

**COMMENTS BY THE NATIONAL HEALTH FEDERATION ON FOOD-ADDITIVE
PROVISIONS FOR ASPARTAME (AGENDA ITEMS NO. 4 & 5(A) AT CX/FA 10/43/4 &
CX/FA 11/43/7)**

In addition to its previous comments submitted at the 40th Session of CCFA in Beijing, China, the National Health Federation (NHF) respectfully submits the following current comments concerning Aspartame as a food additive:

1. Aspartame Contains Toxic Methanol

Methanol (methyl alcohol, wood alcohol), a poisonous substance,^{/1} is added as a component during the manufacture of aspartame.^{/2} The methanol is subsequently released within hours of consumption^{/3} after hydrolysis of the methyl group of the dipeptide by chymotrypsin in the small intestine.^{/4} Absorption in primates is hastened considerably if the methanol is ingested as free methanol ^{/5} as it occurs in soft drinks after decomposition of aspartame during storage or in other foods after being heated." Regardless of whether the aspartame-derived methanol exists in food in its free form or still esterified to phenylalanine, 10% of the weight of aspartame intake of an individual will be absorbed by the blood stream as methanol within hours after consumption.^{/6}

Methanol has no therapeutic properties and is considered only as a toxicant.^{/7} The ingestion of two teaspoons is considered lethal in humans.^{/8} Methyl alcohol produces the Methyl alcohol syndrome, consistently, only in humans and no other test animal, including monkeys.^{/9} There is a clear difference between "toxicity," which can be produced in every living thing, and the "toxic syndrome."^{/10}

The greater toxicity of methanol to man is rooted firmly in the limited biochemical pathways available to humans for detoxification. The loss of uricase (EC 1.7.3.3.), formyl-tetrahydrofolate synthetase (EC6.3.4.3.)^{/11} and other enzymes^{/12} during evolution sets man apart from all laboratory animals including monkeys.^{/13} As a result, there is no generally accepted animal model for methanol toxicity.^{/14} Humans suffer "toxic syndrome" at a minimum lethal dose of < 1 gm/kg, ***much*** less than that of monkeys, 3-6 g/kg.^{/15} The minimum lethal dose of methanol in rats, rabbits, and dogs is 9, 5, and 7 g/kg respectively"; ethyl alcohol is more toxic than methanol to these test animals.^{/16}

The United States Environmental Protection Agency in its Multimedia Environmental Goals for Environmental Assessment recommends a minimum acute toxicity concentration of methanol in drinking water at 3.9 parts per million, with a recommended limit of consumption below 7.8 mg/day.^{/17}

This report clearly indicates that methanol "is considered a cumulative poison due to the low rate of excretion once it is absorbed. In the body, methanol is oxidized to formaldehyde and formic acid; both of these metabolites are toxic."

In particular, the formaldehyde metabolite of methanol is a deadly neurotoxin. Yet, a one-liter aspartame-sweetened beverage contains about 56 milligrams of methanol. Heavy users of aspartame-containing products consume as much as 250 mg of methanol daily, or 32 times the EPA safety limit.

Some have argued that the food additive is no different in having methanol than natural fruits. Yet, the methanol found in fruits and vegetables is bound to pectin and takes it out of the body safely. Also, the methanol in fruits and vegetables is always accompanied by ethanol, which is the classic antidote to methanol toxicity.¹⁸ So, there is no comparison in toxicity between the naturally sourced fruits and the synthetic aspartame.

2. Recent Study Demonstrates Aspartame Toxicity

A very recent study conducted in India, showed that aspartame water given to rats for just six months caused noticeable liver harm.¹⁹ The Abstract²⁰ states that:

"The present study evaluates the effect of long term intake of aspartame, the artificial sweetener, on liver antioxidant system and hepatocellular injury in animal model. Eighteen adult male Wistar rats, weighing 150 - 175 g, were randomly divided into three groups as follows: first group was given aspartame dissolved in water in a dose of 500 mg/kg.b.wt; the second group was given a dose of 1000 mg/kg.b.wt; and controls were given water freely. Rats that had received aspartame (1000 mg/kg.b.wt) in the drinking water for 180 days showed a significant increase in activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP) and γ -glutamyl transferase (GGT). The concentration of reduced glutathione (GSH) and the activity of glutathione peroxidase (GPx), and glutathione reductase (GR) were significantly reduced in the liver of rats that had received aspartame (1000 mg/kg.b.wt). Glutathione was significantly decreased in both the experimental groups. Histopathological examination revealed leukocyte infiltration in aspartame-treated rats (1000 mg/kg.b.wt). *It can be concluded from these observations that long term consumption of aspartame leads to hepatocellular injury and alterations in liver antioxidant status mainly through glutathione dependent system.*" (emphasis added)

This recent study is just one more of many studies revealing the ill health effects of aspartame. The manufacturers of aspartame would have us believe otherwise with doctored studies that even the U.S. Food and Drug Administration harshly criticized until it was captured by the aspartame industry and changed its position.

3. For Safety Reasons, Codex Must Lower the ADI for Aspartame

A serious consequence of the mistake of not being aware of the dangerous metabolism of the methanol in aspartame is that when scientists calculated its ADI (Advised Daily Intake) they did not include the toxic properties of the methanol in their estimations and it was calculated incorrectly by a factor of 35. The current ADI is 40mg/kg (JECFA/Codex at 0-40 mg/kg). However, if one were to correctly take into account the ADI of methanol, then the ADI for aspartame should drop to 1.14mg/kg.

If Codex is about protecting the health of the consumer, then this body would be remiss in not revisiting its ADI numbers and setting them significantly lower so as to protect consumers from the toxic effects of methanol. The National Health Federation strongly urges this Committee not to advance aspartame along the path to approval as a food additive at all, and certainly not until all independent studies of its health effects have been taken into account.

¹ Windholz, M., *Merck Index*. 9th ed. Rahway, New Jersey: Merck & Company Inc. (1976).

² Searle Food Resources, Inc., *Sources and Metabolism of Aspartame and Representative Sweeteners* (1981).

³ Staples, R.E., Teratogenicity of Formaldehyde. Formaldehyde Toxicity. J.E. Gibson, Ed., Hemisphere Publishing Company pp 51-60 (1983).

⁴ Oppermarm, J.A., Muldoon, E. and Ranney, R.E., "Metabolism of Aspartame in Monkey," *J. Nutr.*, 103:1454-1459 (1973)

⁵ *Ibid.*

⁶ Staples, R.E., Teratogenicity of Formaldehyde. Formaldehyde Toxicity. J.E. Gibson, Ed., Hemisphere Publishing Company pp 51-60 (1983).

⁷ Hadden, L., et al., *Clinical Management of Poisoning*, Philadelphia, Pennsylvania: W. B. Saunders Company (1983).

⁸ Gosselin, R.E., *Clinical Toxicology of Commercial Products*, 4th ed. Gosselin, R.E., et al., eds., Baltimore, Maryland: Williams & Wilkins (1981).

⁹ Roe, O., "Species Differences in Methanol Poisoning," *CRC Critical Rev. in Tox.*, pp. 275-286, October (1982); Tephly, T.R., Watkins, W.D. and Goodman, J.I., "The Biochemical Toxicology of Methanol," *Essay Toxicol.*, 5:149-177 (1974).

¹⁰ Tephly, T.R., Watkins, W.D. and Goodman, J.I., "The Biochemical Toxicology of Methanol," *Essays Toxicol.*, 5:149-177 (1974).

¹¹ Roe, O., "Species Differences in Methanol Poisoning," *CRC Critical Rev. in Tox.*, pp. 275-286, October (1982).

¹² Goodman, J.I. and Tephly, T.R., "Peroxidative Oxidation of Methanol in Human Liver: The Role of Hepatic Microbody and Soluble Oxidases," *Res. Commun. Chem. Pathol. Pharm.*, 1(4):441-450 (1970).

¹³ Roe, O., "Species Differences in Methanol Poisoning," *CRC Critical Rev. in Tox.*, pp. 275-286, October (1982).

¹⁴ *Ibid.*; Wimer, W.W., Russell, J.A. and Kaplan, H.L., *Alcohols Toxicology*, Park Ridge New Jersey, Noyes Data Corporation (1983).

¹⁵ Roe, O., "Species Differences in Methanol Poisoning," *CRC Critical Rev. in Tox.*, pp. 275-286, October (1982); Wimer, W.W., Russell, J.A. and Kaplan, H.L., *Alcohols Toxicology*, Park Ridge New Jersey, Noyes Data Corporation (1983).

¹⁶ Roe, O., "The Metabolism and Toxicity of Methanol," *Pharmacol. Rev.*, 7:399 (1955).

¹⁷ Cleland, J.G. and Kingsbury, G.L., "Multimedia Environmental Goals For Environmental Assessment," U.S. Environmental Protection Agency: EPA-600/7-77-136b, E-28, November 1977.

¹⁸ See http://www.mpwhi.com/aspartame_methanol_and_public_health.pdf

¹⁹ RH Nair et al., Mahatma Gandhi U, *Food Chem Toxicol*, 2011.03.02: Rich Murray 2011.03.12 http://rmforall.blogspot.com/2011_03_01_archive.htm (Saturday, March 12, 2011). See also <http://www.ncbi.nlm.nih.gov/pubmed/21376768>.

²⁰ Abhilash M, Paul MV, Varghese MV, Nair RH, "Effect of long term intake of aspartame on antioxidant defense status in liver," *Food Chem Toxicol*, 2011 Mar 2. [Epub ahead of print] (School of Biosciences, Mahatma Gandhi University, Kottayam, Kerala, India, 686560; harikumarannair@hotmail.com, harinair@fastmail.fm)