming to my office at such a rate that for over six months I had to enlist student volunteers to man my telephone when I was in class or working in my laboratory. By far the preponderance of complaints were from women, and these reports consisted mostly of the classic symptoms of early MS.[#58] Some even sent me MRIs depicting images one would expect of MS. I would talk directly to as many as my schedule would allow and encourage all who complained to stop their consumption of aspartame, suggesting that if they felt brave enough, that they wait a month and then resume consumption and share with me the result. Nearly every time, the symptoms disappeared within a week after aspartame consumption was stopped. Those who had the courage to retest all reported a return of symptoms in varying degrees of severity.

On one very notable occasion I was sent a clean MRI from a girl who had stopped diet soda for a year and whose prior MRI showed massive signs of MS. By the way, MRIs taken in the early stages of MS do not show demylination; they show edema in places where the high-fat brain tissue is being displaced by the microglia (the brain's macrophage) and the liquid of inflammation. That was what this girl's initial MRI had shown. At that time, MS was not within my scope of research, so when I had heard enough I made contact with the headquarters of the National Multiple Sclerosis Society in New York City and got the ear of their director of research. I shared with him, with the enthusiasm of a young scholar, why I thought that methanol might be the cause of MS. He asked me to send him what evidence I had, which I did with dispatch. To make a long story short, I never received even the courtesy of a reply to my extensive report and was never allowed to talk to him directly again. Medical researchers hate anecdotal information. The reasons for this are clouded in the evolution of a scientific mindset that specifies data must be generated in the perfect laboratory environment. Unfortunately, this approach does not work well in the absence of suitable animal test subjects in the laboratory.

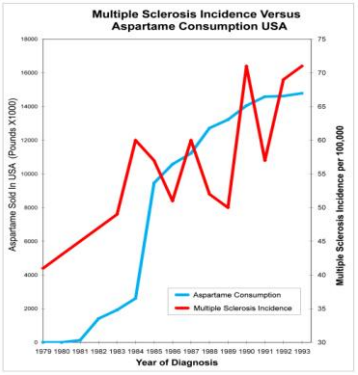
In 1984, I published my first scientific article about the sweetener: *Aspartame; Methanol and the Public Health.*[#1] In the article, I voiced my concern about the new sweetener and the fact that it was increasing the methanol content of the food supply – but made no mention of multiple sclerosis. Publication of the article, along with more publicity over my lawsuit against the Food and Drug Administration (FDA) (which eventually made its way to the US Supreme Court), brought a flood of additional calls and letters to my office from individuals who thought that they had been poisoned by the product. Many of these individuals also had symptoms of multiple sclerosis and some, in fact, were being tested for the disease. A local doctor also visited me to ask whether I thought his patients' consumption of large quantities of diet sodas in the intolerably hot Phoenix summers might be connected to their complaints about symptoms of MS.

Looking into the scientific literature I discovered several articles suggesting that I was not the first to identify methanol as the possible cause of multiple sclerosis. Dr. Hugo Henzi, a Swiss physician who, during his long career, cared for many MS patients, had been struck by the similarities of the

symptoms of methanol poisoning and MS. Henzi's work consisted of a book published in 1980[#5] and three scientific articles[#6],[#8],[#10] that have never been referenced in any MS publication, save my own. I realized that aspartame had provided us with a way to track the public health to the quantitative change of methanol in the food supply. For all intents and purposes, aspartame was methanol, and I had consumption numbers for it. Ending the circumstantial nature of the etiological basis of methanol as the cause of MS would serve as good news for the future of world health, though it would provide little comfort for the innocent populations that had submitted to the Mangle-like enterprise that was the aspartame industry.

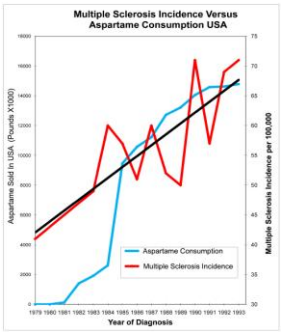
Unfortunately, it would take many years for the MS disease statistics from increasing aspartame consumption to build. When the data became available I was ready. I procured from the Centers for Disease Control its multiple sclerosis incidence data for the United States during the period that consumption of aspartame was building. This was data that could have quickly been made available to the Multiple Sclerosis Society. Figure 4 plots the data in a linear manner just as we did for Alzheimer's disease in the last chapter. The data certainly appears to indicate a marked upward trend in the incidence rate of MS beginning early in the 1980s. In order to get some indication of the strength of this trend I have created another graph of the same data.

Figure 4



In Figure 5 I have had the computer draw a trend line, sometimes called a linear regression line, in heavy black that shows more clearly the direction of the trend of the incidence of multiple scleroses during that important period of time. Just as I had feared, the incidence had gone up 60% during the first decade following the introduction of aspartame.

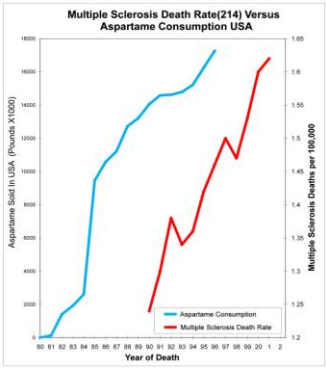
Figure 5



MS usually takes about ten years from first onset of symptoms to reportable diagnosis of the disease.[#86],[#167] This early reporting was evidence, to me, of much worse to follow. How long did it take before the deaths from MS caught up to the consumption of methanol from aspartame? The issue of the death rate from MS has been a very important topic in neurology, particularly with the report of the steady decrease in MS deaths that was observed in the United States during the entire 1970s.[#710] This decline would reflect a continuing decrease in smoking among young adults, as well as a clear change in preference of US consumers for cleverly marketed and delicious frozen fruits and vegetables, which began attracting consumers' attention away from the high methanol canned alternatives, beginning in earnest in the 1960s. The neurological community was taken aback by the clear and apparent reversal of this trend during ten-year study period beginning in 1990, with a dramatic and steady increase in the death rate of MS throughout the period. Their concerns were justified, considering, as the investigators themselves noted, that the treatment of the disease had actually improved since the earlier study.[#214] What they do not mention is that their study was performed just ten years after the introduction of what would be the most important source of methanol in the human diet. This allowed for plenty of time for the added methanol to take its toll on those already suffering from the disease.

I have plotted the data from this study on our usual graph so we can compare the death rate from MS, as we did from Alzheimer's, with the consumption of aspartame. The results are again breathtaking. The mirroring of the two curves is obvious, although expressing a gap of ten years as compared to the 14 years it took Alzheimer's deaths to catch up with increased methanol consumption from aspartame. You will notice that the MS death curve is not quite as smooth as the Alzheimer's curve, but this comes from the much lower number of people who die each year from MS as compared to Alzheimer's, which has a death rate over ten times higher than that of MS. One can, however, have absolutely no doubt as to the strong upward trend throughout the entire study period.

Figure 6



MS: A Disease of Colder Climates and Flush Toilets – Before Aspartame

A number of unusual theories about the origin of Multiple Sclerosis have come directly from the scientific literature. To my mind the silliest of all is the present speculation being acted upon by some of the most influential of the world's pharmaceutical companies: that the innate immune system is its cause. Let me volunteer here, in the spirit of full disclosure, that some of my very closest friends are macrophages, a fact which I admit may cloud my thinking. But in all seriousness when a scientific thesis known as the sanitation hypothesis[#85] comes right out and faults proper sanitation as the royal road to multiple sclerosis and correlates the number of flush toilets in a community to the long term disease incidence, it should give one pause. This is particularly the case when the assumptions on which it is

based appear to be, etiologically, the most reliable proposed to date by the sum total of the neurological research establishment. If this scares you, then you may not have the stomach to be an MS scholar. No matter how misguided, the present medical establishment will consistently reference the most challenged of theories of MS origin – so long as those theories have nothing, whatsoever, to do with a food origin for the disorder. This prejudice against the dietary origin of this disease (or any disease) has always been a serious handicap of the medical community.

By now, if you have read the supporting literature referenced here, the most skeptical among you would have to find at least some merit in the possibility that methanol just might be the cause of MS. I will not waste time reiterating the massive amount of literature on this disease. Other fascinating facts about its distribution across geographical locations, occupations and gender need explaining, along with the changes in all of these categories that have accompanied the revolutionary introduction of the sweetener aspartame. This evidence is circumstantial, but still has considerable merit and is worthy of both your time and consideration.

One thing that you can take away from the latter part of this chapter is important: MS was once considered to be a "rich man's disease" in that its prevalence was positively correlated with the trappings of civilization, including modern sanitation practices.[#85] It is a universal truth that the economically poorest among us – those who cannot afford toilet paper, let alone toilets themselves, those who farm only or forage for all of their sustenance and cannot afford canned fruits or vegetables or cigarettes or diet soda or any other methanol-containing mark of civilization, those poorest of the poor who still make up an alarmingly large percentage of the world's population – are free from autoimmune diseases such as MS.[#168]

A World Awash in MS After Aspartame

Aspartame and cigarettes have two ignoble distinctions that they share. The first is the inordinate amount of money spent by their manufacturers for seriously compromised science,[#249] political influence and extravagant advertising campaigns to assure the public of their safety. The other is both are substantial sources of methanol without any significant redeeming ethanol protection. They stand out in this latter regard, as no other such naked methanol sources could be listed as common consumables.

The epidemic of multiple sclerosis and other autoimmune disease throughout the world over the last 30 years cannot be denied.[#79],[#337-346],[# 80],[# 347-349],[# 81],[#350],[# 82] Multiple sclerosis, once almost unknown in Japan,[#44],[# 85],[#168] has now risen to menace a large portion of the population.[#81],[#350] The lower latitudes and warmer climates, which once "mysteriously" protected people from the full brunt of this tragically debilitating disease,[#83],[#85],[#168] have seen incidence and prevalence of MS climb to as much as four times what they were in the days before summer drinks were sweetened with Aspartame.[#79],[#338],[#340],[#342],[#343],[#344],[#345],[#80],[#347],[#348],[#349] The United States, which has long had a relatively high MS incidence, has seen at least a 50% increase.[#77] Medical journals in Australia[#82] and New Zealand[#90] both report unexplainable increases in their inordinately high[#168] "infection" rates.

I believe that just telling you this information is not going to convince you. My experience has shown that for most people, that is just so many words. In order for you to be convinced of something as important as the methanol theory of MS causation you need to take a little walk in my shoes. What I have done is put together a short slide show, reproduced as Appendix 1, in which I present the literature that I just described above, in an annotated fashion, right out of their respective scientific journals. You will find their reference numbers in parentheses on the lower left corner of each slide. Please take the

time right now to read through this personal slide show made just for you, and I will be waiting right here for you when you return.

As you can see, all the available data does show that the incidence of MS in the United States, and eventually the rest of the developed world, has clearly increased, and that increase appears to have begun with the introduction of the methanol-containing sweetener aspartame in July of 1981.[#194][#586] Spend some time reviewing the maps done by Dr. John F. Kurtzke on slides 10, 11 and 12. John is a physician whose life work has been the epidemiology of MS. He drew these maps at different times during his career. When dealing with such depictions of data, it is a very important consideration that they are all produced by the same competent individual and to the same standard. We will talk more about John's work on the Faroe Islands later. Pay particular attention to the increase in MS incidence in the warmer climates. The growth of the disease into Mexico and South America is easier to see, but have a close look at Europe, where the entire northern Mediterranean basin is now an area of high frequency, and where all of Italy and both Portugal and Greece are now also subject to a high frequency in the disease.[#195] John calls this "diffusion," and that is very descriptive, but to my mind it is all linked directly to the success of the marketing of carbonated diet beverages containing aspartame throughout all of Europe and, interestingly enough, the Netherlands. The second largest aspartame production plant in the world (Japan has the largest) had to be built in Italy in 1985 to supply the tremendous demand for the methanol-containing products by the burgeoning European market.

One last word about the marketing of diet soda – the advertising blitz throughout the South American countries of Brazil and Argentina was noteworthy and appealed quickly to the narcissistic tendencies of those countries' weight-conscious populations, a fact which probably accounts for the increase in their MS statistics. The enhanced importance of this unexpected increased incidence[#77] is that it reversed a clear trend that had been going on for ten years during the entire 1970s[#214] which, looking back, appeared as an apparent consequence of the reduction of smoking with the increased awareness of the health implications of tobacco smoke (which, as mentioned in Chapter 2, is the only known cause of MS). It took about eight years of exposure to aspartame before the death rate from MS began to dramatically reflect the increase in incidence.[#214] The increase in death rate is an eerie mirror image of the graph of the increase in the consumption rate of aspartame and, therefore, of methanol (see Figure 6).

The only logical explanation for an increase in multiple sclerosis in the warmer climates would be an increase in the exposure to the causative agent. If that cause was methanol, then that implies that something consumed in warm climates would have to have had its methanol content increased dramatically. Before the summer of 1981 no diet drinks contained any methanol. Beginning that summer, only powdered drinks had aspartame and, therefore, methanol added to them. These drinks had to be mixed with water and were marketed mostly to women who had a kitchen available to them most of the day. In the fall of 1983, after I lost my bid to prevent aspartame from being approved for uses as an artificial sweetener in carbonated beverages,[#56] the methanol content of diet drinks began an upward trend which was eventually to lead to their exposing consumers to more methanol than canned fruits and vegetables[#1] – or any other food, for that matter. This trend definitely increased the general consumption of methanol in warm climates.

Change in Frequency of MS by Sex: The Methanol Source – Food or Smoke – Makes All the Difference

Modern women bear the brunt of the multiple sclerosis epidemic.[#351],[#352],[#353] Presently three or four women get the disease for every man who does. We have become used to the reality that MS is a women's disease and, as you saw in the slide show, it is becoming more and more of a women's disease as time passes. It appears that each survey that is done and each study that is repeated finds ever greater numbers of women over men who suffer from the malady.[#91] It has not always been this way, and the ratios appear to have changed considerably over time with no apparent reason; however, an explanation is indeed easy if methanol is what causes MS.

Well into the turn of the 20th Century, with the concomitant introduction of methanol into the environment and food supply, the incidence of MS had already grown from that of a rare disease and medical oddity to one of greater prevalence. Before long, it became the subject of much organized medical research and discussion. The group of nine neurologists who made up the New York Neurological Society met to discuss MS in 1902. Recorded in the minutes of that meeting was a general agreement that MS was a rare disease in New York.[#153] MS increased dramatically (over 140%) in Germany and Switzerland between 1906 and 1940.[#153] In 1921 this sea change prompted the American Association for Research in Nervous and Mental Diseases (ARNMD) to theme its annual meeting in New York City *Current Knowledge and Research on Multiple Sclerosis*.[#306] The two day meeting featured presentations on the pathology, epidemiology, etiology and clinical features of the disease and were published the following year. The organization also captured a consensus of the understanding and state of medical research on MS presented by the gathered researchers and clinicians. The conclusions emphasized that MS was among the most common organic disease of the nervous system and that its cause was some "unknown toxin." Most important of all the organization concluded that "males are attacked more often, with a male: female ratio of 3:2" and that the disease occurs more in "skilled manual workers than laborers." It was at this meeting that it was also first noticed that MS is more frequent in the colder climates of both the United States and Europe.[#306]

How methanol gets to the brain makes all the difference. If you were a skilled leather worker employed in one of the many shoe or leather factories in New York City or other major industrial centers of the Unites States during the early 1900s, methanol would be an ingredient in the glue and treatments you used and you would be breathing in these fumes during the work day. This would also be the case if you were a painter or one of any number of other occupations which used cheap methanol as a solvent or to clean surfaces before finishing. If you smoked cigarettes, as many men did during this time period when cigarette smoking was increasing tremendously with the advent of cheap addictive automatically-rolled cigarettes, then you would also be exposed to steady amounts of methanol. These occupational and recreational sources of environmental methanol would supplement the methanol to which both you and your wife were exposed at the evening meal when she opened up a few cans of vegetables and heated them up with the juices for your supper.

The details of how methanol reaches your brain differ, depending on whether you are getting your methanol via your lungs or in your diet. It is this difference that accounts for all of this tweaking of the sexual ratios of MS and some of the other DOC. All is sexually equal when methanol comes from cigarettes or exposure in the workplace air supply or via environmental fumes. Methanol entering the lungs goes directly into the bloodstream equally in both sexes. It is when methanol enters the body via the food supply that things get really interesting, and is what accounts for the difference across sexes in incidence of MS.

A modern study published in the *New England Journal of Medicine*[#94] reports the result of biopsies of the gastric lining of men and women. The astonishing result proved that the concentration of ADH in the gastric lining of men was much higher than that in women. Men, therefore, have the advantage of having large supplies of ADH busily removing methanol from their food at a rate that is four times faster on an equal-body-size basis than women's bodies can remove dietary methanol. [#94] The methanol that men consume, both in the food they eat and the diet soda they drink, is four times more likely to be removed from the blood before it ever reaches their brains. The male brain is spared the onslaught by the Crazy Hawks because the methanol is metabolized to formaldehyde in the gut where it can reap its havoc on a more forgiving organ that has a considerably greater regenerative capacity. Finally, we have an explanation for the disparity between men's and women's reactions to methanol poisoning and the diseases it causes. This may also help to explain why men have more gastrointestinal complaints from both methanol and Aspartame consumption.[#93],[# 99]

The importance of this cannot be overstated. Those presenting their patient reports at the ARNMD meeting were reporting on a preponderance of victims from the occupational exposure to methanol in the workplace and smoking. Certainly, women were suffering from their dietary exposure, but their disadvantage to methanol toxicity was overshadowed by the industrial exposure and smoking to which their male counterparts were much more likely to make contact. During this time the use of methanol in the workplace was being investigated and was receiving considerable negative press, with the result that laws were eventually passed restricting its use and requiring warning labels be placed in plain site wherever it was stored.[#17] As time passed methanol was reduced in the workplace but, unfortunately, not banned completely. The consumption of canned fruits and vegetables literally skyrocketed as canned foods became less expensive and more acceptable. Additionally, home canning of all manner of plant produce increased between and during the two world wars.

In the 1940s, just two decades after the ARNMD meeting, the National Multiple Sclerosis Society found the incidence of the disease to be virtually equally distributed between the sexes. In the 1960s, professor Schumacher found that slightly more women than men were contracting MS.[#306] The '70s reversed completely the ARNMD ratios, with every series reporting women being more frequently affected than men, with the usual ratio being three woman to two men.[#169] By the early 1980s the number of women with the disease rose just a little higher with 1.7 women to each man who had the disease.[#166]

But the real sea change in the incidence of MS in women did not come until after the introduction of a brand new methanol source never before known on our planet or found in our food supply. A can of diet soda sweetened with aspartame has up to four times the amount of methanol as a can of green beans. Worse than that and of much greater significant is that while one would have difficulty consistently consuming six cans of green beans or tomato sauce a day for any length of time, in places like Arizona, Australia or Mexico such consumption of thirst quenching, good-tasting calorie free liquid would be commonplace and has now become customary. The great human health experiment that was begun in 1981 took a great leap forward with the sweetener's allowance as an ingredient in carbonated beverages in 1984, and is going strong right now with aspartame costing much less than sugar to sweeten. The result of all of this was presented at the 59th annual meeting of the American Academy of Neurology in Boston on April 26, 2007.[#351] Dr. Gary Cutter, professor of Biostatistics at the University of Alabama, said women are now four times as likely as men to get multiple sclerosis: "It started at two-to-one and is now four-to-one."

The increase is more pronounced in younger people, with young women especially contracting it at an accelerating rate. "This rapid change suggests that it's not just the disease behaving as usual,"

Cutter said. "It is unfortunate, but it is an opportunity and we can use this information to learn what directions we ought to pursue." Nicholas LaRocca, a VP at the National MS Society, said, "This is an interesting phenomenon, and I'm not sure anyone knows why it's happening."

MS is going through the roof and nobody knows why! As a statistician, Professor Cutter deals with numbers. We thank him for his fine research. But to the victims of this dread plague it's more than "unfortunate," and more than "an opportunity" or "an interesting phenomenon."[#91]

It is interesting to note here that 3:1 is the sexual ratio represented by adverse reactors to aspartame reported by the US Center for Disease Control in its study of serious aspartame health related reactions in 1984.[#58] The Center found three women to every man whose aspartame consumption complaints were serious enough to warrant investigation.[#93] Women's complaints also more frequently involve serious neurological complications that are identical to those of MS. The Centers for Disease Control could not put 2 and 2 together and claimed they could not see a "constellation" of symptoms that would cause them any concern about the new additive. This was after a very unusual two month delay in the release of the report to accommodate an emergency "executive review" of the original document. The executive review has never been released.

Can Methanol Really Cause MS?

The evidence that the formaldehyde produced from methanol causes multiple sclerosis is overwhelming and would be very difficult to refute, particularly if you are debating me, but unfortunately it can be ignored – and has been.

Back in 1990, after the Federation of American Societies for Experimental Biology refused to allow me to give an oral presentation at its annual meeting, I agreed to present a poster session showing a rat model of MS using methanol to damage axons.[#2] I decided I would personally give the presentation, not wanting to put any of my graduate students in a position to take the heat for something that was not merely controversial, but perhaps more importantly, something which could impact the financial stability of many junk food and pharmaceutical goliaths. I had prepared myself to face the toughest questions and was, in fact, anxious to debate the issue as to whether methanol was indeed the cause of multiple sclerosis. To my surprise, however, I was relegated to what turned out to be a very remote corner of a satellite presentation hall with a few dozen foreign graduate students, none of which could converse easily in English – and no one came. It was as if my presentation had a little note attached that warned of the possibility that serious bodily harm would come to any who attended. Our data was submitted to various research publications, in one case several times, but was always refused without consistently remediable comment.

The fact is, sufficient information already exists in the scientific literature to show convincingly that long term exposure to methanol will invariably increase the incidence of MS in human populations, and conversely, to show that the absence of methanol is an important precursor to an MS-free population. Let's take a look at that fascinating data and you can decide for yourself.

MS Can be Found in Some Places, but Cannot be Found in Others

Places Where MS Isn't on the Menu

You can find locations on this planet where MS simply does not exist. This alone should give hope to those who have the disease. The people who occupy these special places are, for all intents and

purposes, made of the same stuff as those who are prone to contracting MS. It has been shown that when they immigrate, at an early enough age, to environments that do foster the disease and they eat the food and drink the water and breath the air of their new environment, they acquire the same risk of the disease as everyone else.[#44] The good news from this is that it isn't you. It is something in what you eat, drink and breathe that is the cause of this disease.

The places where MS is nonexistent or extremely rare can change rather quickly, as we have seen happen over "the last thirty years." This is even more evidence that the disease is not part and parcel of the sunlight or the geography of these sanctuaries, but is strictly part of the changeable environment of food and air. We know that MS is absent from places where the majority of the population lives directly from nature with little or no access to or need for canned fruits and vegetables or diet products containing aspartame. Though the scientific literature points to tropical areas or areas of extreme poverty (where the laughable but statistically valid sanitation theory was born), these MS-free zones have never been strictly limited to the warmer, poorer countries of the world. The exceptions are glaring and can teach us something about MS.

Although central Africa,[#184] Mexico, South and Central America, and good part of India did fit into the MS-free category in 1979 when Dr. Kurtzke produced his excellent MS distribution map, important exceptions to the warm climate rule could still be found. The Inuit or Eskimo people of extreme northern Canada have rarely known MS, and those who still maintain their customary diet remain free of the disease.[#169] Japan is an example of a country which once enjoyed the lowest MS rate of any civilized country in the entire world, and yet it is extremely affluent, without a tropical climate, and has extremely high standards of sanitation.

All we need do is take a look at the diet of the Japanese people and we can lay to rest this whole muddled issue of the epidemiology of MS. The Japanese are sticklers for eating fruits and vegetables fresh and in season. They would not even consider opening a can of green beans or any other such canned plant product. Even though they do have a thriving canned food industry, it is primarily the canned fish and marine mammals that are canned for domestic consumption. The canned fruit and vegetable products make up a small part of the industry and are essentially all produced for export. Furthermore, the Japanese consume a consistent quantity of fermented foods which are abundant in ethanol, another great advantage that protects the population from methanol. In 1960 the prevalence of MS in Japan was a little over 2 per 100,000 population. Compare that to an island at the same latitude, England, at about the same time, where the MS rate was over 50 per 100,000.[#169]

In January of 1985 I received a call from a colleague at MIT asking if I had time for a meeting with an important Japanese scientist and his translators. He was consulting for a major food cooperative in Tokyo, one of the biggest in the country. In Japan, if you have a product that you want the Japanese to consume it must be carried by one of these cooperatives, and they are very strict about what they allow into their stores. Aspartame was at the gate and the Japanese were concerned about what my friend was saying about the possibility that it was causing seizures.[#368] When he showed them my article warning of the dangers of methanol consumption and exposure, they immediately wanted to see me. To make a long story short, we did meet and I did what I could to supply him with numerous articles and evidence that I had uncovered up to that time, but my efforts were fruitless. The fix was in. Ajinomoto, the Japanese chemical giant, had made a deal with Donald Rumsfeld's company and agreed to participate in the mass production of aspartame to supply a market that was expanding faster than anyone's wildest dreams. No one could have stopped the approval of aspartame in Japan with Ajinimoto behind it. To this day the company bears a heavy burden for using extreme political pressure and far worse to protect aspartame from proper scrutiny. Aspartame entered the almost perfect Japanese food supply shortly

thereafter, and as the article that constitutes slide number 14 in the slide show indicates, the prevalence rate of MS in Japan has remarkably quadrupled "in the past 30 years."[#81]

A Group of Islands called Faroe where a Lack of Trees Prevented MS

A group of 17 islands in the North Atlantic has played an inordinately important role in the history and study of multiple sclerosis. A number of medical professionals have made a name for themselves fruitlessly pursuing the cause of MS in those islands – to the point that the island government no longer appears to be interested in proffering its citizenry as guinea pigs for a failed cause.[#195]

The Faroese are a semi-independent part of the Kingdom of Denmark. They are located as far away from Japan as one can get without heading back closer. This small group of islands is situated in the stormiest part of the North Atlantic, midway between Scotland and Iceland. The weather is cloudy and windy throughout the year and the summers are cool and sunless. Daily sunshine in the summer months averages only about four hours a day. If ever a place could be found that would be perfect for disproving the vitamin D deficiency theory of the cause MS, this would be it. The meticulously kept medical history of the native-born residents of this land of over 44,000 in population has been searched back to before the 19th Century began, without finding one single case of MS – until July of 1943. What makes this truly amazing is that this location could be considered to be the geographical heart of MS country. The nearest land masses have MS prevalence rates that are the highest on the planet. Scotland has 62 per 100,000, Iceland has 72, Orkney Island has 108, Shetland Island approaches 300, and so on.[#169] All these countries and islands were originally settled during the same Nordic migrations, thus ensuring populations of similar genetic descent, making the conundrum even more interesting.

The event that occurred that apparently triggered the first case of MS on the Faroe Islands was the occupation of the islands by British military forces for five years during World War II, beginning in April of 1940 and ending in September of 1945. This event is believed to have triggered the "epidemic" of MS on the Faroes. The epidemic began in 1943, after two years of contact with the troops or something they brought with them, and consisted of 21 patients. It is generally believed that the troops brought either an infection or a toxin that was to cause the disease.[#195] A careful study has revealed that those individuals most affected were those who had been in direct contact with the troops and who lived in close proximity to their numerous bases.[#168] It is at this point where the silliness begins, culminating in a scientific piece accusing MS of being a "sexually transmitted infection."[#184] The toxin idea was put to rest early on when it was clear that the disease didn't go away after the troops left and subsequent "epidemics" occurred.

In a review written in 2003 by a man who had spent years on the island looking for the cause of MS, Dr. John Kurtzke explains the phenomenon in an important paragraph which I will quote here exactly.

> *The troops therefore brought something to the Faroes which later resulted in an epidemic of clinical MS. This had to be either an infection or toxin, with either one geographically widespread on the islands from 1941. Now a toxin could not be responsible for later epidemics. Therefore, if there are such (and there are), then there must have been an infection carried by a large proportion of British troops (because of its wide distribution) in asymptomatic fashion (because they were healthy troops). This must be a persistent infection which takes time (here two years) to be transmitted to a naïve populace, the Faroese. As noted, we call this agent the primary multiple sclerosis*

> *affection, which have defined as a specific, but unknown, widespread, persistent infection that will only rarely lead to clinical neurologic MS years after its acquisition.*[#195]

I can't emphasize enough the importance of this paragraph, which exemplifies the extent to which physicians will go to try and show that all disease is caused by bacteria, virus or some other living thing, no matter how far they have to reach. This is the medical mindset and it has cost literally millions of lives to poisonings and nutrient deficiencies throughout the ages. This is not John's fault; this is the fault of his medical training.

The most egregious misstatement in John's paragraph is, "Now a toxin could not be responsible for later epidemics." The troops brought with them items such as canned foods, fruit preserves, marmalades and other rations, along with the ubiquitous cigarette, all of which would be very desirable to the island people, especially during time of war. It is very possible that any of these methanol-containing treats were traded or gifted to the locals, who acquired a taste for, or, in the case of cigarettes, an addiction to them and subsequently became a staple after the troops departed. If this was the case, then of course the "toxin" would have lingered.

To me, the methanol explanation makes much more sense than the strange-new-alien-disease-causing factor, of which the good doctor and all of his many colleagues have never actually been able to find even a trace. Because of the preponderance of physician investigators with preconceived expectations, the diet of the islanders was essentially ignored during the early years when good data could have been gathered from the population while the occupation was still fresh in their memories. In the more than 60 years since MS mysteriously found the Faroes, only one scientist has put any effort into looking at the diet of the islands and has studied seriously the food consumption of the Faroes and its relationship to MS. His work was not published until 1989 and is actually a review of cook books and importation records of foods to the islands before the occupation and shortly afterward. He stresses that until the war, the traditional diet was indigenous and based primarily on fish, as well as mutton, whale, wild birds, and potatoes. This very limited diet is still maintained to some extent, particularly in the smaller villages, which are still free from MS.[#82] This scientist does confirm that after the war, a "rapid" evolution introduced many new food products into the diet, though unfortunately he is not specific about which foods were introduced.[#82]

The truly fascinating angle that the author, Klaus Lauer, takes on the Faroe diet may, in fact, explain exactly why they had been completely exempt from MS all throughout its dramatic increase in the rest of the civilized world and, in particular, their close neighbors during the early 1900s: the complete absence of smoking. By that, I do not mean cigarette smoking, but rather the smoking of fish and other meat products. Unlike the traditions of the islands' neighbors, smoking of meats is not practiced as a traditional method of food preservation in the Faroes.[#82] They preserve fish and other meats by air drying, as is done with cod in many cultures. Their cook books describe in detail salting and wind drying, but do not mention smoking. Outside the Faroes, that area of the world is famous for its smoked foods. The Scots and Icelanders, for instance, have perfected the art of preservation by smoking and it is not unusual for them to offer smoked fish along with their jams and preserves with every meal.[#487] The Shetland Islands have a special process called "reesting" in which they smoke their food slowly over a peat fire.[#82] Unfortunately, the smoke from burning peat contains three times the methanol level of wood smoke.[#180] This could help explain why the Shetland Islands have the dubious honor of having the highest incidence of MS in the world, approaching 300 per 100,000 and climbing.[#169] But what these other cultures have that Faroes do not is trees and/or peat bogs to provide the heat, smoke and methanol to make the food smoking process work. The North Atlantic archipelago

is known for its treelessness. Climatic and geographic conditions and centuries of sheep-breeding have left the Faroe islands all but treeless.

After publishing the Faroe diet article, Lauer published an article attempting to link the disparate incidence of MS in various provinces of France and Switzerland to the use of wood smoke to preserve meat.[#69] He made a convincing story of the high incidence of MS in populations that would customararily smoke meats as compared to to those who air dried their meats. Though he was not aware of the possible impact of methanol at the time, it is interesting and important that he put to pen a clear association between MS and diet that needed exposure. His writings have been largely ignored by the medical community.

Methanol consumption was overlooked as a factor in all the studies done on the Faroes. The fact is that even after the work of Henzi, methanol has never been seriously explored as the possible cause of MS. The Scandinavian countries and portions of the Slavic nations have some of the highest incidence of MS of any population in the world.[#354],[#168] In these countries consumption of both commercial- and home-canned fruits and vegetables is high, as is consumption of smoked food as describe above. Moreover, methanol can be found in traditional liquors made from rotted fruit culled off the ground during harvest and rotted in barrels for months. Some of these liquors have a high enough methanol content to exclude them from international commerce. It may not be a coincidence that the highest incidence of MS is found in cultures which had the potential for very high methanol consumption even before the advent of aspartame.

The Little Village Of Wellington: The World's Highest MS Incidence Rate

It seems like many significant things in my life happen in towns called Wellington. I met MS in Wellington, Colorado. I discovered the very rare dark side of the little country of New Zealand in its capital of Wellington. And as you soon will probably agree, the most interesting Wellington of all was one where I have never been and have no desire to ever visit, the Village of Wellington, Ohio. It isn't that I have anything against Wellington, Ohio. It's just that it appears that an old enemy of mine, multiple sclerosis, chose to take up residency there for a time.

The real mystery of this story is how I discovered this little burg in the first place. With a population of 4600 people – one tenth that of the Faroes, and with absolutely nothing of great importance ever having happened within 100 miles of the place since the last Ice Age, we have the Internet to thank for bringing us together. The juxtaposition of the word's methanol and multiple sclerosis plugged into the Environmental Protection Agency's website brought up something I had never seen before: a Health Consultation. It seems that it is type of investigation done by a group that serves under the US Centers for Disease Control called the Agency for Toxic Substances and Disease Registry. The study was conducted in 2005, during the George Bush administration.

I knew the chances of the work being slanted toward industry and away from the public health were strong. I went to the report's conclusions first, just as one would look ahead to see whether a cheap novel had a good ending before deciding whether to waste much time reading it. As I suspected, it was clear that the anonymous researchers who had conducted the study (none of their names were attached to the document) were themselves unwilling to make a difference. The conclusion began with "no significant contaminants of concern were identified in human exposure pathways." Reading further, however, there appeared a bit of a surprise that made my heart skip a beat or two: "The causes of MS, the primary health concern in this community, are unknown; the disease is believed to be caused by a

combination of genetic and environmental factors." Then this highly unusual statement: "This evaluation did not suggest any additional hypotheses for the cause of MS."[#578] Obviously, this little town had suffered a serious outbreak of MS, but the population was very small and it was located in the middle of farm country, surrounded by rolling hills of grass. The researchers could only point to a mysterious environmental factor, but could not identify the cause of the outbreak.

The other promised word I had entered into my search criteria, methanol, was nowhere to be found in the conclusion. This often happens with modern search engines finding every use of the word. I am often disappointed to find "methanol" being something less than important in the documents found by such searches, with the only use of the word in some minor subscript. Throwing my concern for the forests aside I quickly printed out the entire document and devoured and annotated it in what seemed like no time at all. The document, hidden in the archives of a government agency under siege during the worst of years for public safety issues, contained information that was enlightening and extremely helpful and confirmed my fears of what could/would happen to a population that was consistently dosed with small amounts of methanol over a long period of time. Unlike aspartame, this methanol came from the air.

The report was oddly written as if by someone who was new at such things. It did not disclose exactly why the federal government was called in to investigate this outbreak of MS until halfway through the report itself. Under the topic of "Community Health Concerns" I found that the Ohio Department of Health had, in 1998, identified enough cases of MS in this little community to give Wellington an MS prevalence rate of 600 cases per 100,000. To put this into perspective, this gave Wellington the dubious honor of having the highest MS rate ever recorded in the entire world, worse even than that of the Shetland Islands, which had not yet reached 300. For twenty five people in this little community to have been diagnosed in a period of less than ten years with multiple sclerosis was bad enough, but it did not end there. The incidence of other suspected autoimmune diseases and cancer had also risen to the point of great concern to local authorities. The other disease that most interested me was lupus, another methanol disease which we will discuss in the next chapter.

By the time I got to this earth-shattering bit of information, which was treated with the greatest of nonchalance, I had already learned that the EPA did not arrive at Wellington until September 4-5, 2003 – a year after what it turns out was the offending foundry had fired its workers and closed its doors for the last time, thereby limiting the chances of gleaning any valuable information from the fresh memories of its disgruntled former employees. It seemed, in fact, that this group of Washington bureaucrats came to this village to allay fears and participate in a cover-up of a major cluster of MS that could have done for them precisely what their conclusion said it did not do: "suggest an additional hypothesis for the cause of MS" – Methanol. For you see, the *only* chemical that this group's investigation found that was being flagrantly dumped into the environment and that could have caused this tremendous increase in the MS rate of this little community was methanol.

The report contains a review of the literature in which the researchers admit knowing "a weak association between solvent exposure and the development of MS may exist," but the statement is couched in such a way as to make it appear that the anonymous authors are essentially attempting to cover their own rear ends, since at the same time they also recognize the fact that methanol can cause "damage to the central nervous system" without any reflection whatsoever on the situation at hand. In this age of computerized searches, the literature review by all rights should have mentioned the works by Henzi that clearly and specifically implicated methanol as a causative agent for MS. Obviously, they chose not to mention this, for any competent researcher would have stumbled across Henzi's work and included it. But then how would they not then be required to explain away the possible association

between the methanol exposure in the little town and the resulting MS epidemic. I have always wondered whether the individuals that write such reports are actually clones, created in a mysterious laboratory some place where they are kept dormant and benign until a truly misguided politician comes along who wants a scientific-sounding paper generated that will hide the truth and forward a wrong-sided agenda. Certainly Hitler had no difficulty rounding up as many of these pseudo-researchers as he needed to get his dirty little jobs done. They are also at work at the EPA and, rare as they may be in New Zealand, even its Wellington has its share of them. What a terrible shame.

It would be far too easy to get carried away with politics at this point; suffice it to say, it is clear to me that if this Health Consultation is anything it is a political piece. The long and short of it is that methanol is at the very heart of the health problems that confront the people of this little Ohio community. This methanol came from Sterling Foundry, which was located on the very southwest edge of the city limits of Wellington, just a few hundred feet from the drinking water plant and reservoir for the town. In 1990, new owners bought the foundry out of bankruptcy and it appears that the trouble began at around that time. The prevailing wind in this area of Ohio blows out of the southwest and it wasn't long before townspeople began complaining of "odors" emanating from the plant.

The State of Ohio Environmental Protection Agency investigated the facility in 1995 and discovered the plant was operating without any effective air pollution control equipment, all of which had stopped working during the time the plant was in bankruptcy and out of commission. The plant had "unregulated on-site storage facilities" for large amounts of methanol, with between 20 and 40 tons of it stored on site, outside the plant proper. Nothing anywhere in the report indicates exactly what the intended use of the methanol was, although usually in such facilities it is used to clean freshly cast metal work as it comes out of hot molds. This process usually takes place in outdoor areas to prevent the buildup of dangerous vapors. We can only guess at this point why the foundry workers were never asked by researchers how the methanol was used or why such enormous amounts of methanol were kept on the property at any one time.

After the first Ohio EPA investigation in 1995 the plant was required to keep records of the amount of methanol it kept in storage and to "estimate" the evaporative losses from these storage facilities. The self-reported Toxic Release Inventory from the Sterling Foundry facility in the last three years before it closed (the only inventory available) indicated an annual leakage from storage to the air on-site as 5000 pounds a year. The Ohio EPA admitted that this was evidently not regulated or monitored and never itself did any testing of the atmosphere or verification of any kind. The researchers mentioned this methanol leakage and restated that "methanol can be toxic to humans, targeting the central nervous system." The same paragraph, without scientific reference, states that methanol released into the atmosphere is "rapidly oxidized to carbon dioxide," thus justifying the next statement that the data does not necessarily mean that off-site residents were exposed to methanol at concentrations that would amount to any health concern. The first statement is a lie and the second is silly.

Methanol is very stable in the air and its oxidation to carbon dioxide requires it be burned or the presence of a very special bacterium not found in the atmosphere.[#723] I believe the smells from the foundry that for years had instigated complaints from the townspeople located downwind from it were in part methanol that was leaking from those storage facilities and being used for some unknown purpose outside the facility. The very sad thing is that we could have had a very good idea of just how much methanol was going into the air of Wellington. The foundry was buying its methanol from somewhere and the companies that sell such dangerous substances keep good records of those sales. Why didn't the CDC "investigators" track down Starling's methanol source and ask the right questions? The only possible answer is that they just didn't want to know. Sadly, the only thing we do know is that it was

enough methanol to raise the rate of MS of the citizens of Wellington to a level higher than anywhere else in the world.

Industrial Exposure to Methanol – Jobs that can Last for an Eternity

Organic Solvents and the Risk of MS: Only One Solvent at Cause

According to a review article published in 1996, the theory that prolonged contact with organic solvents can cause MS was first proposed in 1982.[#74] This theory is correct and this book backs it up fully. Only one problem remains, and it brings us to a very important issue. Only one organic solvent, methanol, is the cause of MS. The other few thousand organic solvents have nothing to do with MS. More important of all, only one other organic solvent, ethanol, can, if properly applied, stop or even cure MS, by preventing methanol from transforming into formaldehyde and, therefore, preventing symptoms. This means if you wanted to prove the theory, disprove the theory or reverse the theory you know how that can be done by carefully picking you organic solvent.

It was clear in the early 1900s that more workers in the leather and the shoe-making industries developed multiple sclerosis than in other industries.[#59] The major reason that MS was considered a disease of men in the early 1900s was because methanol was used freely in industry, particularly in the manufacture of glues and many other sorts of liquids used by businesses that hired only men. Methanol was common in industry for two important reasons. First of all, methanol is an outstanding solvent, better than ethanol for use in all manner of glues, paints and varnishes. Second, it has a lower boiling point than ethanol and evaporates off much more quickly, drying paint or hardening the glue faster and reducing labor costs. Methanol is also an outstanding and very inexpensive solvent for cleaning and removing stains and oily contaminants.

I mentioned the association between exposure to solvents in general and incidents of multiple sclerosis in the previous section. The scientific literature is peppered with articles on the pro and con side of this issue, and now that you know more about methanol than the average scientist you can see that the premise is correct, but only for methanol and no other solvent. Knowing that all solvents are not equal when it comes to causing MS you can wade through the industrial solvent MS literature and cull the useless articles from the others.

The removal of methanol from industrial glues has been a very slow process since the advantages are so great. The shoe industry was the last to convert to other solvents, although records are difficult to obtain on such industrial secrets as the exact makeup of this type of product. Unfortunately, the MS rate of a particular factory is the most reliable indication as to whether methanol is being used as a solvent or not. The first published study addressing the question of MS and solvent exposure found that in the late 1970s, the incidence of MS among workers in the shoe and leather industry in Florence, Italy was five times higher than that of the general population.[#245] Another Italian prevalence study done in the mid 1980s reported a prevalence of MS among shoe workers of over three times above the general Italian population, a number that was still extremely high but probably reflecting a reduction in methanol use by the leather industry around the world.

The fact of the matter is that in modern times the danger associated with methanol contact with industrial personnel and the large number of deaths from industrial accidents caused by improper use of methanol has limited the number of occupations and industries where workers make contact with this dangerous organic solvent. Painters were often exposed to methanol in the old days, but again methanol has slowly been removed from most paints due to the danger of both its manufacture and its ultimate

use. Its presence in paint is bound to account for the twofold increase in risk of MS in painters over construction and food processing workers found during a study of 50,000 workers followed over a 16 year period in Norway beginning in 1970.[#480]

Industrial Contact with Methanol

These days warning signs abound and the use of methanol in all industries and even science laboratories is accompanied with stern visible and documented warnings of blindness, irreversible neurological damage and birth defects associated with methanol. In fact, a definite disconnect seems to prevail. The only individuals who seem not to be take methanol seriously are the bureaucrats working at the Food Safety Authority of New Zealand or the Food and Drug Administration of the United States or even the European Food Safety Authority, where I am consistently and repeatedly told that "there is nothing to worry about because methanol is a food." Of course, I tell myself that these dim-witted incompetents are all mere toadies for the powerful food lobbies that proffer them with god-knows-what recompense, either real or implied. It is still painfully insufficient consolation to me, especially when I have no recourse. At any rate, the workplaces of the modern world treat methanol with great respect and it is difficult today to find any profession in which individuals daily and consistently make contact with methanol. Two exceptions, however, help prove my point.

Those Who Work With Hot Wood

The paper and wood industry have a great deal to fear if methanol is banned completely from the workplace,[#553] as it must eventually be, because it can actually be produced as a byproduct of these industries' general operations. Methanol is, after all, just another name for wood alcohol. The heat required to release methanol from wood as a gas into the environment is not as great as one would suspect. No flame is required, and the temperatures at which paper is made from scrap wood fall well within the methanol production range. Even the heat produced from the friction of the blade of a chainsaw can produce methanol when trees are felled. The paper industry does not like to talk publically about methanol, although we know that all 300 paper mills throughout the world produce what they call internally a "methanol-water waste stream," and each must find a way to dispose of it. Some do much better than others. This stream, which comes off the processing wood pulp, is often exposed to the atmosphere, where it releases both methanol and a group of sulfur-containing compounds called mercaptans that smell very bad (like some paper mills). Chances are very good if you smell a paper mill, you are also smelling some methanol.

Some mills incinerate the methanol streams to destroy the methanol, while others turn the methanol into formaldehyde for sale to other industries.[#70] Methanol produced by paper mills and wood processing plants is not carefully regulated by most governmental agencies, due most likely to the political power of organizations such as the American Forest & Paper Association, which collects and uses large sums of money from its members for the purpose of lobbying, much like the methanol and formaldehyde lobbying groups we discussed earlier. Keep in mind that any process that heats wood to high temperatures can and does produce methanol. With the proper precautions, plants can be and have been designed to keep this methanol from causing harm to their employees or the nearby community. Sweden sets a good example of how paper can be made safely.

In a fascinating epidemiological study a number of industrial activities in four European countries were randomly screened to determine if any of them had higher risk than any other for their

workers developing multiple sclerosis. Paper manufacturing was by far statistically the most closely associated with MS in all countries tested except for Sweden. Paper manufacturing was followed by wood processing in Norway and Switzerland and leather processing in those same two countries. The author points out that the "association between MS and paper manufacturing is all the more surprising, since this industry played only a minor role in the respective countries."[#13] This study is in agreement with the high correlation of MS with the felling of coniferous trees in Norway and earlier observations of a high MS rate associated with wood processing occupations in Europe.[#334] This data would mean little if other industries showed a high correlation to MS. The fact is that multiple sclerosis is found more frequently in industries where methanol is used or made as a byproduct that can contaminate the working environment.

It would be ideal to find an occupation that would not be considered one in which practitioners would normally make contact with any toxic chemicals except for methanol. Then we could look at health records and see just what happens to these people in the course of their working lives. It sounds like a scientists daydream - and yet we have done just that in the next section.

Teachers' Paradigm

Methanol is a powerful killer of humans. Our tolerance to acute dosages is low, much lower that the toxicology literature reflects. This discrepancy is due primarily to the accompanying ethanol that is almost always co-administered with the methanol, either as a portion of the lethal concoction or as an attempt at life saving that goes hand-in-hand with dialysis. A patient who survives the first week of an acute bout of methanol poisoning may not always enjoy a complete recovery but the patient usually lives. We don't know experimentally what chronic administration of methanol over a long period does because it can only be tested on humans. To my mind the perfect human testing has already been done by the double blind introduction of aspartame to the world's food supply. I have shown you where that testing has gone, but due to the magnitude of my claims I felt it important to find another human study group with another approach to the administration of methanol – one that allows for consistent administration of methanol on as regular a basis as would be feasible with a human test population while at the same time limiting the contact these individuals might have with other dangerous industrial chemicals. Where was I to find such an idyllic study group who would stand still and expose themselves willingly for their entire careers to this dangerous poison? Little did I imagine that this perfect experimental study group would turn out to be one of which I was a member for a number of years.

A Cold Awakening in Riverton

I woke up with a start in the middle of a wintry New Zealand night late one July with something on my mind. I ran to my office without properly protecting myself from the cold, turned on my computer, which seemed to take forever to find the Internet, and, teeth chattering, I inputted the key word, "Ditto."

I had started teaching right after graduating from the New Mexico Institute of Mining and Technology. My high school chemistry and physics teacher and mentor, Oscar Weisberg, was not about to allow his protégé to get killed in Viet Nam. Oscar had heard that I was back in New Jersey staying at my parents' summer home at the Shore. He called, and after we had a good catch-up he summoned me to his laboratory. Although Oscar was essentially a high school teacher, he was also a brilliant scientist who was exempted from service during the Second World War to work on the atomic bomb. The short story is that he would be taking a sabbatical and I was the only one he was willing to trust to teach his

flock while he was gone. I told him I was a scientist and had no intention of teaching, having taken not one class that would count toward a teaching certificate. All he had to say was, "Well, you are going to let me down then."

Somehow he managed to obtain an emergency certification for me that would allow me to teach in my old high school while he was gone. After a little initial awkwardness teaching a summer school class in chemistry for practice, I found that I loved the experience enough to begin taking night school classes that would allow me to continue teaching for a few years. Eventually, I was lucky enough to get a fantastic job at one of the best teaching establishments in the world, a private school called Colorado Academy just outside of Denver. In all, I taught young kids in secondary schools for five years before I went on to earn my Doctorate and return to University.

So why am I telling you this? Part and parcel of learning to teach, particularly in secondary schools with large classes, was learning and gaining proficiency in the care and use of a device called a Ditto machine. The dream I had many years later on that chilly Riverton morning was one of me back in New Jersey working in the faculty lounge at my high school. I was retrieving a rectangular one gallon metal can of liquid from a closet stacked high with such cans. In my dream I was looking at the label and, sure enough, it was Ditto fluid. I turned it over to view the contents declaration. What woke me up was the horror of seeing only a brightly colored cautionary skull and crossbones and noticing that one of the eyes in the skull was looking back at me with a slow, menacing wink. Fear quickly turned into curiosity and my search began.

A Ditto machine (also referred to as a spirit duplicator) is a low-volume, very inexpensive printing method used by primarily by schools. The term "spirit duplicator" comes from the alternative term for alcohol, which is "spirits." Methyl alcohol was the only component of the liquid in that can. It is used as a solvent to transfer inks from a template typed by the teacher onto the blank paper that would become the handout for the students in class that day. The spirit duplicator was invented in 1923 by Wilhelm Ritzerfeld. The best-known manufacturer in the United States was Ditto Corporation of Illinois, hence that name. My search turned up some amazing information about this ubiquitous secondary teaching tool. By ubiquitous I mean that the duplicators were everywhere, but particularly in the teachers' lounges of every school where I ever taught or visited. Our school had four of them, and they were always kept in tip top shape. While other teachers were having a coffee or chatting or preparing for their next classes, someone was usually busy in the background inserting their templates and running off purple copies.

I taught secondary science classes that required numerous handouts, as do most secondary school classes. I remember doing some presentations in the primary school on occasion and having to wait in line at their one and only machine. It sounds silly, I know, but this bit of information will turn out to be important very soon. Even the advent of the Xerox machine didn't change this; because of the inexpensiveness of the Ditto machine copies and of the machines themselves, teachers even today are encouraged to use the Ditto to make the copies for their classes. The copies that come from the Ditto machine have a distinctive light blue print and a strong methyl alcohol smell that some find appealing. I can't tell you how many times I would watch as my colleagues would pick up a ream of Ditto copies and fan them in front of their face and smell to see if they were dry enough not to smear as they handed them to their class that day. Often the pages coming off the machine would be saturated with methanol and would have to sit on a desk in the faculty lounge to "dry." The reason that ethanol would not work as a substitution for methanol in these machines was that it would take too long to evaporate and was not a satisfactory solvent for the ink that was impregnated into the original template.

All this methanol – but what was it doing to these poor teachers? I would like you to see what I saw as I progressed through my literature review. The slide show in Appendix 2 is an abbreviation of what I found and may mean more to many of you than my discussion. Please take the time right now to read through it.

Safety Studies – but Nothing Changes!

My computer search of Ditto machines brought up some fascinating information and good evidence that over the years, concerns have been raised about the methanol from these machines and just how dangerous exposure to it might be. These machines have been used in schools since the 1930s. The first investigation was by the Connecticut State Department of Health, which found that the average Ditto machine, without a great deal of use, could easily evaporate up to a gallon of methanol a day, all of which was subsequently vaporized and went into the atmosphere of the room in which the machine was located. Researchers measured methanol concentrations of well over 200 parts per million in the air of the rooms that contained heavily used Ditto machines.

Researchers warned of this in the 1948 issue of the *Industrial Hygiene Newsletter* and again in 1954 in an article entitled, "Exposure To Methanol from Spirit Duplicating Machines," which was published in *The Industrial Hygienist*.[#411] This article was specific and stated that "this type of duplicating machine is in common use in schools and business offices." The article further stressed that all of the duplicating fluids contain methanol, with some being made up of 100% of this solvent and rarely any containing less than 40%. One gallon of the fluid can produce between 8,000 to 12,000 copies with wide variability. The paper came out of the machine wet with methanol and, as the copies were handled or riffled, large volumes of methanol vapor rapidly evaporated into the room air and the lungs of the operator, who was often exposed to concentrations of over 1000 parts per million of the poisonous substance. They warned that they tested the air in one very large 5000 square foot office with a 10 foot high ceiling, even leaving the door in the room open during the run. This decreased the methanol concentrations, but even so, they remained above the allowable limit. In the end, researchers recommended that all such machines be hooded and vented out of the building.[#411] I taught in several schools between 1967- 71 (twenty years later) and visited many others, and I can attest to the fact that I never saw a single vented Ditto machine until in 1997, when ASU began a program to put vents above all spirit duplicator machines in the University, including the one in our department.

This was not the end of the warnings. Again in 1980 and 1981the US National Institute for Occupational Safety and Health (NIOSH) was called in to investigate two major cases involving numerous complaints. The first was in the Everett School District in Everett, Washington, where operators of spirit duplicators throughout the very large public school district were reporting MS-like symptoms and blaming it on the duplicators. Then they were called in to do another extensive Health Hazard Evaluation Report for very similar complaints, this time to the main campus of the University of Washington.[#208] These cases were the most extensive studies done to date on the exposure risks of individuals operating Ditto machines, researchers verified that within just 15 minutes of operation, 75% of the duplicators they tested produced concentrations of methanol that were well over 1100 parts per million. They also noted that 45% of the operators experienced symptoms such as blurred vision, headache, nausea, dizziness and eye irritation, all of which are consistent with the toxic effect of methyl alcohol. The conclusion was that "a health hazard due to excessive exposure to methyl existed in the operation of spirit duplicators."

Teachers, it seems, had been exposed to methanol for their entire careers. This has been going on certifiably since at least 1948. But when did it stop? On March 21, 1995 the nationwide newspaper, *USA Today* published an article entitled "Ditto Sheets in Schools Hazardous."[#209] The piece was written as a result of the release of a study done by the American Federation of Teachers at the request of numerous member teachers complaining of health problems that they had linked to their use of Ditto machines. The article starts, "Those smelly, purple-ink sheets that teachers regularly crank out on low-tech ditto machines may be hazardous to everyone's health." The study revealed that a spot check of schools from all over the country, including the most populous states of California, Pennsylvania and Michigan, found that the Ditto machines are still in frequent use. Most important, the study showed that exposure to methanol in schools can be much higher than levels allowed in workplaces by the Occupational Safety and Health Administration. The article goes on to reveal that methanol has a "high odor threshold," and by the time you smell it, "you've already been overexposed."

Teachers Become the Perfect Test Animal for Methanol Poisoning

We can document that at least in the United States, most teachers have been exposed to methanol on a workday basis for their entire careers. We can't say that they received equal doses as we could if we were experimenting with rats in a controlled laboratory environment, but we can make up for that by having a large population of exposed teachers over a very long period of time. We know a little more about our subjects as well; we know that those who teach on the secondary level have been consistently exposed to more methanol than the average primary school teacher, who has need for fewer handouts. We know that some equanimity may be found among these two classes of teachers because even though some teachers have used more handouts than others, the machines have usually traditionally been located in the teachers' lounges, thus exposing even those teachers who didn't do many handouts.

The Diseases of Teaching

While still in secure self exile in New Zealand I continued my literature review, looking to see if teachers' health was any different from that of the general public. The results of my search shook me seriously to my very core. It was while still in lovely Riverton among my many Kiwi friends that I really did begin to wonder whether some sort of conspiracy could have been involved in the fact that science seemed to have entirely missed the connection between methanol exposure and MS. The title of this book, "While Science Sleeps," was thus born, as was my determination to circumvent the establishment and bring all of my findings directly to the public – the people who needed it most.

The first article I remember reading was a review that described a French survey which had the perfect title: *Do teachers have more health problems*?[#215] Sure enough, the author admitted that the literature comparing diseases between teachers and the general public was rare, but he pointed me in the right direction to other articles that have shown an increased incidence in breast and thyroid cancers in the teaching population, two methanol-sensitive organs that we will talk about in the cancer chapter. The "Eureka!" moment, however, came when I read this phrase: "and surprising enough, there is an association between school teaching and mortality from autoimmune diseases." I just wanted to stop right there, go no further, and avoid the risk that the reference was a weak one or poorly done, or worse, that it had a strange sample selection. I think that I floated on the little bit of good news for at least an hour before I built up the courage to look up the reference number in the bibliography.

When I did finally look it up I was both relieved and pleasantly surprised. The article had been published in one of the few journals in the area that I regarded as both untouchable and prestigious; I would trust the results of any article its editors selected to publish. *The Journal of Rheumatology* published only the best work, and now I couldn't wait to get the article into my shaking hands. When I did, I was blown away by it, and I think you will be, as well.

Excess Autoimmune Disease Mortality Among School Teachers

The name of the article was "Excess Autoimmune Disease Mortality Among School Teachers."[#210] The work was faultless and the data was conservatively compiled from the death certificates of all deaths in the US for the ten year period between 1985-95. School teacher deaths were compared to those of persons in other professional occupations. The conclusion: "Our results substantiate excess mortality from autoimmune diseases among teachers and suggest that, relatively early in their careers, teachers experience an occupational exposure that increases the risk of autoimmune diseases." They found that "excess mortality was significantly greater in secondary teachers than elementary teachers." Most astonishing of all, "the greatest relative excess in autoimmune disease mortality among teachers occurred for multiple sclerosis."

In fact, the rate of multiple sclerosis among secondary school teachers was almost twice that of their professional counterparts. "The autoimmunity disease mortality was most strongly elevated among secondary school teachers. In particular, significant excess mortality from multiple sclerosis and lupus occurred in the 35-44 age interval of secondary school teachers." We will discuss lupus and the other autoimmune diseases that this study found associated with secondary school teaching in the next chapter and the cancers of methanol in the chapters after that. The one other statistically significant statement that can be taken directly from this study was that "among white males teaching was associated with excess mortality from multiple sclerosis."

In explaining the rationale behind doing this study the investigators said that the National Institute for Occupational Safety and Health had produced a survey of occupational mortality that unexpectedly pointed to elevated autoimmune disease mortality among school teachers during the 1984-88 period. "Our results show that significant elevated mortality also occurred in the subsequent 1989-95 period."

MS Treatment – Pharmaceutical Placeboes or Perhaps Worse

All modern treatments for MS and all treatments that are in "the pipeline" are dangerous and should be avoided at all cost. MS has no known cure, and after reading about all of the many modern treatments,[#615] I can only conclude from the evidence provided that over the last 50 years the only one that showed statistically valid improvement in double-blind studies, albeit for a relatively short period of time, is plasmapheresis.[#186] Plasmapheresis involves removing the liquid portion (plasma) of a patient's blood, then returning the red and white blood cells to the patient without the plasma. The reason this procedure has a positive effect may very well be something other than the official reason it is performed; you see, the process could be expected to remove much of the methanol from the bloodstream, reducing its concentration substantially in the tissues. Transfusions seem also to have similar effect.[#43] I cannot recommend these two treatments because it would be far easier to just avoid methanol.

Conclusion and Review

You can see for yourself now that the daily administration of methanol to the human organism does not go unnoticed by the immune system. The evidence is simply far too overwhelming for the pharmaceutical industries to credibly justify ignoring it any longer. As a scientist I can do little more than present a coherent molecular theory, and then prove the hypothesis using three paradigms with two distinct methods of methanol administration. Viewing methanol toxicity as the etiologic cause of MS answers all of the nagging questions and unexplained anomalies that have stalled the search for the cause of this disease. I realize that absolutely nothing can convince the pharmaceutical giants, who are now heavily invested in developing their own useless palliatives for MS, to give them up and rally around the methanol hypothesis. In the end, however, I believe that the truth will win out. Henry Miller prophesied over 50 years ago:

> *It is possible that the cause of multiple sclerosis lies buried somewhere in these lengthy protocols waiting to be found by someone ingenious enough to unearth it.*[#306]

Review

1. MS is a disease that begins around brain blood vessels, adjacent to the exact locations where methanol converts to formaldehyde, very much like Alzheimer's Disease.
2. MS was first discovered long before formaldehyde, making the determination of its cause impossible.
3. The vast majority of early researchers believed that the cause of MS was a "toxic substance" that forms in and is distributed via the blood vessels of the brain. "Whatever is being produced within the vessel walls is the cause of the disease."
4. All symptoms of MS can be found during the course of methanol poisoning if the patient lives long enough.
5. Myelin Basic Protein (MBP) is the protein of the myelin sheath that is removed during MS plaque development. MBP contains a high percentage of arginine, which acts as a trap for formaldehyde. The MBP of MS patients has been shown to have reacted with formaldehyde and cause a marked increase of the methylation of its arginine.
6. The MBP of MS brain tissue has been shown to be severely deficient in phosphorylation, which we know can be caused by formaldehyde.
7. The Smoking Paradigm: Cigarette smoke is high in methanol and is the only etiological cause of MS that is generally accepted by the scientific community.
8. Consistent circumstantial evidence links increases in methanol-containing food consumption and in industrial use of methanol to corresponding increases in MS incidence during the transition from the 19th century into the 20th century.
9. The advent of aspartame, a methanol carrier, has introduced an opportunity to quantify additional methanol in the food supply since 1981.
10. The Aspartame Paradigm: statistics show convincingly that as more and more aspartame is consumed by the US population the incidence of – and perhaps more importantly the death rate from – MS has also increased dramatically.

11. The higher incidence of MS in colder climates was due to the higher consumption levels of canned fruits and vegetables in temperate climates. This began reversing shortly after methanol-containing diet sodas and other thirst quenching products became popular and inexpensive in the tropics.
12. MS was at one time a disease of men when it was caused by industrial contact. It is increasingly more of a women's disease. When methanol is inhaled as a gas during cigarette smoking or industrial contamination the distribution tends to be equal between the sexes. The stomach of the man, however, has 4 or 5 times more ADH in its lining than that of a woman. When methanol is consumed via diet soda, the ADH removes methanol before it can get to the brain, so less of it reaches men's brains than women's brains. As more and more methanol has become a dietary poison, the shift from male to female disease has followed.
13. The Faroe Islands are surrounded by countries with very high incidence of MS, yet the country traditionally did not have the disease represented in its population until after the occupation of large numbers of British Troops during the Second World War. Faroes have no trees or peat deposits and, therefore, developed methods to salt and air dry fish and other meats for preservation, unlike its neighbors, who dine on smoked foods at each meal. The indigenous diet of the Faroans contains no methanol.
14. The Village of Wellington, Ohio experienced an epidemic of MS that should have been traced to the escape of methanol fumes from a foundry, affecting the populace located downwind of it.
15. Professions such as shoe making and papermaking that have been shown to have high incidence of MS can also be shown to have exposes their workers to levels of methanol.
16. The Teaching Paradigm: The US teaching profession might just be the best profession to use to link methanol exposure to increased incidence of MS. Secondary school teachers suffer an incidence of MS almost twice as high as their professional counterparts. They also can be shown to have had consistent workday exposure to methanol fumes by the ubiquitous use of Ditto machines that use high concentrations of methanol as a print transfer agent.

It has been over 30 years since I heard my first unsolicited plea for help from an aspartame consumer who had linked consumption of the product to her suffering. My first thought after an hour's listening was that this courageous young woman would soon be diagnosed with Multiple Sclerosis. It is in her honor and in the memory of my friend from Wellington, Colorado that I seek to explain the compelling link between methanol and MS.

Post Script:

A Word About Depression, a Common Complaint of Methanol Poisoning and all the DOC

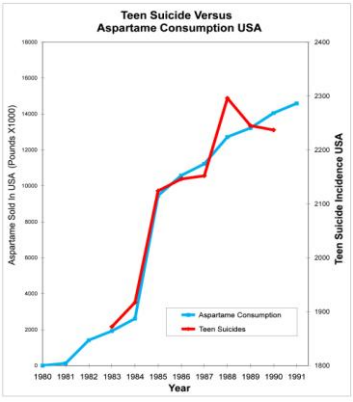
We have charted two methanol diseases that have long gestation periods. However, methanol poisoning, MS and aspartame consumption have one symptom that is a quick responder (24-48 hours) that permeates the literature of both methanol poisoning and MS and is a constant anecdotal complaint from aspartame consumers. Depression was the second most reported complaint from aspartame consumers, next to headaches, in the study done by the CDC when it finally took a cursory and shallow look at the health impact of aspartame in a very conflicted and highly unusual report published by them in 1984.[#58]

A comprehensive double-blind crossover study designed to ascertain whether individuals with mood disorders were vulnerable to developing depression after consumption of moderate amounts of aspartame concluded that "individuals with mood disorders are particularly sensitive to this artificial sweetener and its use in this population should be discouraged"[#54] The result of this experiment were statistically significant even though the experiment was halted prematurely by the Ohio Universities College of Medicine Institutional Review Board due to the severity of reactions within the group of patients with a history of depression. Such major reactions are uncommon in the aspartame toxicity literature. The reason for this may have to do with the difficulty in procuring the chemical for testing purposes without having to rely on the honesty of the manufacturer. Dr. Ralph G. Walton chairman of the department of Psychiatry first author of the article explains in his own words what he went through.

"Nutrasweet Company did try very hard to prevent me from getting aspartame for our double blind study. The company had stated that they would supply aspartame and placebo capsules free of charge to any "legitimate researcher." As a full professor and chairman of a department at a major medical school I think I qualified as "legitimate" but they refused to provide it to me. When I pressed they said it was "unnecessary" research. At that point I said I was willing to buy it from them rather than have it provided free. They still refused. I then turned to bottlers of diet soda and was told that they had been instructed by the company not to sell it to me. We did eventually obtain aspartame, but only after a world-wide search. In retrospect I am glad that I did not get the aspartame or placebos from the company - who knows what they would have sent me?"

As a teacher, I immediately noticed that college students and teenagers, in particular, were quick to pick up on the great-tasting aspartame sweetened diet sodas as a way to keep their weight down. I asked the CDC for the data it had collected on successful suicides of teenagers during the years after aspartame was added to carbonated beverages and its consumption exploded. The response was startling. Figure 6 represents this data in the usual fashion. Unlike Alzheimer's, with a 14-year delay until death, the teen suicide rate of this important period spirals around the aspartame consumption curve like the two snakes slithering up the caduceus.* This quick response would be expected since the mood alterations generated by both methanol and aspartame take hours, not years. There is also some evidence that the anhedonia produced by methanol may be identical to that induced by alcohol withdrawal and linger for some time.[#38] This striking yet tragic illustration required the deaths of 1600 teenagers above the normal rate in the US to generate. Their parents would not consider their loss anecdotal.

Figure 7



* The caduceus the magic wand of the Greek god Hermes (Mercury) and is used by the medical pharmaceutical establishment as their symbol. Hermes is considered the protector of merchants and thieves.

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