

A deadly experiment?

» Viewpoint | By Dr Woodrow Monte



Is there a link between multiple sclerosis and aspartame? After 25 years of research into the matter, Dr Woodrow Monte presents a strong argument to suggest the rise in both MS and aspartame consumption is no simple coincidence

{ We cannot introduce a poison into the food supply without paying a terrible price, and I am convinced that such a scenario is currently playing itself out. In an earlier article regarding the dangers of aspartame (*Fitness Life*, 2007, Nov 33: 31-33), I explained how this artificial sweetener can degrade into methanol and then into formaldehyde, and highlighted some of the deleterious effects of formaldehyde on the human body. In this issue, I make the case that increased methanol consumption is implicated in the initial appearance and increased incidence of multiple sclerosis

(MS)... and aspartame is one of the two primary sources of methanol in our diets. The other is canned food.

What price aspartame: The early warnings have been ignored

We have consumption data for aspartame in the United States from mid-1981, when a ban on its use was reversed through political intervention. However, the increase in aspartame production and consumption was relatively slow until it was approved for widespread use in fizzy drinks. Such a turning point offers a perfect time to look for anomalies that might help us to hypothesise about the extent to which such a substance may be toxic.

I have examined data on those diseases and conditions that are most likely to have been affected by the increased consumption of aspartame. These include depression, autoimmune disorders and birth defects (terata) – both methanol and aspartame are proven teratogens in animals far less sensitive to methanol than humans are. Data from the US National Center

for Health Statistics, regarding morbidity numbers during those critical early years, are presented here in graph form.

They are unmodified, save for those figures regarding autism. These have been displaced by six years, because they concern patients diagnosed at six years of age; this reflects the fact that exposure to methanol occurred in the womb. If these numbers teach us anything, it is that the first question we should ask a depressed child is: "Do you drink diet soda?" And that pregnancy is not the time to consume aspartame.

However, I find the most striking graph is the one showing the increase in diagnosed cases of MS. It usually takes at least 10 years from the first onset of symptoms to full diagnosis of the disease, so this early reporting was evidence, to me, of much worse to follow. Science has been seeking the cause of MS for 150 years, and it has repeatedly been suggested there is evidence to implicate a small molecule – a solvent – in the development of this autoimmune disorder. Methanol is the smallest of the solvents.

The results of 27 years of aspartame consumption: The worst of timing

The 1980s were well into the era when laboratories performing methanol toxicity research were being paid by the company that invented aspartame to prove the safety of the sweetener. Dr Hugo Henzi, an M.D. now deceased, published a book in 1980 that set out to prove dietary methanol was the cause of MS. His clinical logic and anatomical observations were impeccable, but he made a major mistake. He erroneously believed that the methanol that caused MS came from fresh fruits and vegetables, and consequently proposed a curative diet that we now know had little chance of success. However, several lines of evidence are now converging to support Dr Henzi's primary assertion.

The secret battle that is autoimmunity

From an early moment in the evolution of humankind, a biochemical battle has been quietly raging inside our complex anatomies. Two alcohols have been competing for the

attention of one serendipitously distributed and poorly understood enzyme – an enzyme that has become the lonely suitor to, and only benefactor of, their advances. The outcome, after years of struggle, determines each time who will and who will not die from MS. Those alcohols are ethanol and methanol.

Methanol is the smallest known alcohol, containing only one carbon atom; ethanol has two carbon atoms. The enzyme in question has had several names as it has been discovered and rediscovered over the years by the physiological, neurological, and ophthalmological sciences. However, it is most commonly referred to as ADH – alcohol dehydrogenase.

ADH is known to serve a number of functions in the healthy human body, but its ability to convert alcohols to aldehydes is what is pertinent to our discussion here. ADH is a large protein molecule, and due to its structure, it much 'prefers' coupling with ethanol. It converts this to acetaldehyde, which our body makes use of for many important purposes.

It is only when ADH finds no ethanol in the

blood, or when the methanol concentration in the blood is 10 times greater than the amount of ethanol, that ADH 'reluctantly' turns methanol into formaldehyde. So, without the inhibiting effect of ethanol on formaldehyde formation, humans would have become extinct eons ago.

Another bit of good fortune for the majority of us is something that was noticed during the development of breath analysers for the detection of drunk drivers, and that has subsequently been confirmed in the scientific literature. That is, we nearly always have ethanol circulating in our bloodstreams.

Ethanol generation is a natural outcome of digesting plant material in the gut, but the amount of endogenous ethanol in our blood varies greatly across individuals. Some subjects show no ethanol at all, while others have enough natural ethanol in their blood to be considered impaired drivers. Individual differences in the presence of endogenous ethanol, and the location of tissue containing ADH, may well account for how some people die or are blinded by one teaspoon of methanol, while some consume

it – mixed with ethanol – as a preferred source of intoxication. I also believe that these individual differences in the presence of endogenous ethanol provide an explanation for why some people develop autoimmune disease and others do not, no matter what they consume or smoke.

Interestingly, the location of ADH in our bodily tissue seems to vary with our genetic makeup. ADH might, for reasons not fully understood, be found in the breast, liver, gut, brain, eye, skin and sinew. These hereditary differences are most likely responsible for the varied manifestations of autoimmunity. Higher enzyme representation in the brain might predispose an individual to develop MS, while its presence in the skin might be required for the evolution of lupus.

Autoimmunity:
Teaching our body to
produce antibodies
against our own tissue

Macrophages are large white, amoeba-like blood cells, whose key purpose is to destroy attacking life-forms, and consume foreign or broken proteins. For reasons yet unknown, evolution equipped macrophages with chemical receptors that can detect and vigorously destroy any protein treated with formaldehyde. In addition, they can signal the immune system to produce antibodies to the proteins that they consume.

During the evolution of vaccines, not long after the pioneering work of Jennings and Pasteur, the pharmaceutical industry noticed and took good advantage of the new ‘trick’ of toxoid production. A toxoid is a bacterial or viral protein that has been treated in the laboratory with a low concentration of formaldehyde – just enough to attract the attention of a macrophage, but not enough to completely change the protein’s structure. The toxoid – or vaccine – is injected into humans, who then produce antibodies, developing immunity to the

original, offending organism. It is interesting to note that injection of the original protein, without formaldehyde treatment, often causes little or no antibody production.

As discussed in my previous article, once methanol is changed into formaldehyde, and then into formal hydrate when it meets water, all hell breaks loose. Unbeknown to most researchers, formal hydrate is extremely acidic and is also a powerful esterifying agent. These two characteristics make it irresistibly attractive to our body’s protein molecules, especially to those found in the insulation of the human brain and nerves. Opposites attract, and the basic protein (MBP) component of myelin quickly falls prey to formal hydrate. And, this is a dark side to the good work of the macrophage. What happens when the protein in our brain comes in contact with formaldehyde from diet soda?

An increase in MS

It cannot be denied that there has been an epidemic of autoimmune disease worldwide over the last 30 years. MS, once almost unknown in Japan, has now risen to menace a large proportion of the population. The lower latitudes and warmer climates, once mysteriously protected from the full brunt of this tragically debilitating disease, have seen its incidence and prevalence climb to as much as four times what they were in the days before summer drinks were sweetened with aspartame. The United States, which has long had a relatively high MS incidence, has seen at least a 50% increase in the condition. Medical journals in Australia and New Zealand both report inexplicable increases in their inordinately high rates of MS.

Where does methanol
come from, aside from
aspartame?
Cigarette smoking

After 150 years of study of MS, only

cigarette smoking is universally accepted as a causative agent. Smoking has also been causally linked to the progression of MS, transforming a relapsing-remitting clinical course into a much more serious secondary progressive course. Tobacco leaves contain large amounts of pectin, and although most scientists are unaware of it, they are left in barns for weeks, to ferment. This releases much of the available methanol from the pectin into the moisture content of the tobacco, before it is sold to be made into cigarettes. Consequently, methanol is one of the most abundant toxic compounds found in cigarette smoke.

Food processing

Although acknowledging the role played by smoking, I believe that food processing and preserving is what first dramatically increased our methanol consumption. At some point in our genetic history, a mutation occurred that disrupted the ability of the liver enzyme, catalase, to quickly and safely clean methanol from our blood. For several million years, there was no downside to this and no ongoing autoimmune ramification. We ate fresh food, and what very little methanol is found in fresh fruits and vegetables is countered by their own ethanol content and their ubiquitous gift of slow, steady ethanol production in the gut. However, when the fruits and vegetables, and their naturally occurring pectin, is placed in a sealed container – as in canning – and sterilised, heated, or even just stored at room temperature for months, the normally unavailable, chemically bound methanol is released from the pectin. It slowly builds up, trapped in the container, to hundreds of times more than its concentration in fresh food. Hence my assumption of canning being the major source of free methanol in the human diet before aspartame.

The history of MS
recapitulates the history
of the canning industry

Nicolas Appert invented canning in the 1790s, and the first canning factory was fully operational in England by 1813. Due to the expense, early canning was undertaken primarily with meats, which have no pectin content and therefore would not have caused methanol accumulation. Soon, however, came the canning of fruits and vegetables, and as it became more prevalent and less expensive, the consumption of canned food skyrocketed... as did the incidence of MS. 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 throwing them away.

The first documented case of MS was reported by Jean-Martin Charcot in a lecture in 1868, although it is thought that the first identifiable instance of the disorder was that of Augustus d’Este, whose symptoms started between 1822 and 1843. During the 19th century, MS was recognised as a disease, but considered “quite rare”, with Charcot reporting fewer than 40 cases during his long career. However, increasing numbers of cases were being reported by the late 19th century. Although co-occurrence is not proof of causality, similarities in initial appearance and in the rate of increasing incidence are consistent with a close link between MS and the consumption of canned fruit and vegetables.

Explaining the
unexplainable: MS is
a disease of the colder
countries

The differential prevalence of MS across different geographical regions also supports this connection. The ‘latitude gradient theory’ is used to

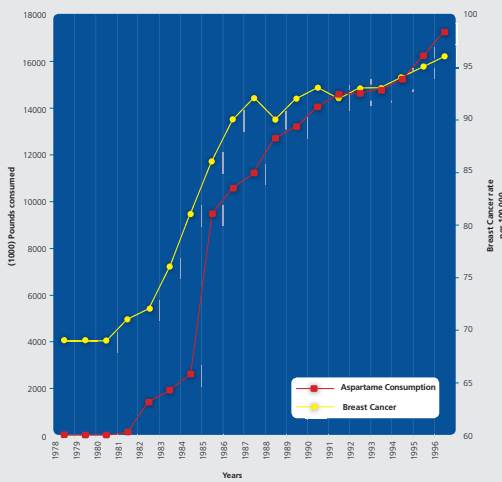
explain the occurrence of higher instances of the disorder in the colder regions of the world. The tropic zones have, until recently, been blessed with very low MS figures.

These warmer climes are, of course, regions in which the ongoing supply of fresh fruit and vegetables has obviated the need for more expensive canned produce. Exceptions to such a generalisation include those areas with established canning industries that can offer their products so economically that they are a tempting alternative, even in the summer months. For many years, this has included both Australia and New Zealand, and these countries are significant exceptions to the latitude gradient theory. Both have an extremely high prevalence of MS.

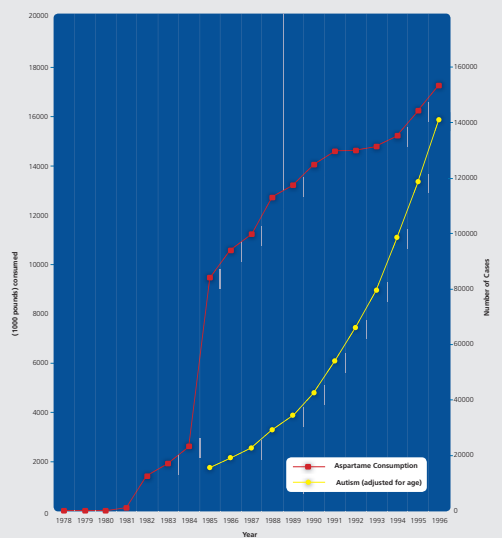
Another exception is Japan, which lies in the colder latitudes, but which has had little MS incidence through most of its recorded history. Note, however, that the Japanese cultural habit of eating everything in its season did not foster the production or importation of much canned plant material. Although far from tropical, until recently Japan has had one of the lowest rates of methanol consumption per person in the world. This rate has increased with the growing popularity of diet beverages; the world’s largest manufacturer of aspartame is a Japanese company.

Seasonally speaking, in the northern latitudes – before aspartame – patients with relapsing-remitting MS could expect episodes to be experienced in the winter or during periods of peak methanol consumption. However, with the increased consumption of diet beverages, Japan and the warmer countries are now reporting their worst relapse time to be the summer. The difference between warm and cool regions in remission-relapse cycles appears

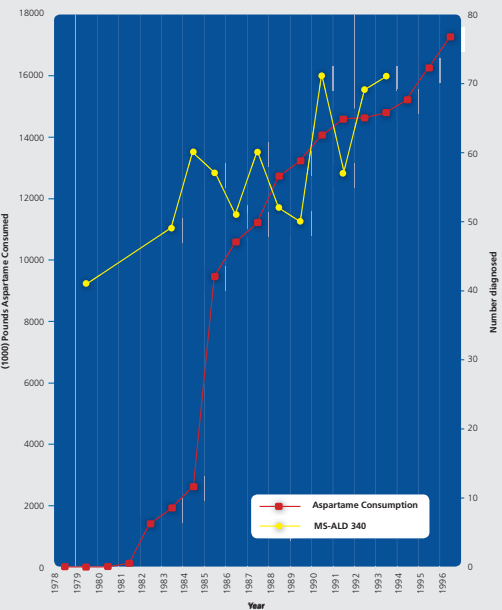
Breast Cancer and
Aspartame Consumption



Autism and
Aspartame Consumption



MS and
Aspartame Consumption



to parallel periods during which toxic canned vegetables and toxic thirst quenchers are most frequently consumed.

Epidemics of MS

There have also been bizarre epidemics or clusters of MS on the Faroe Islands, Orkney and Shetland Islands, and in Iceland. All of these have been studied in great detail and all involve the influx of massive numbers of British or Allied troops. One researcher concluded that those individuals most affected were those who had been in direct contact with the soldiers; another article goes so far as to accuse MS of being a sexually transmitted infection.

An alternative explanation is that these islands had a very low incidence of MS to begin with, due the lack of an established canning industry. Troops brought with them items such as canned foods, fruit preserves, marmalades, and canned rations, along with the ubiquitous cigarette, all of which would be very desirable to the islanders, especially during war time. Methanol consumption was overlooked as a factor in all these studies.

The Scandinavian countries and parts of the Slavic nations have some of the highest MS rates of any populations in the world. Here, consumption of canned and home-canned fruits and vegetables is high, as is the intake of smoked foods and traditional liquors made from fermented culled fruit. Some of these drinks have a high enough methanol content to exclude them from international commerce. In fact, the alternate name for methanol is ‘wood alcohol’, due to its original source as a condensate made from wood smoke. Smoked meats and fish are the exception

to the rule that animal products do not contain methanol.

The white man’s burden

MS was once considered to be a rich man’s disease, in that its prevalence was positively correlated with the trappings of civilisation, including modern sanitation practices. It now appears that the consumption of canned produce provides a better explanation for the relative dearth of MS in many economically depressed regions. To this day, the poorest people of the world are free from autoimmune diseases such as MS; while they lack proper sanitation, they also cannot afford canned fruits and vegetables, or diet soda.

The identical symptoms of MS, methanol poisoning and aspartame toxicity

The symptoms of MS, chronic and acute methanol poisoning, and aspartame toxicity, are in all ways identical. There is nothing that methanol does to the human body that is not expressed during the course of MS, including the very unusual destruction of the myelin sheath with no harm to the axon itself.

This generalisation extends even to the remarkable opthomological 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. In fact, these symptoms have been thought of for years in their respective literatures to be tell-tale indications for the diagnosis of each malady. The common symptoms of headache, nervousness, depression, memory loss, tingling sensations, pain in the extremities, optic neuritis, seizures,

bright lights in the visual field, inability to urinate or to keep from urinating, are all shared by both illnesses, and also by aspartame poisoning. I take this strikingly similar symptom pattern as evidence that these disorders act on identical components of the central nervous system, and in exactly the same way.

The ‘miracle’ that MS shares with methanol poisoning

In the early stages of MS, or when a non-lethal dose of methanol has been administered, complete recovery is a possibility. The only two afflictions in which such dramatic remissions from identical neuromuscular and opthomological damage – including blindness – are reported, are relapsing-remitting multiple sclerosis and methyl alcohol poisoning.

Sex ratios for MS and aspartame reactions

Women bear the brunt of multiple sclerosis and lupus (SLE), being three times more likely to suffer from these diseases than men. This is exactly the proportion represented by adverse reactions to aspartame, in a study released by the US Center for Disease Control in 1984. The Center found that three women to every man had aspartame consumption complaints that were serious enough to warrant investigation.

What might account for such a difference across the sexes? A study published in the *New England Journal of Medicine* reports biopsies of the gastric lining of men and women. One outcome was the fact that the concentration of ADH in the gastric lining of men was much higher than in woman. Thus, in men, methanol

is more likely to be removed from the bloodstream before it reaches the brain. So, the brain is spared, but the methanol still has to be metabolised to formaldehyde in the gut. This may help explain why men have more gastrointestinal complaints from both methanol and aspartame consumption. On the other hand, women’s complaints from both more frequently involve serious neurological complications.

MS cures and treatments

Viewing methanol toxicity as the ethnologic cause of MS seems to answer all the nagging questions and unexplained anomalies that have stalled the cure for this increasingly persistent disease. And there is no known cure for MS. After reading up on the many treatments, I conclude that the only one that shows a statistically valid improvement in double-blind studies – albeit for a relatively short period of time – is plasmapheresis. This involves removing the liquid portion (plasma) of the patient’s blood, then returning the red and white blood cells without the plasma. Although not prescribed for this condition, the process would be expected to remove much of the methanol from the bloodstream, reducing its concentration substantially in the tissues.

Conclusion

Consumption of aspartame always results in methanol consumption. Methanol will always convert to formaldehyde where it finds an idle ADH. When this happens in the brain, any protein changed by formaldehyde will be destroyed by white blood cells. The protein most likely to be destroyed would be the myelin basic protein found in the axons. Over a long enough period of time, even without concomitant antibody production, there are those who would

call this MS.

This ends my case for considering methanol to be the cause for MS. Call all this hypothesis and circumstantial evidence if you like. The best experiment to confirm it would never have been allowed by any human subjects committee, even though it has been going on for 27 years. As far as I am concerned, it is time to call that experiment complete and to count the bodies.

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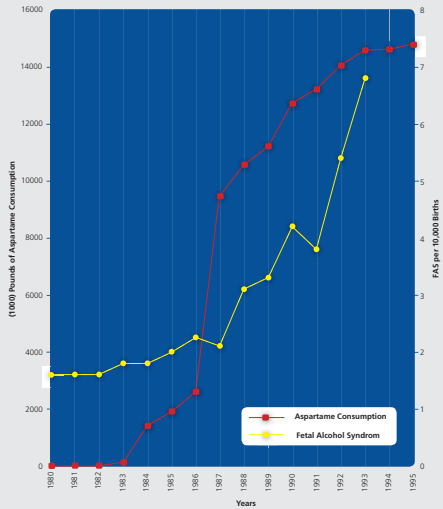
Note 1: It has been more than 25 years since I heard my first unsolicited plea for help from an aspartame consumer who had linked consumption of the product to her suffering. My first thought after an hour’s listening was that this courageous young woman would soon be diagnosed with MS. It is in her honour and that of courageous consumer advocate, Abby Cormack, that I seek to explain the compelling link between aspartame, methanol and autoimmunity.

Note 2: A fully referenced version of this article is available at: TheTruthAboutStuff.com.

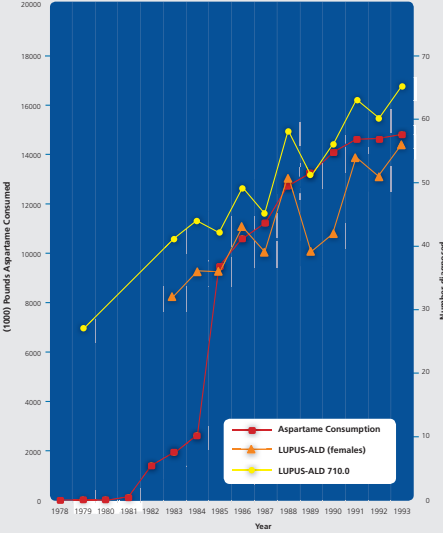
What are your thoughts about the safety of aspartame? Do you believe products containing aspartame should carry a health warning? Email us at fit@fitnesslife.co.nz

Ed’s note: Due to the proliferation of pro-aspartame data available and provided regularly by the NZSFA, soft drink, gum and food manufacturers, and the media in general, we have chosen to report the other side of the aspartame debate only.

Fetal Alcohol Syndrome and Aspartame Consumption



Lupus and Aspartame Consumption



Teen Suicide and Aspartame Consumption

