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Polystyrene Industry Responds to Facts To Act On No. 5 "Are Polystyrene Food and Beverage Containers A Health Hazard?"

This Facts to Act On (FTAO) was written by the Technical Committee of the Styrene Information and Research Center (1275 K St., N.W., Suite 400, Washington, D.C. 20005, 202-371-5514) in response to FTAO No. 5 "Are Polystyrene Food and Beverage Containers A Health Hazard?" August 1990. ILSR prepared Facts to Act On No. 5 based on a paper entitled "Styrene Migration Into Human Adipose Tissue" by George Baggett. Baggett and Dr. Louise Rainey, member of the Technical Committee of the Styrene Information Research Center, debated this issue at the National Citizens' Congress of the Grass Roots Alliance for Solid Waste Solutions in Indianapolis, IN, November 1990. This FTAO is part of an ongoing dialogue between industry and public interest representatives on the potential health impact of using polystyrene food and beverage containers. Facts to Act On No. 23 contains George Baggett's comments in response to this FTAO. The opinions expressed in both FTAOs are solely those of the authors.

Polystyrene Food and Beverage Containers Are Not Health Hazards

Background

Polystyrene plastic resin is made by a process in which molecules of styrene monomer are reacted to form a high molecular weight polymer. Small amounts of unreacted styrene monomer remain in the polystyrene. According to an overwhelming body of evidence, the quantities of styrene that could migrate from polystyrene food packaging to food or drink do not pose a threat to human health.

Styrene Health Issues

Styrene has been extensively studied during the past several decades, and the available published toxicological information is voluminous. Small amounts of styrene monomer can be ingested or inhaled without toxic effects of any kind. At very high exposures (whether from inhalation or ingestion), styrene can cause sedation and drowsiness similar to excessive alcohol intake, but these effects are temporary and there is full recovery within a short time after exposure is discontinued. The recently revised Occupational Safety and Health Administration (OSHA) permissible exposure limit of 50 ppm in air was established to be well below the level that would have adverse effects of any kind. The

effects of styrene on the nervous system have not been linked in any way to any type of spontaneous neurologic disease.

In a recent comprehensive review of the extensive reproductive and developmental toxicity data on styrene, it was concluded that overall there is little indication that styrene can cause any specific reproductive or developmental toxicity (Brown, 1991). Russian studies of questionable quality have suggested an association between styrene exposure and menstrual abnormalities, but a comprehensive U.S. study has shown no styrene-related menstrual dysfunctions in workers exposed occupationally to styrene (Lemasters et al., 1985).

Styrene has not been associated with any type of adverse hematologic (blood) effects in humans, and human monitoring studies have not shown any differences in chromosomal aberrations between control and exposed workers that are clearly attributable to styrene alone (Preston, 1990).

The epidemiological data on styrene is quite substantial, involving nearly 50,000 employees during the time period between 1940 and 1986. The combined weight of the evidence argues against a carcinogenic role for styrene at levels of occupational exposure that are drastically higher than those likely to be encountered in the environment (Fielder et al., 1981; Boyd et al., 1990). The available long term animal data is likewise quite substantial and indicates no carcinogenic response related to styrene exposure (Fielder et al., 1981; Bond, 1989).

Sources of Exposure to Styrene Monomer

The general public may potentially be exposed to styrene from a variety of sources. including ambient and indoor air, as well as the diet. Styrene has been shown to be a natural component of a wide variety of foods that we eat and drink every day, including many fruits, vegetables, nuts, meats, and diary products, as well as coffee, tea. cocoa. wine, and beer (Maarse, 1989). In addition trace amounts of styrene monomer do remain in polystyrene and, under certain cirumstances, even smaller amounts can "migrate" from polystyrene into food or drink. The amount of styrene monomer that can migrate from polystyrene depends on many factors including the type of food, how long the food is in the container, the temperature of the container, as well as the amount of unreacted monomer in the container initially (Durst and Laperle, 1990; Reid et al., 1980; Till et al., 1982; Till et al., 1987). In any event, the amount of styrene monomer present in foods packaged in polystyrene has been shown to be very low, in the range of a few parts-perbillion (Gilbert and Startin, 1983; MAFF, 1983). These levels of styrene in food are thousands of times lower than those which could have any adverse health effects. The Food and Drug Administration (FDA) regulates the amounts of styrene monomer that are allowable in food packaging to ensure that the amounts that could migrate into food are extremely small.

Airborne exposure to styrene monomer are known to be much more significant than dietary exposures. Styrene has been identified as a component of both cigarette smoke

and automobile exhaust. Quantitative determinations have shown styrene concentrations as high as 18 micrograms per cigarette (Baggett et al., 1974). Expired air analyses by the Environmental Protection Agency (EPA) have shown that cigarette smoking was responsible for greatly elevated breath concentrations of styrene and several other substances, including benzene (Wallace, 1988); the styrene breath concentrations were found to be 6-fold higher in smokers than in non-smokers. Analyses of automobile exhaust have shown styrene emissions up to 0.53 mg/mile (Warner-Selph and de Vita, 1989).

As a result of cigarette smoking, automobile exhaust emissions, and other factors, airborne exposures to styrene have been estimated by the EPA to be 2000 times higher than are dietary exposures (EPA, 1988).

Human Adipose Tissue Studies

When ingested or inhaled, styrene is distributed throughout the various organs and tissues of the body. As a result of its high lipid solubility, the concentration of styrene in adipose tissue (fat) is approximately 10-fold than in other organs and tissues (Withey and Collins, 1979). Styrene is rapidly metabolized and excreted, and at low doses there is no tendency toward long term accumulation in any organ or tissue (Ransey and Young, 1978).

The EPA National Human Adipose Tissue Survey (NHATS) reported by Stanley (1986) is an annual program to collect a nationwide sample of adipose tissue specimens and to analyze them for the presence of a wide variety of organic chemicals. The Stanley (1986) report involved a total of 763 individual human adipose tissue specimens that were coalesced into 46 composite tissue samples. Several dozen substances were detected in the composite tissue samples, including dioxins, halogenated solvents, and pesticides. Styrene monomer was reported to be present in all of the 46 composite samples, at wet tissue concentrations ranging from 8 to 353 ng/g. (A ng is one billionth of a gram.) These results have been misinterpreted by George Baggett in his paper, "Styrene Migration Into Human Adipose Tissue," to suggest that styrene was found in 100 percent of the human tissue specimens, and that there were adverse health effects related to the presence of styrene in adipose tissue. The facts do not support either of those suggestions. The actual percentage of individuals with detectable styrene concentrations was not determined, since only the composite tissues samples were analyzed. Moreover, the NHATS did not establish any relationship between the presence of styrene and any type of adverse health effect. In fact, much higher styrene adipose tissue concentrations than those reported for the NHATS specimens have been documented for individuals who were exposed occupationally to styrene showing no adverse effects (Engstrom et al., *1978*).

In view of the relative importance of airborne exposures, together with the widespread natural occurence of styrene, it is incorrect to attribute the presence of styrene in the NHATS adipose tissue specimens to migration from polystyrene food packaging.

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