BRAIN NEOPLASMS

ASPARTAME AND BRAIN TUMOR RISK

The potential link of aspartame to rising brain tumor rates was analyzed using 1975 to 1992 data from the US National Cancer Institute in a study at the Departments of Psychiatry and Biostistics, Washington University Medical School, St Louis, MO. The incidence curve of primary brain tumors for the years 1975 to 1992 is biphasic, phase I from 1975-1984 and phase II from 1985-1992. In phase I the increased incidence was small and unsustained, whereas in phase II a sharp increase in incidence was accompanied by greater malignancy. Aspartame was first approved in the US in 1981 and wider approval was extended in 1983. The authors propose that aspartame usage may be linked to the onset in 1985 of a sustained increase in rate of malignant tumors in the elderly population, or to the steady climb in incidence of various tumors in younger age groups beginning in 1987. The criteria invoked in judging the carcinogenic potential of environmental agents appeared to be met by aspartame: 1) aspartame has in vitro mutagenic potential related to its nitrosation to nitrosourea; 2) aspartame-fed rats have a high incidence of malignant brain tumors; and 3) an increased incidence of brain tumors in humans following the introduction of aspartame in the diet. (Olney JW, Farber NB, Spitznagel E, Robins LN. Increasing brain tumor rates: is there a link to aspartame? J Neuropathol Exp Neurol Nov 1996;55:1115-1123). (Respond: John W Olney MD, Department of Psychiatry, Washington University School of Medicine, 4940 Childrens Place, St Louis, MO 63110).

COMMENT. This detailed analysis of data pertaining to the potential carcinogenic effects of the artificial sweetener aspartame, conducted by scientists at an independent prestigious university and published after peer review in a recognized medical journal, warrants further assessment of the safety of this widely used dietary additive. Aspartame has been linked to precipitation of migraine headaches, and exacerbation of epileptiform discharges in the EEG of children with seizures. (Progress in Pediatric Neurology I and II, PNB Publ, 1991, 1994). Despite the controversy over the validity of these studies, aspartame is becoming a "food for thought" rather than ingestion, until its safety is reviewed by an unbiased panel of experts.

SEIZURE DISORDERS

CORTICAL HYPOMETABOLISM IN WEST SYNDROME

Serial PET scans and MRIs were performed in 18 infants with West syndrome (WS) to determine the relation between cortical hypometabolism and delayed myelination in a study at Nagoya University School of Medicine, Japan. All 8 patients with symptomatic WS showed hypometabolism at onset, with persistence in 5 at 10 months, whereas only 4 of 10 patients with cryptogenic WS showed hypometabolism at onset and none at age 10 months. Localization of EEG abnormalities at 10 months correlated with the region of hypometabolism in only 2. MRI showed delayed myelination in 2 at onset of epilepsy and in 12 at 10 months. Delayed myelination at 10 months was correlated with hypometabolism. (Natsume J, Watanabe K, Maeda N et al. Cortical hypometabolism and delayed myelination in West syndrome. Epilepsia Dec 1996;37:1180-1184). (Reprints: Dr J Natsume, Department of Pediatrics, Nagoya University School of Medicine, 65 Tsurumai, Showa-ku, Nagoya 466, Japan).