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Subject: 2016 CIC Prioritization

Dr Ramirez

Please accept the attached Chapter 12 The Cancers of Aspartame of my book While Science Sleeps and all the references as my submission to the Office of Environmental Health Hazard Assessment in support of prioritizing aspartame as an important carcinogen.

I have attached a pdf copies of both for your connivance.

Many of the cited references can be found on my website: http://www.whilesciencesleeps.com/references/
If I can be of further help please advise.

Woodrow Monte PhD
Emeritus Professor of Nutrition
Arizona State University
Aspartame’s proclivity to cause cancer in humans should be a surprise to no one. The unimpeachable biochemical pathway whereby its methanol transforms into formaldehyde at cancer prone ADH-containing tissue locations throughout the human body constitutes sufficient scientific basis for considerable concern. Animal feeding studies of aspartame show statistically significant, dose dependent increases of breast and kidney cancers in test animals even though these animals are known to be more resistant to the toxicity of methanol than humans. The Centers for Disease Control SEER program, which monitors the wellness of the US population, has published data showing an alarming increase in these and other important cancers that mirrors the popularity and consumption of aspartame over the last 30 years. The real question is, why hasn’t this overwhelming commonality of logic and fact been sufficient evidence to remove aspartame from the marketplace?

Considerable evidence against the safety of aspartame comes directly from its regulatory file history, stored in the vaults of the US Food and Drug Administration. Aspartame’s carcinogenity (ability to cause cancer) in laboratory animals is documented in studies done on the sweetener before its approval. Cancerous brain tumors found in laboratory rats in a dose-dependent relationship to the content of aspartame in their diet were among the most compelling reasons it was consistently rejected for use in foods. It took the hiring, by the G.D. Searle company that invented the sweetener, of a Washington insider, Donald Rumsfeld, as CEO in March of 1977 to force aspartame into the food supply.

The exquisite and pivotal laboratory work done by Professor Maria Alemany despite the strenuous, though unconvincing, objections of the opposition is proof conclusive that aspartame and, therefore, methanol does turn into formaldehyde within the living organism. Dr. Alemany skillfully used radioactive tracing of the methanol component of aspartame and thus clearly and unequivocally demonstrated methanol’s evolution into the formation of formaldehyde modified proteins and DNA. How strange it seems now that anyone would ever doubt that formaldehyde is produced from methanol or be convinced to think otherwise! For his exquisite work, Dr. Alemany had his university funding revoked and, like so many other scientists that dared go against the corruption that abounds in the western bureaucracies, was made to endure indignities designed to discourage dissension.

Laboratory animals that take our place in safety testing have catalase enzyme in sufficient quantity within their bodies to protect them from the lion’s share of the carcinogenic effect of methanol’s formaldehyde. When an animal does show signs of cancer the ramifications for humans are ominous. Therefore, when it comes to making a fair determination of the carcinogenic potential of methanol or aspartame, we are dependent on careful epidemiological observation of exposure of human populations to either methanol or aspartame. Nothing is wrong with this approach; it brings us back to the roots of the health sciences where observation leads to the application of logic and, in more cases than modern practitioners of medicine would like to admit, to the development of methods to prevent disease.

For aspartame, we have excellent data of its consumption rate during the extremely important time its popularity was ramping up in the United States. The entire US population has been a study group, with no knowledge or forewarning of its participation in a ghastly experiment. Using this data may be the very best way that its cancer causing potential can be determined. Toxicologists could only dream of such a large blind research population, which normally would make the job of the epidemiologist quite simple. Unfortunately, many toxicologists owe their allegiances to various industrial and pharmaceutical groups who insist they
waste their skills proving how safe new chemicals are rather than showing the havoc that they might cause. No worries; we will see for ourselves where the truth lies and make our own determinations by looking at the graphs of the aftermath of thirty years of aspartame consumption. We will put ourselves in the position of the ancient Aztec, Inuit or Judean intelligentsia whose insights guided their people away from foods that might do harm. The edge we have over the ancients is that our culture has uncovered knowledge of the chemistry of nature, and this can reinforce our observations with reasonable insights as to the molecular details that might lead to cancer production.

**Formaldehyde’s Cancer-Causing Prowess Is Enhanced When It Comes from Methanol**

What exactly is the link between cancer and aspartame’s methanol component? Without a doubt, a poison such as methanol that produces formaldehyde within cancer-prone tissue throughout the human body will eventually cause cancer. On the very day this paragraph was begun, June 10, 2011, the government of the United States finally caught up with the rest of the world and officially declared formaldehyde a “known human carcinogen.”

A full grown human can die from ingestion of just a “few drops” of formaldehyde with death from acute formaldehyde poisoning often taking only minutes. Air contaminated with formaldehyde, even at very low concentrations, is now known to cause cancer in humans. Again let me stress that formaldehyde is one of a handful of chemicals classed as a Group I carcinogen by the IARC, the International Agency for Research on Cancer, Lyon, France. No known safe level of formaldehyde exposure exists; it is in all amounts a dangerous carcinogen and mutagen.

Even so, were it not for one major obstacle, formaldehyde might reach the status of one of the most perfect and powerful of carcinogens. Its only weakness is that it is so highly reactive that its destructive power is usually used up before it can travel all the way to the nucleus of the cell, where cancers originate. Like an overanxious lover, it spills its energy long before it makes its mark, prematurely squandering itself prior to reaching the perfect site for its application. On the other hand, methanol as a source of formaldehyde acts as a partner to its carcinogenic potential, easing it into places much closer to where it can more successfully work its dark magic, potentially producing cancers in all the human sites of ADH activity, including the liver.

It only takes one cell succumbing perfectly to formaldehyde’s attack to produce a full blown cancer. Other methanol diseases, such as multiple sclerosis, atherosclerosis and Alzheimer’s, are all slowly developing diseases that require years of collective damage caused by countless formaldehyde interactions that eventually add up to a defect that manifests as a chronic disease. Not so with cancer. A single cell’s bad encounter with a carcinogen can initiate cancer progression that is virtually unstoppable. The exact behavior of the cancer from then on depends on which type of cell is affected and how quickly it can multiply. In some cases, individuals have been exposed to known cancer-causing agents for years and never experienced cancer, but in others, after just one encounter with asbestos, plutonium or formaldehyde, a cellular change was induced, turning one cell into the seed of a fatal malignancy.

Does that mean that just one sip of diet coke could start a cancer? Regrettably, the answer is yes. Cancer, therefore, is a purely statistical phenomenon in a game of environmental roulette. We turn the wheel each time we take a sip of diet soda or a puff of cigarette smoke and the wheel does not stop spinning until all the resultant formaldehyde has found its final home. If science understood the exact details of what cancer “really” is, I would certainly spend time in its explanation. Here again, we have put our trust to find the cure for cancer into the hands of those who have the most to lose when a remedy is found. We know little more about the truth of cancer than we did when lone scholars worked long hours in underfunded university laboratories out of the reach of Big Pharma.
What we do know with certainty is that unwanted methylation and inappropriate cross-linking of chromosomal tissue play a major part in the production of cancers. Both these phenomena are the handywork of our Crazy Hawk. Formaldehyde is actually just a reactive methyl group, a “methyl molecule” that is essentially a methylation machine. It is capable of the methylation of DNA as long as enzymes are available, as they are in the living cell, to finish the job. Over 60 methylation defects of DNA from human cancer cells have been identified thus far. This hyper-methylation of DNA causes the shutdown of the expression of genes involved in preventing the production of important proteins that have been coded into the methylated portion of DNA. These are the changes that also come into play during the evolution of Autism and other birth defects, which we will discuss in the next chapter.

As for cross-linking of DNA, few chemicals are more likely to form a bridge between a DNA molecule and protein or other DNA than formaldehyde hydrate. The indiscriminate attachment of formaldehyde to the chromosomes in the nucleus of a living cell has been shown time and time again to cause the inappropriate linking together (cross-linking) of these molecules in such a way as to induce cancer. This works much the same way as the linking together of the Tau proteins, resulting in Alzheimer’s. In humans, the methanol placement of formaldehyde is the key to its ultimate perfection as a carcinogen. Methanol is a very small and nonreactive molecule. It can go anywhere in the body with no effort at all, as a Trojan horse delivering formaldehyde to where it can cause severe damage.

You should come away from this discussion with the knowledge that the ultimate cancer-causing agent in humans just might be a trinity of unfortunate circumstances allowing methanol to make contact with ADH, which converts it to formaldehyde. Show me a place where ADH can be found in the human body and I will more than likely be able to show you a cancer that has increased in incidence since that fateful day in 1981 when a new source of methanol was forced into the diet of the unsuspecting consumer.

**Thousands of Carcinogens Exist**

We have a number of new chemicals in our environment which, to my way of thinking, have not undergone proper screening for carcinogenicity. This is the result of a lack of genuine governmental oversight and the extraordinarily unrestrained involvement of chemical and pharmaceutical interests into the bureaucratic decision-making process relative to the safety of chemical substances (along with the largely industrial leanings of the toxicological and epidemiological professions in general). It is still technically against the law of the United States to allow any chemicals known to cause cancers in man or animals to be an ingredient added to foods (pesticides have been exempted). Since we now know that aspartame turns into a cancer-causing agent, we need to look at what the effect of adding millions of pounds a year of that carcinogen to the food supply over the last thirty years has done to the cancer rates in the US. We will concentrate on those cancers that originate in the known centers of ADH I
concentration in the human body where formaldehyde is the predictable outcome of aspartame consumption.

Cancer statistics for the United States are published by the Surveillance Research Program of the National Cancer Institute, which manages the Surveillance, Epidemiology, and End Results (SEER) Program. The SEER program is an authoritative source of information on cancer incidence in the United States. SEER collects and publishes these statistics from population-based registries covering 26% of the US population. Looking first at an overview of the total cancer burden for the United States, I have plotted the CDC SEER data for incidence of “all cancer” occurrences in the United States for the period since the introduction of aspartame (Chart 1). This chart clearly shows the full impact of the sudden increase in aspartame consumption. But why does the cancer incidence appear to peak in 1993?

**The Sensitive, Vulnerable, and Susceptible Will Be Lost First**

To answer that question, I will need to relate a story. A few years back, upon landing in Christchurch on one of my many flights from the States to sanctuary in New Zealand, I was contacted by a good friend who asked if I wouldn’t mind sitting for the filming of a TV interview that might never see the light of day. The interviewer, a Kiwi with a reputation as a hard-hitting investigative journalist, was said to have an interest in aspartame, but he didn’t think he could talk his managers into what would be an outright challenge to the cola industry, a major source of advertising dollars to his company. New Zealand media, once run by the government with no need to pander for its survival, had deteriorated quickly to the US model after deregulation. I accepted and we asked the airport hotel to let us use an unoccupied garden area as a temporary studio.

I will never forget one of his questions, obviously something that he had researched and which had been on his mind for some time. “Professor Monte, do you think that the unexplained increase in teen suicides in New Zealand has anything to do with allowing carbonated diet beverages into the tucker (school lunch) programs?” I explained that I did not realize that New Zealand had experienced a similar problem to ours in the US. My answer was in the affirmative, and followed the lines of logic that I used to explain the chart in Chapter 9 of the teen suicide experience in the US. He quickly countered with a question that I am sure he hoped would please his employers. “Well, how do you explain that the consumption of diet soda has been steady over the last few years and the teen suicide rate is now in decline?” Without hesitation, I countered with “It is obvious your advertiser has succeeded in killing off the most sensitive kids.”

The show never aired, but I relate the incident to you because it points out a common outcome of toxicological experimentation done on large populations of test animals. The introduction of a poison into the diet of a colony of test animals is the very most important stage of the experiment. It is at some point after this that we expect to see the most susceptible individuals of the colony, never before stressed at these levels, killed off. I have done such experimentation myself and know what to expect. The horror of this, to me personally, is that the curve I would expect to see in a laboratory study of a deadly poison would be shaped very much like Chart 1, with a spiking of incidents followed by a slow down as the more resistant animals remain alive and in the study. The time it takes to achieve this peaking of the curve varies, depending on the characteristics of the poison administered. The most interesting indication of this phenomenon is the curve never drops all the way back down to the baseline (where it started before the experiment began) for as long as the poison is being administered to the colony. This indicates a definite cumulative effect on the population as a whole. The “depletion of the weaklings” is what we used to call it before we came to realize that in the case of aspartame each of these data points represented a person.

**Brain Cancer: the Killing Begins**

*Increasing Brain Tumor Rates: Is There a Link to Aspartame?* is the title of an article published in *Chapter 11*
1996 by the American Association of Neuropathologists and written by the renowned research neuroscientist John W. Olney. John cites the results of the original aspartame animal studies I mention above “revealing an exceedingly high incidence of malignant brain tumors in aspartame-fed rats” and notes that “spontaneous brain tumors in laboratory rats are quite rare.” He goes on to point to the relevancy of a Public Board of Inquiry that had been called together by the US Food and Drug Administration in 1980 to evaluate these studies, and had “included prominent neuroscientists Walle Nauta and Peter Lampert.” This Board subsequently concluded that “aspartame may contribute to the development of brain tumors in the human population.” They recommended that the Food and Drug Administration withhold approval of aspartame as a food additive. Within months of this decision, Donald Rumsfeld’s team was in control of the White House, and within days aspartame was a “food.”

Nauta points out that a sharp upward trend in deaths from brain tumors was detected in the mid 1980s in countries that approved the use of the sweetener, including the United Kingdom, France, Germany and Italy. Nauta found during his study similar increases in brain cancers in the States, noting that “it was not a fleeting phenomenon. Rather, it was the initial phase of an upward swing in the brain tumor incidence which evolved into a sustained increase in both the per capita number and malignancy of brain tumors, a phenomenon that has endured for at least 8 years.”

I have taken the liberty to chart the data from Figure 3 found on page 1119 of Dr. Olney’s article on our standard graph of aspartame consumption (Figure 11.2). You can see from this chart that the brain cancer rate in the US population more than doubled after the introduction of aspartame. Olney goes on to warn that “Compared to other environmental factors, aspartame appears to be a promising candidate for explaining the surge in brain tumors in the mid-1980s.” In his concluding sentence he pleads for a “reassessment of the carcinogenic potential of aspartame which is currently being ingested widely throughout many parts of the world.”

John’s fine article is a professional plea from a world renowned scientist and physician who is respected by not only his neurological peers, but by the scientific community in general. The plea is in the form of a scientific article that had been reviewed and published in the premier journal of a leading neurological association whose scope includes this exact type of research. Reading the article you will see the high standard that is used to evaluate the potential of aspartame to act as a human carcinogen. John asks three questions: 1) Does the agent have carcinogenic potential? (This is John’s weakest argument since he cannot use the formaldehyde link); 2) Do experimental animals show an increased incidence of specific types of
This man could not have done anything more. He is an expert in his field and he proceeded in the proper way, and yet absolutely nothing changed. He was ignored by the FDA and all the other such public organizations of the world that are charged to protect their respective populations from poisons. The argument used against John’s premise was the so-called “ecological fallacy.” The meaning of the fallacy goes something like this: “The temporal coincidence of two events observed at an ecological level without examination of individual data can lead to faulty conclusions regarding risk association.” In other words, because two things happen at the same time, that doesn’t mean they are related. This, of course, could be true in this case except for the ignored fact that John went beyond this and made a good case for the science behind the cause of the cancer and showed that the same cancer was demonstrated in laboratory animals. John did all this and his premise should have won the day. His argument should have swayed the FDA to immediate action against the use of aspartame in foods. He, in fact, went well beyond the standard used to remove Thalidomide from the marketplace.

Breast Cancer

Something quite remarkable happened during the aspartame feeding studies done by Dr. Morando Soffritti—something that prompted him to break with tradition, go beyond what John Olney had done and call publicly for an “urgent reevaluation” of the current guidelines for the use and consumption of aspartame. During a long term aspartame feeding study done by the industry-independent European Ramazzini Foundation headed by Dr. Soffritti it was shown conclusively that Sprague Dawley rats, which have the highest resistance to methanol toxicity of any laboratory animal, developed breast (mammary) and other cancers on a statistically significant and dose dependent basis when fed aspartame at low levels. Morando reports the results eloquently in his own words: “Our study has shown that aspartame is a multipotential carcinogenic compound whose carcinogenic effects are also evident at a daily dose of 20 milligrams per kilogram of body weight (mg/kg), notably less than the current acceptable daily intake of aspartame for humans.” The horror that Morando witnessed happening to his test animals was a partial reflection of what the aspartame-consuming world has been going through for “the last thirty years.”

The considerable evidence linking breast cancer to methanol is very convincing. There is good reason to believe that the epidemic of breast cancer over the last thirty years has more to do with the introduction of aspartame into the food supply than any other proposed cause. The production of breast cancer in laboratory animals fed aspartame, the increased rate of breast cancer in school teachers exposed to occupational methanol (see below), and the direct implication of high ADH tissue levels in those more prone to developing breast cancer over the course of their lifetimes goes a long way to prove methanol as an etiologic agent of breast cancer.

Why Is the Human Breast Particularly Sensitive to Methanol?

Methanol travels easily to breast tissue and has been found in human milk. The cells that produce milk within the breast, cells prone to develop the most common of breast cancers, adenocarcinoma, contain high concentrations of ADH enzyme, allowing methanol’s conversion to formaldehyde. Mammary epithelial cells have no way to protect themselves from formaldehyde—no means to render it harmless. They, unlike other breast tissue, contain no aldehyde dehydrogenase enzyme (ADH III) that could transform formaldehyde into the non-carcinogenic formic acid.

Of particular interest are recent findings implicating ADH as playing a pivotal role in the formation of breast cancer.
cancer, documenting a greater incidence of the disease in women with higher levels of ADH I activity in their breasts. These articles go on to indict acetaldehyde as the potential culprit. Remember acetaldehyde, as stressed in previous chapters, is essentially vinegar that results when ethanol is changed by ADH; it is a largely benign molecule with no link to cancer in humans. The role of acetaldehyde must be questioned when the same researchers report: “Very heavy drinking has not been associated with increased breast cancer risk relative to moderate drinking. We cannot explain why we have not observed an increase in risk from heavy lifetime consumption of alcohol.” What they are saying is that those who are constantly inebriated would have the cells in their breasts continuously producing acetaldehyde, thereby increasing the chances of breast cancer if acetaldehyde was indeed the culprit. This, of course, is not what actually occurs. Acetaldehyde is not the problem. Formaldehyde is the problem. The correct answer here is that the cause of breast cancer is not the ADH metabolite of ethanol, but rather the ADH metabolite of methanol. As you know it is ethanol in the blood that keeps the ADH busy and prevents it from producing formaldehyde from methanol.

**School Teachers Have Higher Risk of Breast Cancers**

In Chapter 9 we learned of the long term exposure of school teachers to methanol vapors from Ditto machines and the increase of autoimmunity that appears to be endemic in that group. Statistically significant evidence, developed from several different sources during large cohort studies, shows that teachers in the United States and Canada also have been at much higher risk of developing and dying from breast cancer than professional women in any other occupation. In a comprehensive study done of school teachers in British Columbia from 1950 to 1984, long before aspartame was allowed into carbonated beverages, it was shown that school teachers had a 70% greater chance of dying from breast cancer than women in other occupations. A study done at the Yale Cancer Center of breast cancer incidence in Connecticut women found only one occupational group of women with a statistically increased risk of breast cancer. That group – teachers – had twice the chance of developing breast cancer even after adjustment for other major breast cancer risk-factors.

An article entitled High breast cancer incidence rates among California teachers, published in 2002, found that “teachers have long been suspected to be at high risk of breast cancer.” In a mortality study done between 1979-1981, the California Department of Health Services noted a substantial excess mortality from breast cancer in California teachers. A large cohort study of over 133,000 California female teachers showed these women were statistically more likely to develop invasive breast cancer, in-situ breast cancer, and localized invasive breast cancer than other female professionals with 51%, 67% and 65% respective greater risks. The Yale study cited above points out that increased breast-cancer risk among teachers is “consistent with most earlier studies.” It continues, “it is currently unknown, however, what factors may explain the observed increase,” however, “considering teachers represent one of the largest single occupational groups among employed US women, further investigation of this association is warranted.”

**Aspartame Causes Breast Cancer in Human Feeding Study Done by Searle**

Remember from the introduction that in the summer of 1983 I spent time in a cramped conference room in Washington, DC that housed the docket file and application made by the G.D. Searle company to the US FDA to allow aspartame into carbonated beverages. A nameless FDA employee handed me one of the files he had been reading. Staring straight into my eyes he said in a whisper, “Did you see this?” I was taken aback, but before I could ask any questions he had disappeared, just as other such angels had into the endless corridors of that bleak federal building.

The study he handed me was damning evidence of the danger aspartame posed to the public. It was a long-term human study of diabetics who consumed 6 capsules daily containing a total of 1.8 grams of pure aspartame for 13 consecutive weeks. This dosage of aspartame is tantamount to taking 200 milligrams of
pure formaldehyde daily. This was the equivalent to the daily consumption of the amount of aspartame to be found in between three and 5 liters of diet cola. The subject base consisted of a total of 77 individuals, only 39 of which were aspartame consumers, while the others were controls. As a prerequisite to being accepted into the study, each participant had to pass a through physical examination that included considerable blood work. It was a double blind study, so neither the subjects nor the researchers had any idea who was taking the aspartame capsules or who was taking the placebo until the end of the experiment or until the subject was dropped from the study or died. Yes, one of the aspartame-consuming subjects died, while two others from the aspartame group developed cancer (one of these was breast cancer) during the study. 

Three subjects had to be dropped from the study before the 13 weeks ended. The culls had three things in common: they were female, they were dropped due to disease developed during the study, and they were all from the group that was consuming aspartame. None were dropped from the control group. The one death resulted from a stroke experienced after 8 weeks of aspartame consumption. The remaining two women survived 11 weeks on aspartame before developing epithelial cell cancers, one of the lining of her stomach, with hyperplasia reported in her lymph nodes during her pathology. The other woman’s breast cancer resulted in a mastectomy and she was diagnosed with adenocarcinoma. Both breast adenocarcinoma and hyperplasia had been found in laboratory rats fed aspartame during the testing of the product by Searle. 

This damning animal carcinoma information tragically was kept from the pathologist who reviewed the tissue samples from the two women who developed cancers during the study. In the final report of the results of the feeding study it states that the reason these human cancers were ignored was because there was no instance of carcinogenicity in animal feeding studies. To quote the report, the cancers were considered “coincidental … in view of information showing no evidence of such carcinogenesis in animals who received large amounts of Aspartame over a prolonged period.”

The pathologist’s report was sent directly to the G.D. Searle company. He refers to viewing slides of breast and stomach tissue from the long term rat studies done by Searle. It is obvious that the cancerous tissue slides were kept from him. To my most profound horror, the executive summary of the human diabetic study concluded, “In summary, aspartame appears to be well tolerated by diabetic subjects as a safe sugar substitute.” The rationale used to ignore the fact that fully 8% of the Aspartame consumption subjects, developed epithelial cancer during the study was that “no such cancers were seen in the numerous animal studies.” The Bressler Report, which resulted from an audit of all of Searle’s aspartame experimentation by a group of FDA investigators, reports that this was a lie, and that breast cancer was indeed an outcome of the animal feeding studies.

There are only 2 ways to get Formaldehyde into the Human Breast

The cancers found in the diabetic women should never have been ignored. Such a high mortality and cancer rate in a human study should have raised red flags and prevented aspartame from being rushed into the market against the clear intent of the US food law, which prohibits cancer causing additives.

The Scene of the Crime

The only reliable way that formaldehyde can reach the mammary tissue, aside from purposely injecting formaldehyde solution into the breast, is to administer the formaldehyde as methanol.
I don’t always remember to bring my reading glasses with me when I go to professional offices, but only once did I not regret the oversight. Since my only chore on this occasion was to pick up a good friend who had been deposing another attorney at the offices of that attorney I had no intention of spending much time in the waiting room. As fate would have it, the deposition was taking longer than expected and the receptionist said that I could make myself comfortable in the lounge. Without my spectacles I had to look through the pile of surprisingly up-to-date periodicals for something that might have lots of pictures – big pictures. I found a favorite, *Time Magazine*, that looked as if it would fill the bill and sat down to make myself as comfortable as I could in what was essentially enemy territory.

I quickly came to a two page fold-out map of the world, big enough to easily make out the countries. From the shading I was certain I was looking at a recent full color rendition of one of Dr. Kurtzke’s Multiple Sclerosis disease distribution maps that can be found in the second of the slide shows in Chapter 9. It would be great news if *Time Magazine* was doing an article on the MS epidemic, I thought. I returned to the receptionist and told her my story and she not only found me a pair of magnifying glasses, but she also gave me the magazine. I was awestruck by what I could finally see clearly. Here was a feature article about the epidemic of breast cancer, not MS. How could I have missed this epidemic? One look at the disease distribution throughout the World (Figure 11.3) and I knew I was looking at what would turn out to be the twin sister of MS. Breast cancer was a disease of well-to-do northern women, a disease of “affluence,” that was now spreading quickly to their poorer sisters in the warmer climates – all coinciding with the timing of the popularity of aspartame. I shook with a strange mixture of rage and excitement as I vowed to learn all I could about this epidemic of breast cancer.

**Figure 11.3**

*Distribution of Breast Cancer 2007*
It turns out that breast cancer is no stranger to women suffering from multiple sclerosis. Those afflicted with MS have over a twenty percent greater chance than women in the general population of developing breast cancer. This increased risk cannot be attributed to reduced parity or delayed first child birth, and is an increased risk that the authors of the study who found the association admit it is “consistent with previous observations and remains unexplained and warrants further attention.”

A quick look at Figure 11.3 will bring back memories of Chapter 9 on multiple sclerosis. The darker shades of grey in the map of Figure 11.3 indicate higher incidence of breast cancer. Note the abundance of countries that seem to have matching high or low incidence of both diseases, particularly in the most recent MS distribution chart. Take note particularly of Argentina and how that little country was so quick to become a nation committed to consuming aspartame.

Breast Cancer Increases as Does Popularity of Aspartame

Figure 11.4 illustrates the SEER numbers for the incidence of breast cancer in the United States during the ramp up of aspartame in the US diet. The tight relationship of the two curves is indicative of a relatively short time period between methanol consumption and breast cancer development. However, it is important to note that methanol began increasing in the US diet with the growth in consumption of aseptic canned juice drinks some years before aspartame’s official introduction. This was also a period with an increase in breast cancer screening, which may account for the meandering of the incidence curve between the years 1987 to 1994. Implementation of more accurate screening methods will only temporarily appear to increase incidence with a sudden rise for a period of time and then a snap back a few years later. A good example would be a haystack that contains only so many needles, and whether you count them by hand, which may take a long time, or use a magnet to gather them more quickly, the end results is always the same number of needles. This improved screening effect has been used to explain away the apparent incidence increase in breast cancer over “the last 30 years,” but the truth is that it cannot fully account for the relentless rise in the cumulative breast cancer rate since the introduction of aspartame.

It can be shown that the incidence of breast cancer has increased over the last 30 years, and particularly in populations exposed to aspartame. The uneven distributions of both MS and breast cancer throughout the world show a disarming similarity, including the unexplained increases to epidemic proportions in the same regions, such as Japan, over that short period of time. Figure 11.4 shows a disconcerting and abrupt rise in the incidence of breast cancer coinciding exactly with the ramp up of aspartame consumption after it was approved for sweetening carbonated beverages in the United States. The breast cancer rate of the

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other countries throughout the world, even in countries with normally low breast cancer rates, also appears to have risen soon after aspartame was introduced into their diets.

Genetic Distribution of Slow ADH Verses Fast ADH Linked to Breast Cancer Risk

In Chapter 7 we discussed the discovery that three different genetically defined versions of ADH I exist. They vary based on the speed at which they metabolize ethanol. Our genetic makeup determines which one of these is distributed throughout our bodies. The fast-ADH is 2.5 times faster at removing ethanol from the bloodstream than the slow-ADH. When it comes to heart disease, individuals who are genetically endowed with the slow-ADH that metabolizes ethanol 2.5 fold slower than the fast-ADH have a 86% reduction in risk from developing myocardial infarction – but only if they consume ethanol on a daily basis.

Interestingly, the role of ADH metabolism in the development of breast cancer mimics exactly its role in the development of heart disease. Women who are genetically endowed with the slow-ADH have less of a risk of contracting breast cancer during their lifetime than those that remove ethanol more quickly from their blood. This remains true even when both groups consumed the same amount of alcohol on a daily basis. Fast metabolizers who drank 15-30 grams of alcohol a day had a 2.3 greater risk of breast cancer even than non-drinkers who were slower metabolizers. The only explanation for the advantage to having an ADH that more slowly removes ethanol from the blood would be the ability of these individuals to maintain low levels of ethanol in the body fluids for longer periods of time, thus preventing the ADH in their breast tissue from converting methanol into the powerful carcinogen formaldehyde.

The U-shaped curve of ethanol’s protective effect against breast cancer has not yet been elucidated, and the present mindset of the breast cancer community may never allow for such a study to be performed. Time will tell, but this information would be vital before we could make a recommendation as to safe levels of ethanol consumption. The literature clearly indicates that the beneficial effect of ethanol for women is limited to one standard alcoholic drink a day, and no more. One issue that must temper the enthusiasm of all of us who consider low dose ethanol beneficial is the results of one large study that claims to show an increase in breast cancer among women who drank.

Avoiding methanol consumption appears to be the best way to help prevent breast cancer (see Chapter 2).

Exposure to Organic Solvents and Breast Cancer

An article published in 1997 stresses that the incidence rates for breast cancer have increased steadily in both the US and Canada over “the last 25 years.” The author points to the increasing number of women in the workforce and the possibility that the problem may be caused by occupational exposure to hazardous agents. No such increase was seen during the entire Second World War, when women, in mass, replaced their solider husbands in heavy industry. That aside, the author has some insight into the sensitivity of breast tissue to solvents such as methanol. He reminds us that solvent molecules quickly reach the breast epithelium cells, where they cannot be detoxified effectively because of the reduced activity of the “necessary enzymes” (aldehyde dehydrogenase) in breast cells. Second, because of the unique physiology of mammary glands, solvents or their “metabolites” accumulate in the milk ducts long enough to exert detrimental effects locally.

Cigarette Smoking with a U-Shaped Twist

Women with a history of having smoked are more likely to develop breast cancer than those who never smoked. It has been shown that a consistent dose-response enhances the plausibility of an increased breast cancer risk due to cigarette smoking. Most intriguing of all is that women who start smoking as teenagers and continue to smoke for at least 20 years have a statistically significant (50% greater) chance of developing...
breast cancer compared with never smokers. This association, however, only applies if the women studied were nondrinkers of alcohol. Drinkers who smoke showed no significant increased in breast cancer incidence in the same study.\textsuperscript{191} This appears to be an indication of the protective effect of ethanol against methanol’s conversion to formaldehyde.

**Cancer of the Kidney (Renal Cancers)**

**Kidney: a Target Organ of Methanol Poisoning**

Early pathology of humans who die from methanol poisoning consistently shows considerable gross anatomical and histological damage to the kidneys and the lungs.\textsuperscript{416} These organs both have extra hepatic stores of ADH I. The adult kidney contains ADH in measurable quantities.\textsuperscript{640} The exact location of these ADH rich tissues coincides with the epithelial tissue of the kidney, which remarkably is where the cancers in question originate.\textsuperscript{636}

Cigarette smoking has been the target of 24 investigations looking into its contribution directly as a cause of renal cell carcinoma. A Meta-analysis of all 24 of these studies published in 2005 shows a strong dose-dependent increase in risk associated with the numbers of cigarettes smoked per day and a substantial reduction in risk for long-term former smokers. For those who smoked over one pack of cigarettes a day the risk of developing renal cancer was about twice that of non smokers.\textsuperscript{652}

Spontaneous carcinomas of the kidney are extremely rare in laboratory rats. During the Ramazzini Institute study described above, male and female rats that had been fed aspartame developed statistically significant and dose related increases in kidney cancers. Twenty one carcinomas of the renal pelvis developed in their study whereas the control rats had none.\textsuperscript{50} The generation of cancers of the kidney, liver and lungs by aspartame was intrusive and unexplainable except by the unpopular formaldehyde theory. The results for all three cancers were not only highly significant but strongly dose related, showing that stronger doses were having a greater cancer-causing impact.\textsuperscript{50} The real tragedy here is that similar cancers were generated in test animals by the company that invented aspartame 30 years before the Ramazzini study. The data was kept out of sight of the FDA until the Bressler investigation,\textsuperscript{197} which came too late and fell on deaf ears.\textsuperscript{39} This becomes remarkably important immediately upon the visualization of the human experience with identical cancers exploding in the US population in the years since aspartame

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entered the food supply. Kidney cancer incidence rates, for instance, have inexplicably doubled “in the last thirty years.”

I have plotted the Centers for Disease Control SEER data for the US in the customary format in Figure 11.5. A Journal of the American Medical Association article entitled Rising Incidence of Renal Cell Cancer in the United States, published just months ago, unsuccessfully attempts to link the soaring rate of these adenocarcinomas (remember this term) to increased detection techniques. Its authors admit in their conclusion that increased detection alone “does not fully explain the upward incidence trends of renal cell carcinoma. Other factors must be contributing to the rapid increasing incidence of renal cell cancer in the United States, particularly among blacks.”

**Kidney Damage beyond Cancer**

The constant production of formaldehyde from methanol within the epithelial cells of the kidney as a result of consuming diet soda may be responsible for other damage beyond cancer. Aspartame has been shown to damage the kidneys of fetal rat pups whose mothers were fed it during pregnancy. We can learn something from the autopsies and histological workups done to those who have died from acute methanol poisoning. The tubules of the kidneys of these individuals often reveal extensive degeneration. Formaldehyde is a fierce deactivation agent for a number of enzymes, and even the mitochondria, which plays such an important role in kidney function, is itself extremely vulnerable to formaldehyde’s presence. It has been convincingly shown that heavy cigarette smoking more than doubles the risk of Chronic Renal Failure. A recent study done at Harvard Medical School looking into the possible association between consumption of sweetened beverages with kidney function decline showed no such relationship, but the collected data inadvertently resulted in the conclusion of the report that more than two servings of diet soda a day is responsible for a statistically valid two fold increased risk for kidney function decline. One liter of diet beverage, sweetened with aspartame, contains the same amount of methanol to be found in the average pack of unfiltered cigarettes.

**Melanoma and Cancers of the Skin**

*The Incidence of Skin Cancer Is Increasing at an Alarming Rate*

“The incidence of skin cancer has reached epidemic proportions. Only through heroic efforts by health care professionals and the general public to prevent the development of progression of skin cancer will this epidemic be abated.”

This quote from The Epidemiology of Skin Cancer, an article published in the Journal of Dermatological Surgery in March of 1996, indicates that this increase in skin cancers had been going on for the previous 25 years. What makes this particularly confounding to dermatologists is that although nonmelanoma skin cancer is directly related to cigarette smoking, the nonmelanoma skin cancer incidence has climbed steadily since the end of the of the late 1970s during the same period of time in which cigarette smoking has steadily declined. The explanation tying this cancer increase to the thinning of the ozone layer can apply well only to sparsely populated segments of the Earth that lie close to the world’s polar extremes. For all other areas the ozone layers have healed with no timely response from the skin cancer curves.

**Skin Fibroblasts Are an ADH I Stronghold**

Dr. Nelson Novick, whose patient developed macrophage-filled deep skin lesions (granulomatous panniculitis) over her lower body after consuming aspartame proved that it was indeed aspartame consumption that caused that serious skin reaction. The skin contains numerous fibroblasts that contain ADH I. This ADH will
convert methanol to formaldehyde as easily as the ADH found in the other tissues we have discussed. The reaction that Dr. Novick’s patient had was an inflammatory response to something produced in her skin from the aspartame, a substance made up only of two amino acids and methanol. Fibroblasts, when activated, are associated with cancer cells at all stages of cancer progression. Their functional contributions to cancer production may emerge to have more to do with this unappreciated production of formaldehyde from environmental methanol than anything else. In the mean time, what better mechanism to increase the incidence of skin cancer than the production of formaldehyde directly within the skin?

**Formaldehyde from Aspartame**

The confirmation of aspartame’s formaldehyde in human skin is important to our present discussion on aspartame skin issues. Though methanol has been known for almost a hundred years to cause several forms of dermatitis, the descriptions vary widely. Individuals who are extremely sensitive to formaldehyde can develop contact dermatitis reactions to a minute amount of it, such as that found in newsprint. Dermatologist have used this “exquisite sensitivity” of the human immune system to formaldehyde to show compellingly the connection of aspartame consumption to formaldehyde formation within skin tissue.

Two professors of Dermatology from the University of Kansas Medical Center, in a case report titled *Systemic Contact Dermatitis of the Eyelids Caused by Formaldehyde Derived from Aspartame*, published in the journal *Contact Dermatitis*, were the first to report eyelid dermatitis caused by aspartame consumption in one of their formaldehyde sensitive patients. Since then, “severe systematized dermatitis” has been confirmed to be linked directly to aspartame consumption in patients who have tested positively for formaldehyde sensitivity. It is interesting to note that positive reactions to formaldehyde on patch testing have now repeatedly been linked to both “systemic contact dermatitis” and a new syndrome called “aspartame-induced migraines,” often presenting in the same patients. In the most recent article confirming the positive link between formaldehyde sensitivity, systemic dermatitis and migraines you may find the published rebuttal letter from the manufacturer of aspartame, Ajinomoto, both enlightening and entertaining.

**Young Women Take the Burden of the Epidemics of Two Skin Cancers**

Dermatologists from the Mayo Clinic reported in the *Journal of the American Medical Association* (JAMA) in August of 2009 that...
they had been keeping track of the incidence of Basal Cell Carcinoma in their home county of Olmsted in Minnesota since 1976. They documented and histologically confirmed that since exactly 1980 the incidence of that non-melanoma cancer steadily increased by 400% (see Figure 11.6). This county, in which Rochester is a major city, is halfway between the North Pole and the Equator – far from any ozone hole and in an area with average daily sunshine saturation.

The most sobering aspect of the research reported in JAMA is that women younger than 40 years were at a significantly greater risk than their male counterparts. The exact quote is “This incidence of skin cancer is significantly higher for women than for men. A trend toward higher incidence of basal cell carcinoma in young women than in men has been reported by others.”

By far the most deadly form of skin cancer is melanoma, the origins of which are controversial. In fact, it appears to be much less related to UV light exposure than traditionally thought, and in fact is often found in areas of the body where scant exposure to the sun’s rays would be expected. The year before the JAMA article was published another much more ambitious study performed by investigators from the National Cancer Institute’s (NCI) Division of Cancer Epidemiology and Genetics studied the incidence patterns of invasive cutaneous melanoma throughout the United States. Figure 11.7 shows the change in incidence of melanoma for the population as a whole in the years since the introduction of aspartame. The study done by the NCI and published in the Journal of Investigative Dermatology broke down the original data to look for trends based on age and sex. I will let the National Cancer Institute’s results speak for themselves. “Invasive cutaneous melanoma, the deadliest form of skin cancer, increased among Caucasian women in the United States aged 15 to 39 by 50 percent between 1980 and 2004.” Further, they conclude, “Our analysis of SEER data suggests that melanoma incidence is increasing among young women.”

The outcome again goes to our premise that methanol in food will always have a more devastating effect on women due to their reduced ability to detoxify methanol in their guts. This is compounded considerably by the poorly kept secret that young women are the prime target of multimillion dollar diet soda advertisement campaigns, which we can presume have been very successful.

It is a hot summer day on a lovely Mexican Pacific coast beach. A beautiful young person is lying on the white sand renewing her tan before she hits the surf just one more time before lunch. She reaches into the cooler and pulls out an ice cold diet cola to quench her thirst. The soundtrack now switches to the unforgettable theme from Jaws. The
aspartame in the carbonated beverage starts to turn to methanol before she can empty the can. By the time she hits the beach again after lunch, the methanol is already turning into formaldehyde in the fibroblasts of her skin. Fibroblasts are known to have a pivotal, yet little understood role in cancer formation. Her skin is already under attack from the very short UV radiation from the equatorial sun. Does this sound to you like the makings of a perfect surfer movie or a perfect storm?

Cancers of the Liver and Lungs

The Liver: The Ultimate Source of ADH

Alcohol Dehydrogenase Class I (ADH I) is also called Human Liver Alcohol Dehydrogenase because it was originally discovered in – and is still mistakenly thought by many to only be found in – the liver. In fact the highest concentration of ADH I in the body is in the liver and this is what may account for the considerable carcinogenic stress on that organ from aspartame consumption. During the aspartame feeding studies of Swiss mice by the Ramazzini group, results showed a significant dose-related increase in both liver carcinomas and adenomas.

We have said many times that the liver contains large quantities of protective enzymes such as aldehyde dehydrogenase that help protect it from formaldehyde. However, since cancer is a statistical disease that only requires one molecule of formaldehyde in the wrong place at the wrong time, it is probably the number of molecules of formaldehyde produced in the liver that will eventually work against a heavy aspartame consumer. When we look at the tripling of the incidence rate of human liver cancers in the US during the aspartame years (see Figure 11.8), nothing more needs to be said.

What about the Lungs?

One cannot come away from the study of methanol without having some very serious concern about the lungs. All the autopsies in the older literature of humans killed by methanol ingestion always point to oddly congested or hemorrhaging lung tissue. Often lungs at autopsy show marginal emphysema with petechial hemorrhages. Again we have a significant dose-related increase of alveolar/bronchiolar lung cancers in the aspartame feeding studies done by Dr. Soffritti. Why? Lung tissue contains fibroblast tissue with a heavy burden of ADH that is ready and able to turn methanol into formaldehyde. During our experiments with pregnant rats fed methanol, we often autopsied pups with lung damage, as can be seen in the slide show from Chapter 12 (see www.whilesciencesleeps.com, Chapter 12 slideshow, “Birth Defects Caused by Aspartame”).

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Cigarette smoking has a direct and undeniable correlation and dose relationship to lung cancer. Most scientists would find it difficult to associate lung cancer or Chronic Obstructive Pulmonary Disease (COPD) with a food product. In the last thirty years, however, a considerable decrease in smoking in the United States has not been paralleled by a degree of decrease in lung cancer as was expected. Instead, the incidence of fibroblast centered lung adenocarcinoma has increased. The incidence of COPD has more than doubled, and in the last ten years the mortality from COPD has gone up over 20%. Could this all be related to the methanol from aspartame? I just don’t know; however, I can say with certainty that a lung bombarded daily by methanol-placed formaldehyde should be under suspicion for such developments. There would be good reason for serious consideration of methanol’s role in these disorders.

The Strange Case of the Female Thyroid Gland

During the Bressler investigation of the hidden aspartame toxicity files, one of the tumors that were recovered from those “lost” from Searle’s animal study of aspartame’s safety was of the thyroid gland of one of the experimental animals. Since that time a methanol vapor exposure study of Sprague Dawley rats of both sexes noted in particular that “The thyroid gland in females appeared to be a target organ for methanol.” This all would have been of little import had it not been for a Swedish study that identified, in particular, school teachers who were in the workforce between 1971 and 1989 as having a significantly increased incidence of cancer of the thyroid. The California teachers study that we used to point out the considerable increase risk of breast cancers gives the teachers they studied a 16 % greater chance of developing thyroid cancer.

We cannot do much more with this limited data. I was able to procure from the CDC SEER data thyroid incidence data that separated out the sexes. When graphed in the usual fashion, the data produced Figure 11.9, which does indeed seem to indicate that the thyroid gland may indeed be an organ sensitive to methanol that has been overlooked by those who have searched for sites of ADH activity.

Limitation of Animal Studies

I have mentioned a number of times now that animals are not an acceptable venue for testing methanol safety. Only humans will suffice for testing methanol. We have been very fortunate to have good scientists, such as Morando Soffritti and his dedicated staff at the Ramazzini Institute in Italy, who are willing to spend the extraordinary
time, effort and money to do long term animal studies. We must make the most out of it. To do that we have to understand the limitations of the species with which they are working. Rats and mice are not only between one and two hundred times less sensitive to the acute lethal effect of methanol, but also are blessed with a powerful enzyme, catalase, that protects them in ways that we don’t understand from its many toxic and disease outcomes. To use the results of their testing we must take seriously any disease outcome that their experimental animals exhibit in excess of the controls and allow the experimenters to use, at the very minimum, a hundred fold greater dosage concentration of methanol than the equivalent dose to extrapolate to humans. Even at this level, some human diseases or cancers will never become apparent in these poor surrogates, no matter how long the study.

The only appropriate test animal for methanol’s toxicity is the human. The innocents who have suffered and died to unknowingly test the safety of aspartame and, thus, methanol must not be allowed to have given their lives in vain.

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