## Aspartame and Drug Interaction

The following pages are taken from a chapter on aspartame and its interference with drug action: "*Aspartame Disease: An Ignored Epidemic*" by H. J. Roberts, M.D., F.A.C.P., F.C.C.P. www.sunsentpress.com

This is some of the most important information you could read on aspartame (NutraSweet, AminoSweet, E951, Canderel, Benevia, Naturataste, Spoonful, Neotame, etc.) because it can easily take your life if you are using prescription drugs.

More than 2 million people in the US with 100,000 fatalities suffer drug interaction, and I feel certain a great majority are due to the fact they are using aspartame. <u>https://www.worstpills.org/public/page.cfm?op\_id=4</u> I've been warning people for over two decades off this addictive, excitoneurotoxic, carcinogenic, genetically engineered drug, teratogen and adjuvant. It damages the mitochondria, the powerhouse of the cell and interacts with drugs and vaccines.

With the help of our Mission Possible chapters throughout the US and over 43 countries of the world, physicians, researchers, health organizations, attorneys, victims, MP activists, and responsible and knowledgeable media, and citizens we have become an army against this chemical poison.

While I read every label in a store I agree with the late Dr. H. J. Roberts who said its hard to avoid. Indeed, on a couple of occasions I've gotten it and broken out in urticaria (hives). Just recently I almost lost my own life from it. Even though I had educated many physicians and hospital personnel in a nearby hospital I was in, prescriptions were written for Zofran <a href="http://www.rxlist.com/zofran-drug.htm">http://www.rxlist.com/zofran-drug.htm</a> which turned out to have aspartame and Reglan <a href="http://www.rxlist.com/reglan-odt-drug.htm">http://www.rxlist.com/zofran-drug.htm</a> The physician who actually wrote one of the prescriptions himself had been warning about aspartame, and had no idea these Rx had the poison.

In the ER I broke out with urticaria and then stopped breathing. With life saving efforts I woke up from the cold air of the oxygen mask. It wasn't until I left the hospital did I realize what happened. I didn't get home until late and hadn't used medication, and yet was feeling fine. So I took the first drug, the Reglan and instantly felt the brain fog, pain, nausea, blurred vision, etc. I looked it up and there it was - aspartame. Now it was obvious why I had almost lost my life.

I had also noticed the constant ads on TV wanting those who had used Zofran for nausea in pregnancy and had babies with birth defects, cleft palate, etc. to contact attorneys. Again I found aspartame. This toxin had caused neural tube defects in original studies: autism, spina bifida, cleft palate. The way I found out is I contacted Jerome Bressler who retired from FDA some years ago to thank him for his Bressler Report, giving the facts on the fraud and shenanigans of the original manufacturer. At the time he didn't know me but was adamant that I find studies that were removed from his report. He said the public had no idea of the horrors to be sustained from this poison. I notified immediately Dr. H. J. Roberts and Dr. Russell Blaylock who both called Bressler. Dr. Roberts notified his congressman to get him the info the FDA had sealed from the public. The FDA wrote it was confidential.

I became friends with Jerome Bressler as did Lane Shore, Mission Possible Chicago, and visited him in a Chicago nursing home. It took 8 years to find the omitted data which I returned to the Bressler Report. Lane Shore gave it back to Jerome just before he died. <u>http://www.mpwhi.com</u> - front page banner.

Once the info was out and FDA could not hide the facts anymore, it was if they didn't care now and released more information to Dr. Woodrow Monte who then wrote the book, "*While Science Sleeps: A Sweetener Kills*". The last chapter on aspartame and autism is on his web site, www.whilesciencesleeps.com He explains the FDA had made a deal with the manufacturer, G. D. Searle, to keep this information from the public. Dr. Monte had warned on aspartame for years and his house was even blown up - with him in it. Fortunately he survived. Be sure to read this chapter and get the book. You won't be able to put it down.

Dr. Ralph Walton is now doing a study on aspartame and birth defects, so if you have given birth since 1984 please fill out the short form on www.mpwhi.com, second banner and email to Dr. Ralph Walton at rwalton193@aol.com "Someone" keeps removing this form from my web site so if you don't see it, please email Dr. Walton direct. This is very important. Just how heinous a crime when an agency of the government who is suppose to protect our health, hides the facts and allows autism and other birth defects to flourish throughout the world. Dr. Adrian Gross, lead scientist, testified against his own FDA to Congress, Senate, 1985 - "If the FDA violates its own laws who is left to protect the public?"

I don't want what happened to me, when I almost lost my life on a couple of occasions, to happen to anyone else. The chapter on aspartame and drug interaction is typed below and I'm asking for everyone who reads this to put it on web, email it to every physician you know, and every hospital, and every pharmacist. Don't let it stop until physicians stop prescribing drugs with aspartame. Remember the words of Dr. M. Alemany who did the Trocho Study which showed the formaldehyde converted from the methanol embalms living tissue and damages DNA. He told me personally in Barcelona, "Aspartame will kill 200 million people." Further notes at end of drug interaction chapter below.

Dr. Betty Martini, D.Hum, Founder, Mission Possible World Health Intl

# I. INTERFERENCE WITH DRUG ACTION

 The author's clinical observations indicate that aspartame products can alter the action of important drugs. They include coumarin (Coumadin<sup>®</sup>), phenytoin (Dilantin<sup>®</sup>), antidepressants, other psychotropic agents, propranolol (Inderal<sup>®</sup>), methyldopa (Aldomet<sup>®</sup>), thyroxine (Synthroid<sup>®</sup>), and insulin (Chapter XIII). This phenomenon is illustrated in many case reports presented in other sections.

It is also likely that some herbs interact with aspartame. Noting that numerous herbdrug interactions, Fugh-Berman (2000) stated, "Health care practitioners should caution patients against mixing herbs and pharmaceutical drugs."

# **General Considerations**

Aspartame may either reduce or potentiate drug action by various mechanisms. A few of the possibilities are listed

- •Alteration of the blood proteins to which drugs attach.
- •Alteration of drug receptors on cell membranes.
- Changes in the sites at which impulses are transmitted along nerves and to muscle.

•Metabolic abnormalities in the elderly that are known to enhance their vulnerability to drug reactions (Weber 1986). This problem increases in the case of persons taking multiple drugs ("polypharmacy") prescribed by several physicians.

•Interference with drug action by amino acids and protein. An example is the erratic therapeutic effects when patients with parkinsonism who were controlled on levodopa began to use aspartame products (Chapter VI-J). The antagonism if levodopa by dietary protein presumably reflects impaired transport from serum across the blood-brain barrier by neutral amino acids (Pincus 1986).

The <u>methanol</u> component of aspartame (Chapter XXI) might interact with compounds containing ethanol. Examples include the hypoglycemic sulfonylureas, metronidazole (an antibacterial drug), and allopurinol (commonly used in the treatment of gout). In his review of this subject, Posner (1975) emphasized that such interactions pose "an important area for careful clarification."

# Drug Reactions After the Cessation of Aspartame

The phenomenon of increased sensitivity to a drug after the removal of some interfering factor is known to clinicians. Examples include severe insulin reactions in diabetics after cure of an infection, and bleeding from coumarin after terminating a drug that influenced its binding to carrier proteins. This type of encounter probably reflects an increase in the "Free" forms of such drugs, It occurred, for example, when patients on maintenance coumarin or phenytoin avoided aspartame.

## A. Coumarin (Coumadin)

Coumarin is commonly prescribed as a anticoagulant ("blood thinner") for the treatment or prevention of serious problems caused by thrombosis (clot formation) and embolism (the migration of a thrombus). The vascular beds frequently affected include the coronary arteries, the carotid arteries, the inner lining of the heart (endocardium) in patients with heart attacks or irregular heart action (atrial fibrillation) from which embolism to the 7brain or lower extremities may originate, and veins in the lower extremities and pelvis that are frequent sites for pulmonary emboli.

This form of therapy constitutes a major commitment for physicians, particularly the need for continually monitoring the prothrombin time. Patients may bleed if the anticoagulant effect becomes excessive. Conversely, the loss of desired anticoagulation invites recurrence of thrombosis of embolism.

The likelihood of interference by aspartame was raised in patients who had been maintained on coumarin for extended periods with difficultly. Unexpectedly, their prothrombin times

approximated the control values (meaning a loss of anticoagulant effect), coupled with recurrent thrombophlebitis or angina pectoris.

### **Representative Case Reports**

## Case IX-I-I

An 82-year-old woman had received coumarin for two reasons: recurrent transient cerebral ischemic attacks ("small strokes") due to embolism from the carotid arteries, the heart, or both; and severe thrombophlebitis in the lower extremities with multiple attacks of pulmonary embolism. A registered nurse administered her medication daily.

The patient's prothrombin time, monitored every 3-4 weeks, had remained stable a long time. It was found to only 14 seconds (control, 13 seconds) on August 19, 1986. Several days thereafter, she developed severe pain in the lower extremities and exquisite tenderness over the deep veins, consistent with recurrent thrombophlebitis. The dosage of coumarin was increased.

On direct injury, it was determined that she recently began to use aspartame products. These were discontinued. Three days later, blood was noticed in the urine. At that time, her prothrombin time had returned to the therapeutic range - namely, 21 seconds. Coumarin was withheld several days. The failure of urinary bleeding to recur when coumarin was then resumed probably reflected a return of full anticoagulation after the loss of aspartame interference.

# Case IX-I-2

A 72-year-old woman had been treated with coumarin for longstanding angina pectoris and thrombophlebitis. Her prothrombin times generally ranged between 19.5-22 seconds. The patient then developed unstable angina pectoris and rapid heart action (tachycardia) for which she was hospitalized. No precipitating cause could be determined. At the time, little significance was paid to the slight decrease of her most recent prothrombin time (17 seconds). Even though the dosage if coumarin was increased, the prothrombin time then declined to 15 seconds.

The occurrence of other complaints suggesting aspartame disease raised the question as to whether aspartame also had influenced the prothrombin time. They included severe "burning and swelling" of the lips and tongue, headache, increased visual difficulty (despite recent cataract surgery and prescription glasses), marked "nervousness," and unexplained nausea. Although she initially denied using aspartame, the "diet" ginger ale she drank did contain it. The prothrombin time rose to 22 seconds (control, 12 seconds) one week after shopping aspartame. During this time, her intense fatigue and lip-tongue reactions subsided.

# B. Phenytoin (Dilantin) and Other Antiepileptic Drugs

Phenytoin is a key in managing epilepsy. When convulsions are associated with or aggravated by aspartame products (Chapter III), the patient confronts several dilemmas. First, the dose of phenytoin is likely to be increased, possibly to the point of toxicity (see Case III-35). Second, other anti-epilepsy drugs may be added, thereby increasing the potential for side effects. Third, the

continuation of these drugs in high doses could result in "rebound" toxicity after stopping aspartame.

The apparent potentiation of valproic acid (Depakene<sup>®</sup>; Depacote<sup>®</sup>), another antiepileptic drugs, was personally reported to the author by a 51-year-old man who drank considerable diet cola daily. When his physician increased the dose, he became comatose and required hospitalization.

### **Representative Case Report**

### Case IX-I-3

A 28-year-old man suffered seizures after a head injury while serving in the Marines. As a civilian, his convulsions unexpectedly increased despite multiple anti-epileptic medications, causing him to lose his job.

The patient's mother noted that he "acted like he was intoxicated." She expressed concern over his large consumption of aspartame sodas. Shortly after discontinuing them, he had to be taken to an emergency room for phenytoin intoxication - presumably representing the rebound phenomenon.

#### C. Antidepressant Drugs

Aspartame may interfere with the action of important drugs used to treat depression, particularly <u>imipramine</u> (a tricyclic antidepressant). Others have made similar observations.

Walton (1986) described a 54-year-old woman with recurrent major depression who had been well controlled on a maintenance dose of 150 mg imipramine at bedtime. She subsequently experienced marked behavioral changes, manic behavior characterized by inappropriate euphoria and flighty ideas, and a grand mal seizure. It was then learned that she had been drinking considerable aspartame-sweetened iced tea for several weeks prior to the seizure. All evidence of manic activity subsided within four days after stopping aspartame and the addition of lithium. Her depression recurred, however, two months later. Imipramine was resumed at the previous dosage with no recurrence of severe depression or mania over the ensuing 13 months.

The <u>monoamine oxidase inhibitors</u> (MAOIs), another group of antidepressant drugs, can have additional adverse effects when aspartame is consumed. These include phenelzine (Nardil<sup>®</sup>), isocarboxazide (Marplan<sup>®</sup>), and tranylcypromine (Parnate<sup>®</sup>).

It is pertinent that hypertensive crisis have occurred in patients so treated after they consumed foods and beverages containing tyramine and tryptophan. This response probably represents vasospasm caused by amino acid-derived sympathomimetic substances such as norepinephrine and tyramine (Chapters IX-C and XXIII). The serotonin-elevating action of <u>fluoxetine</u> (Prozac<sup>®</sup>) for treating depression could be counteracted by aspartame. It can block tryptophan entry to the brain, thereby inhibiting synthesis of serotonin (Chapter VII).

A Minnesota physician called to inquire about a possible interaction between Prozac and aspartame. He had suffered depression for which Prozac was prescribed, but without much benefit until he avoided aspartame. At what point, the drug proved helpful.

# D. Propranolol (Inderal)

The occurrence or aggravation of benign ("essential") tremor by aspartame was described in Chapter VI-J. This condition usually can be controlled with small or modest doses of propranolol. Several patients and correspondents stressed that their tremor intensified after consuming aspartame - even with increased dosage of propranolol - and improved when they avoided when they avoided such products.

# E. <u>Thyroid Replacement Therapy</u>

The increased susceptibility of hypothyroid to aspartame (Chapter IX-E) has been repeatedly emphasized. Conversely, there may be interference with the activity of thyroid replacement therapy.

# Case IX-I-4

An aspartame reactor had been taking thyroid hormone several years. After avoiding aspartame products, he noted "My replacement thyroid dose, which had been steadily increasing for the last few years, dropped by 30 percent. My doctor was quite surprised, and had no other explanation for it".

# F. Female Hormone Replacement Therapy

Patients with aspartame disease expressed firm convictions about interactions involving female hormone replacement therapy.

# **Representative Case Report**

# Case IX-I-5

A 40-year-old saleswoman consumed considerable diet sodas and other aspartame products, especially during the summer. She supplemented her description of reactions in the survey questionnaire with a two-page analysis of major changes when taken in conjunction with Premarin/Provera<sup>®</sup>. They included cloudy vision, dizziness, tenderness and tingling of the feet, joint pains, numbness of the lower limbs, headache, "foggy thinking," confusion, slurred speech and extreme fatigue. She took as many as 18 ibuprofen tablets daily to obtain relief of her constant pain. Extensive neurologic studies - including electromyography, nerve conduction studies, x-rays and MRI scanning - failed to revel a cause.

Considering a possible connection to the hormonal therapy, she reduced Premarin by half and quit Provera. There was considerable relief from the joint pain and muscle aches within two days, along with cessation of her neuropathic symptoms. After then avoiding all aspartame products, she wrote, "I felt IMMEDIATE relief within two days of what remained of the lumbar ache, muscle aches, foggy thinking, dizziness, and cloudy vision. All these improvements continue in the week since. Walking seems less of an effort; hip pain improved."

# G. Methyldopa (Aldomet)

There have been references to enhancement of seizures and other disorders in patients receiving methyldopa hypertension who also consumed aspartame. Seven of the initial 397 reactors completing the questionnaire were taking this drug when they experienced aspartame disease. Maher and Kiritsy (1987) demonstrated that aspartame administration decreases the entry of methyldopa into rat brain.

# **Representative Case Report**

# Case IX-I-6

A 67-year-old retired hypertensive woman had been treated with methyldopa. She experienced three unexplained seizures while drinking one can of diet cola and eating various aspartame puddings daily. The convulsions stopped when she avoided aspartame products, as did her sensitivity to noise and attacks of rapid heart action.

# H. Analgesics

<u>Lidocaine (Xylocaine</u><sup>®</sup>) is an important drug used for local anesthesia and the treatment of ventricular arrhythmias in intensive care units. Alterations of its pharmacology by aspartame require study.

Kim et al (1987) reported that the intraperitoneal administration of aspartame significantly reduced the 50 percent convulsion dose of lidocaine. They indicated that PKU patients and asymptomatic PKU heterozygotes may be more sensitive to the toxic effects of this and relate3d local anesthetics.

Nikfar et al (1997) noted that aspartame increased <u>morphine</u> analgesia in the early phase (wherein saccharin had no effect), and further enhanced morphine analgesia during the late phase. The sister of heavy diet cola consumer related the apparent potentiation of morphine by aspartame while he continued drinking it in an intensive care unit after a heart attack. This interaction became evident when the aspartame was stopped, coupled with halving the insulin required for managing his diabetes.

# **BIOOD AND LYMPH NODE DISORDERS**

Bone marrow and lymph nodes, the organs chiefly responsible for blood cells, are highly vulnerable to toxic substances. For example, aplastic anemia and leukemia may occur after treatment with potent drugs, or exposure to pesticides and other chemicals in the environment (Roberts 1984, 1990b). Aspartame products could be another cause of disorders therein.

# A. Bone Marrow and Blood Changes

Aspartame disease may be evidenced by changes involving red blood cells, white blood cells and platelets. The aspartame reactors reported below and elsewhere evidenced either anemia, a striking elevation of the white blood cell count suggestive of leukemia, or markedly decreased platelet counts (thrombocytopenia).

• A patient with recurrent "histiocytic leukemia" following aspartame ingestion (Case IX-J-1) offers an intriguing clue to the so-called histiocytosis syndromes in children (The Lancet 1987; 208-209). The Langerhans cell in histiocytosis is a dendritic cell derived from the bone marrow.

• Many drugs and other substances have been implicated in "immune thrombocytopenia."

• Case IX-F-6 mentioned the finding of "enlarged red blood cells." Deficiency of folic acid or its altered metabolism (see below) causes macrocytic (large red blood cell) anemia, while deficient absorption of vitamin B<sup>12</sup> can result in a similar anemia.

• Patients with pernicious anemia may be more vulnerable to aspartame. For example, a 51-year-old woman with severe aspartame disease gave a history of treatment for documented pernicious anemia as her only significant past medical history.

# **Representative Case Reports**

# Case IX-J-I

A 10-year-old girl began consuming various aspartame products at the age of eight, initially during summer weekends. She developed marked swelling of one shoulder which then involved the neck. Her arm almost tripled in size. There was no history of allergies or aspirin use existed. The patient also evidenced a high fever, pleural effusion (fluid in the lung cavity), striking enlargement of both the liver and spleen, and a precipitous decline of the platelet count to 1,000 per cubic mm (normal 150,000 or higher). A striking increase of histiocytes was found in her bone marrow. Several "liver enzymes" were markedly elevated - i.e., SGOT 3,080 units/L (normal, up to 50); CPK 30,000 units/L (normal, up to 50).

Numerous physicians and consultants saw this child. Most diagnosed histiocytic leukemia. The patient received large doses of prednisone.

Dramatic clinical improvement and virtual normalization of the foregoing blood changes occurred when the mother closely monitored her diet and eliminated additives. The prednisone was then stopped.

The patient subsequently ate several bowls of an aspartame cereal. Marked swelling of the cheeks developed, coupled with recurrence of the aforementioned features. When aspartame was discontinued, the swelling receded <u>without</u> prednisone.

Several months later, the girl was given aspartame chewing gum without the mother's knowledge. Swelling of her entire body, recurrent enlargement of the liver and spleen, a dramatic increase of bone marrow histiocytes, and severe pain in many joints ensued. Total abstinence from aspartame again effected the disappearance of her symptoms and the blood abnormalities within six months. At the time of my last discussion with her mother, the child had minimal enlargement of the liver, and was receiving prednisone in low doses only intermittently.

This patient had two sets of head x-rays, three CT scans of the brain, two spinal punctures, four bone marrow studies, two electroencephalograms, two heart monitoring studies, two barium enemas, and a host of other studies. Her mother estimated the medical costs at \$750,000!

### Case IX-J-2

A 62-year-old man developed severe gastrointestinal problems while ingesting aspartame products. He developed "an erratic blood count, with red and white cell imbalance, and platelets off some." He received "cortisone" for six months when his condition was diagnosed as "an immune deficiency problem."

His daughter suffered intense abdominal pain, a bleeding peptic ulcer, severe headache, and repeated grand mal convulsions when she used aspartame products. A granddaughter had phenylketonuria (Chapter XVII) at birth, and subsequently manifested severe learning deficiencies.

## Case IX-J-3

A 61-year-old personnel director began drinking diet colas at the age of 59, and consumed up to one liter daily. His platelet count declined to 54,000/cu mm thereafter. He also became concerned over "forgetting to perform things for which I was responsible." Other complaints included "double vision," severe sensitivity to light, headache, dizziness, two convulsions, "fits without convulsions," marked sleepiness during the day, insomnia, slurred speech, and a nonspecific rash. Extensive studies failed to uncover a major medical problem.

His symptoms improved within 10 weeks after avoiding aspartame, although some problems with vision, memory, sleep and "concentration" persisted. There was an **immediate** exacerbation during one retest trial. After avoiding all aspartame products, the platelet count had increased to 75,000/cu mm at the time of his subsequent correspondence.

### Case XI-J-4

A registered nurse developed hypertension and a platelet count under 30,000 during her third pregnancy. Neither of these features had been noted in previous pregnancies. She began using diet colas after the birth of her second child. Other symptoms included severe headache, depression, loss of hair, and symptomatic reactive hypoglycemia.

### Case IX-J-5

A 41-year-old woman consumed diet cola for 14 years. She experienced many symptoms of aspartame disease for three years, including headache, visual problems, severe aching of the

joints, numbness of the left hand, and easy agitation. Her white blood cell count was only 460 (normal 4000-10,000), for which no other cause could be determined.

### B. Lymph Node Enlargement

Enlargement of the lymph nodes (lymphadenopathy) occurred in several aspartame reactors without the finding of another convincing cause. This featured differed from the striking salivary gland enlargement in other with aspartame reactors (Chapter IX-D).

## **Representative Case Reports**

## Case IX-J-7

A mortgage broker began drinking diet cola in January 1995. She developed markedly enlarged glands in the neck ("the size of golf balls") one year later, along with a constant sore throat. These features persisted despite considerable doctoring. Subsequent symptoms included blurred vision, migraine headaches, severe joint and muscle pains, constant diarrhea with bloody stools, ringing in the ears, palpitations, night sweats, "a tongue as black as leather," a 30-pound weight gain, and "incapacitating" chronic fatigue. Various suggested diagnoses - including carbon monoxide poisoning, fibromyalgia, lupus erythematosus, and Epstein-Barr infection - could not be confirmed.

The patient was "absolutely in shock" on learning about aspartame disease. Improvement proved gratifying after discontinuing aspartame products. The tongue returned to a normal healthy pink state. The enlarged glands in her neck decreased within several months.

## Case IX-J-8

A 44-year-old loan officer experienced headache, drowsiness, hyperactivity, severe tingling, irritability, personality changes, palpitations, a recent elevation of blood pressure, abdominal pain, itching, hives, other rashes, marked frequency of urine, and joint pains. These symptoms were provoked "immediately" on three retesting trials with aspartame products.

His <u>son</u> had aspartame disease. It was primarily evidenced as "noncancerous lumps under the arms" and a rash.

## C. Altered Folic Acid Metabolism

Aspartame may deplete important nutrients related to folic acid. Folic acid assists in eliminating the formic acid derived from methyl alcohol degradation (Reitbrock 1971). It tends to decrease with higher methanol concentrations (Chapter XXI). In view of the rarity of low blood foliate levels in contemporary clinical practice, this finding could prove a clue to aspartame disease.

Aspartame-induced decrease of folic acid could have clinical significance, especially anemia and birth defects.

A physician in the State of Washington noted anemia in a patient who ingested large amounts of aspartame sodas, and suspected folic acid deficiency. He found the blood methanol level to be elevated. Unfortunately, the tube sent for a foliate level broke in transit after he started replacement therapy.

There has been a recent impressive decline in neural-tube following the fortification of grain products an vitamin formulations with folic acid, coupled with an associated increase of median serum foliate concentrations. This decline might be less evident, however, among pregnant women who consume aspartame products that can deplete foliate levels as methyl alcohol is metabolized.

# **K. LIVER DYSFUNCTION**

The vulnerability of patients with pre-existing liver disease to aspartame was noted in the introduction of this section. These disorders include hepatitis, cirrhosis (Case IX-K-5), hemochromatosis (iron storage disease), and the liver dysfunction complicating many infections and drug reactions.

Several aspartame reactors <u>without</u> known liver disease evidenced marked elevation of blood aminotransferases ("liver enzymes"). They are variously referred to as alanine aminotransferases (ALT; SGPT), aspartate aminotransferase (AST; SGOT) and glutamyl transpeptidase (GGT). (Serum levels of ALT and AST in normal persons are less than 35units/L. In Case IX-J01, a 10-year-old girl, the SGOT was 3,080 units/L. One such aspartame reactor had been told, "Your liver is pickled."

Monte (1984) made a reference to such an association. Taylor (1996) also raised the issue of liver toxicity caused by aspartame in a 51-year-old man who evidenced repeated jaundice.

### **General Considerations**

In a recent National Health and Nutrition Examination Survey, 2.6 percent of the United States population surveyed had elevated serum ALT for which no cause of chronic liver disease could be found (James 1999). Even though 70 percent of the adult population consumes aspartame products, aspartame disease was not suspected as a likely causative or contributory cause. It is of historic interest that Dr. Misael Uribe (1982) expressed concern that the FDA had not adequately considered the potential toxicity of aspartame patients with liver disease shortly after its forthcoming release was announced.

Persons with cirrhosis of the liver are at increased risk because they may be unable to metabolize aspartame and its breakdown products adequately (Jagenberg 1977; Herberer 1980; Dhont 1982).

The possibility of aspartame toxicity should be entertained in patients diagnosed as having **nonalcoholic steatohepatitis**. This liver disorder has become a common liver disorder in North America, and can progress to cirrihosis (James 1999). Its frequent features of persistent fatigue,

upper abdominal discomfort, diabetes and hyperlipidemia are also encountered in aspartame disease.

Obese persons and diabetic patients who consume considerable aspartame appear to be at higher risk for the development of chronic nonalcoholic steatohepatitis, especially when addicted to such products (Chapter VII-G). The term "nonalcoholic" is misleading in this instance because aspartame contains ten percent methyl alcohol.

Admittedly, it may be difficult to single out precise symptoms attributable to aspartame products in patients with liver disease. Yet, few physicians considered the contributory role of aspartame to hepatic changes among patients in this series. Some even were hostile to the idea (see Case IX-K-3).

By contrast, astute persons and various consumer groups submitted related observations to the FDA. The husband of one patient, an attorney, became irate over the absence of any wearing labels on aspartame products for persons with liver disease.

#### **Representative Case Reports**

#### Case IX-K1

A 68-year-old housewife described how she became "incapacitated" form aspartame products in these terms: "My eyes would not focus. I had confusion, dizziness, depression, insomnia and memory loss. I could not drive or leave the house alone." Other complaints included severe nausea, diarrhea, marked thirst, considerable frequency of urination, and attacks of hypoglycemia. Her "liver reading" (presumably the ALT) rose from 44 to 264. It declined to 78 after she avoided aspartame. Although her physician assumed it was due to "drinking," the patient empathically stated that she <u>never</u> drank alcoholic beverages.

### Case IX-K-2

A prospective blood donor was surprised, and angered, when a blood bank would not accept her blood (SGOT and SGPT) were markedly elevated. She consulted a gastroenterologist. He confirmed the finding, and by exclusion diagnosed hepatitis C infection. Abdominal ultrasound studies and other tests were normal. Aside from advice to stop her modest drinking, the only other treatment option offered was interferon. She declined the latter because of its inherent severe side effects.

Her teats remained unchanged over the next two years despite total abstention from alcohol. She would not kiss persons for fear of transmitting the presumed infection.

A friend commented that aspartame products could harm the liver. As personal proof, she pointed to the normalization of her own blood studies after discontinuing diet sodas. The patient thereupon discontinued such products - including the seven packs per day (!) of aspartame chewing gum. A dramatic decline of her liver enzymes ensued.

Overwhelmed by this experience, she reported her case to the FDA and criticized its approval of aspartame as "total disregard for human health and safety."

### Case IX-K-3

A man had consumed 24-60 ounces of diet sodas daily for a year while on "a very low calorie diet." He experienced numbness of his left hand, left foot, face, and then the right hand. Two MRI studies and other neurologic tests were normal. His liver enzymes, however, were elevated (GGT 350; SGOT 150); repeat testing confirmed these abnormalities. A CT scan of the liver and an ultrasound study of the abdomen were not revealing.

This patient was scheduled for consultation with a gastroenterologist. Learning about aspartame disease in the interim, he stopped aspartame products, and consumed considerable filtered water "in an attempt to flush my liver." Repeat liver tests evidenced progressive declines of the GGT to 15, and of the SGOT to 50. The gastroenterologist disregarded these observations, and criticized the patient's inference about aspartame being a possible cause.

#### Case IX-K-4

The 28-year-old manager of an architectural firm, and mother of a six-month child, had markedly elevated liver enzymes for four years, the cause of which eluded multiple physicians. Her AST, ALT and alkaline phosphates levels average 200, 200 and 1100, respectively. The bilirubin levels remained normal.

She never used drugs of abuse or smoked, and had not ingested alcohol for three years. No birth control pill was used since the age of 21. Numerous studies for hepatitis and HIV viruses, and for various auto-immune diseases were negative. In addition to multiple CT scans and an MR study, two liver biopsies proved normal.

The patient had "practically lived on" diet cola for ten years... "drinking more than water or any other beverage." This soda was avoided during pregnancy.

After reading about aspartame disease, she stopped all diet products. There was a prompt and striking decrease of all the enzymes, which progressively normalized with no other interventions.

## Case IX-K-5 (FDA Project #3898; Courtesy, Dr. Linda Tollefson)

A 76-year-old woman with longstanding cirrhosis died in progressive "liver coma." In his correspondence to a consumer advocate, her husband indicated that she had been "in pretty good health" until October 1983 when an elevated blood glucose concentration was found. Tolbutamide and diet were prescribed. In an attempt to avoid sugar, she used four packets of an ATS daily in coffee, cereal and desserts.

The patient evidenced dramatic deterioration within two weeks. It was manifest by indifference to her surroundings, forgetfulness, slurred speech, dizziness, impaired vision, loss of interest concerning food and her favorite television programs, and a change in gait. She made irrelevant

statements as if hallucinating. Her condition deteriorated to the point of becoming incontinent and bedridden. A diagnosis of "hepatic encephalopathy" was made.

## **Related Biochemical Aspects**

Deterioration of brain function in patients with severe liver disease has been attributed to the <u>retention of phenylalanine and other amino acids</u> (Fischer 1971). Striking changes in amino acid metabolism are known to occur in liver disease - particularly the hydroxylation rates of phenylalanine, tyrosine and tryptophan. The elevated phenylalanine concentrations in patients with cirrhosis rise significantly after protein consumption (Fernstrom 1979). <u>Methanol poisoning</u> (Chapter XXI) creates another serious problem in patients with liver disease. The enzymes alcohol dehydrogenase and aldehyde dehydrogenase are involved in methanol metabolism. The considerable alcohol dehydrogenase activity of the liver decreases when it is diseased.

One mechanism may be the production of tumor necrosis factor (TNF)-*a*. It is an early event in many types of liver injury that triggers the release of other cytokines, and then the destruction of liver cells.

A vicious cycle involving <u>aggravated hypoglycemia</u> also can occur. Contributory causes include the tendency to "hepatic hypoglycemia" among persons with severe liver disease (owing difficulty in storing and metabolizing glycogen), aspartame-induced hypoglycemia (Chapter XIV), the use of glucose-lowering drugs for control of diabetes, and the failure of diabetics receiving such drugs or insulin to take interval feedings. For example, the physician who prescribed tolbutamide for Case IX-K-4 "said nothing about diet."

<u>Hemochromatosis</u> (iron storage disease; hepatic iron overload; bronze diabetes) might be aggravated in its pre-cirrhotic stage by aspartame consumption (see Case XIII-1), especially through insulin stimulation (Chapter XIV). Related features of insulin resistance - in addition to hyperinsulinemia - are increased body mass, elevated lipids, and abnormal glucose tolerance.

The separate entity of primary hepatic iron overloads differs from genetic hemochromatosis because transferring saturation is normal, as is the frequency of the HLA A3 genotype (Ferrannini 2000). It has been suggested that insulin's primary action in stimulating glucose transport is coupled with a redistribution of transferring receptors to the cell surface where they mediate extracellular iron uptake.

Martini: This is the end of the chapter. Please also see the Aspartame Resource Guide: <u>http://www.mpwhi.com/aspartame\_resource\_guide.pdf</u>

We've all heard that Vice President Joe Biden's son, Beau Biden has died of brain cancer, and some years ago had a stroke. Keep in mind that one of the main issues in the beginning was that aspartame breaks down to diketopiperazine, a brain tumor agent that triggered brain tumors in original studies. Also a study showed that aspartame triggered myocardial infarctions and strokes. I do know that Beau Biden drank soft drinks but I don't know if he used those that contained aspartame. Maybe someone can let me know. There is a copy of Dr. Roberts medical text in the White House, it was personally delivered to Michelle Obama and put in her hands, although Dr. Roberts never heard from her. <u>http://www.mpwhi.com/h j roberts has died.htm</u> It goes into the brain tumor issue at length. Also in" *Excitotoxins: The Taste That Kills*" by Dr. Russell Blaylock there is much information on aspartame and brain tumors.

I really don't see how physicians practice medicine without these medical texts on aspartame by these brilliant and courageous doctors and researchers. They should be in every hospital and pharmacy.

Please keep this going around the world so physicians can find out why so many are dying of drug interaction. In the Aspartame Resource Guide there is information on a safe sweetener that can be used that has no chemicals, "Just Like Sugar".

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More info on aspartame on www.dorway.com, www.wnho.net, www.holisticmed.com/aspartame