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Arthur Greenberg

*University of New Hampshire*, [Art.Greenberg@unh.edu](mailto:Art.Greenberg@unh.edu)

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## PULLING EDB-CONTAMINATED FOODS OFF SUPERMARKET SHELVES: A COLLEGIAL APPROACH TO COMPLEX AND UNCERTAIN SCIENCE

Arthur Greenberg  
*Professor of Chemistry*

In September 1983, the United States Environmental Protection Agency (USEPA) initiated a ban on EDB (ethylene dibromide), a fumigant widely used on fruit crops and stored grain, after discovering high levels in groundwater.<sup>1</sup> At that time, EDB was also employed as an additive in leaded gasoline. EDB was known to cause cancer as well as birth defects in test animals. It is capable of chemically bonding with DNA as well as proteins. Since EDB has two bromine atoms, it can create DNA cross-links making mutations particularly difficult for cellular repair. Subsequently, California officials reported alarmingly high levels of EDB in fruits. Reports of high levels in muffin, corn flour, and cake mixes, as well as some imported fruits, started to appear. The EPA adopted guidelines on February 3, 1984 restricting maximum levels in ready-to-eat foods to 30 parts per billion (ppb), 150 ppb in foods to be cooked, and 900 ppb in raw grain.<sup>1</sup>

The present essay revisits why and how decisions were made to remove EDB-contaminated foods from supermarket shelves during a month-long period of intensive concern. It is a process that had scientific, medical, political, and financial dimensions. Risks to public health as well as millions, possibly billions, of dollars rested on these decisions. Typically, the average citizen only learns of final decisions in such cases, the processes leading to them being both complex and mysterious. In the EDB case study summarized very briefly in this essay, the most reassuring aspects were the “conservative” (i.e., human-protective) approach and the collaboration between industry, government, and academe that led to action based upon complex, albeit uncertain, science. The author had the good fortune to be a member of the state panel that made recommendations enacted by the state of New Jersey.

### **A Brief Primer on Human Exposure to Toxic Substances**

Carbon monoxide is a toxic substance<sup>2</sup> that produces acute (short-lived) effects.<sup>3</sup> While acute exposure to levels of 150-200 parts per million (ppm) or higher may well result in death, exposure to levels around 70 ppm causes nausea and/or vomiting. Fresh air restores normal health. In contrast, chronic exposure to exceedingly low levels (ppb, ppt, or lower) of carcinogens may result in irreversible development of cancer. What is a ppb (part per billion)? Imagine a single drop of EDB in a municipal swimming pool. A ppt (part per trillion) is one thousand times even more dilute. Although humans have DNA-repair mechanisms, it is theorized that, in principle, cancer can be initiated by a single mutation. This is the basis for the USEPA classifying the MCL (Maximum Contaminant Level) of carcinogens such as EDB as zero. However, since chemical analysis is amazingly sensitive (not uncommonly 0.001 ppt), the USEPA issues practical standards for MCLG (Maximum Contaminant Level Goal). The MCLG for EDB is 0.05 ppb in drinking water.<sup>4</sup>

Humans are exposed to toxic substances through inhalation, ingestion, and dermal absorption. Consider the example of the carcinogen trichloroethylene (TCE),<sup>5</sup> an industrial degreaser used to clean metal prior to painting. Waste TCE may ultimately flow into sources of drinking water. How does one assess risk? First, measurements using extremely sensitive chemical analyses are performed to survey a representative set of groundwater systems. Of 55 systems serving populations in the U.S. greater than 100,000 people, it was determined that 41 had TCE levels lower than 0.5 ppb, 14 had TCE levels in the range 0.5

to 5 ppb, and none had levels exceeding 5 ppb.<sup>5</sup> At the other extreme (systems serving 25 to 100 people), of 19,125 estimated systems, 18,506 were estimated to have levels less than 0.5 ppb, 465 between 0.5 to 5 ppb, and 156 higher than 5 ppb including 26 greater than 100 ppb.

Next one considers human exposure to highly-polluted water (100 ppb). This is a “conservative” (i.e., worst case, human-protective) approach. Exposure routes include ingestion of water, inhalation of indoor air as well as shower air, and dermal absorption (bathing, swimming). Based upon fairly rough assumptions, total daily exposures to TCE are formula-fed 9 lb infant: 80 µg (microgram = millionth of a gram); 70-pound pre-teen: 320-640 µg; 132-pound woman: 600 µg; 154-pound man: 490 µg. Why the higher number for the woman? When these estimates were made (1980s), a considerably higher percentage of women compared to men, spent more time at home.

Although estimates of source concentrations and especially human exposure scenarios have large uncertainties, the greatest uncertainty comes from extrapolation of animal testing to realistic human exposure scenarios. Animal studies of carcinogens are usually impractical using realistic exposures. Even a very potent carcinogen may produce one case of cancer in 1000 animals at realistic levels. To make animal carcinogenicity testing feasible, unrealistically high doses are administered. Extrapolation of “megadose” data to environmentally-realistic levels can easily account for an uncertainty factor of ten thousand or more. In the absence of more precise science, the “conservative” (people-protective) approach is a linear extrapolation from the POD (point of departure): the estimated dose near the lower end of the observed range.<sup>6</sup>

### How Were Decisions Made?

New Jersey assembled an EDB Advisory Group

consisting of 10 scientists and medical doctors and chaired by an assistant commissioner of the New Jersey Department of Health.<sup>7</sup> The group included epidemiologists, toxicologists, chemists, biochemists, medical doctors, a medical ethicist, and scientists from the food industry. There was constant exchange of data and information with the USEPA, as well as with the health departments of large states including California, Connecticut, Massachusetts, New York, and Texas. The goal of the Advisory Group was to make a recommendation to the New Jersey Department of Health about which foods must be immediately removed from market shelves and not sold (or donated) to the public.

The first analyses were conducted on baby foods. Happily, the results were negative at the limits of detection. A sample of Mexican oranges obtained in a New York City market was the “record holder:” 41,590 ppb (peel); 2,173 ppb (pulp). A box of long-grain rice was found to have a level of 351 ppb EDB and the batch was immediately recalled by the manufacturer. A survey of 23 commercial bread samples found that 20 had undetectable levels of EDB, with an overall average of 0.88 ppb (one sample had 7.4 ppb); seven ready-to-eat corn muffins had an average of 3.1 ppb, and 12 cold cereals all had levels lower than the limit of detection.

As these chemical analyses continued, the next step was estimation of total human exposure to EDB. To simplify the process, three classes of people were recognized (with estimated daily consumption in parentheses): 1) General Population (grains: 270 grams; citrus crops: 74 grams); 2) Vegetarian Population (grains: 970 grams; citrus crops: 200 grams); and 3) Two-Year-Old Child (grains: 103 grams; citrus crops: 125 grams). Total ingestion of EDB was then estimated for various EDB concentrations from 1 ppb to 50 ppb. While removing contaminated foods from the shelves would effectively eliminate ingestion of EDB,

inhalation of EDB from ambient air was unavoidable (in 1984 EDB was still added to leaded gasoline). A survey of the literature indicated average concentrations of EDB in ambient air of 27 ppt. A two-year-old child inhales about 8 cubic meters (m<sup>3</sup>) of air per day; an adult inhales about 20 m<sup>3</sup> of air each day. The exposure comparison was very illuminating. If the limit adopted for ready-to-eat food was the USEPA recommended maximum at 30 ppb, a two-year-old child would ingest 6.9 µg of EDB per day and inhale 1.7 µg from average ambient air. If the food limit was designated at 5 ppb, daily ingestion would account for 1.1 µg while inhalation would still account for 1.7 µg. If the limit for ready-to-eat food was designated at 1 ppb, daily ingestion would account for 0.2 µg and daily inhalation would account for 1.7 µg—almost an order of magnitude greater. It must be pointed out that in 1984 little was known about the relative health hazards of ingested versus inhaled EDB.

Analysis of lifetime (70-year) risks of cancer deaths due to exposures to EDB at various levels then followed. It is generally accepted that one in a million “excess deaths” over a lifetime due to a specific source is an acceptable benchmark since that is below the “background noise” of other lifetime risks such as automobile accidents and various health risks. Among many scenarios, the EDB Advisory Group made the following comparison of Excess Cancer Risk per million for the General Population for ready-to-eat foods (citrus and grains): At 1 ppb: Risk due to grains + citrus: 6; Risk due to air: 80. At 5 ppb: Risk due to grains + citrus: 33; Risk due to air: 80; At 30 ppb: Risk due to grains + citrus: 190; Risk due to air: 80. In the decision-making process, Massachusetts considered adopting guidelines of 1 ppb for both intermediate products (e.g., muffin mixes) as well as ready-to-eat foods. That would have resulted in the elimination from supermarket shelves of roughly 11 percent of ready-to-eat foods and 87 percent of mixes. Ultimately, New Jersey settled

on the USEPA guidelines, eliminating roughly 3 percent of ready-to-eat products and 2 percent of intermediate products.

### The Aftermath

The use of EDB as a fumigant was banned in 1984 and all traces disappeared from the food chain during the next two to three years. (The 70-year lifetime exposure scenario noted above was also part of the human-protective decision-making strategy.) Leaded gasoline was totally eliminated from on-road vehicles by the USEPA as of January 1996 and EDB was no longer a gasoline additive. Its lifetime in the environment is only on the order of months so it is no longer a problem. Ironically, EDB’s first replacement as a fumigant, methyl bromide (more volatile and less toxic), was found to be damaging to the ozone layer and banned by 2005. Fumigation remains an important technique for producing and storing food at reasonable cost. Typically, modern fumigants such as arsine are volatile and short-lived in the environment but their high toxicity requires great care by those who apply them to crops or stored grains.

And what of the foods removed from supermarket shelves and the food supply during 1984? One could readily make the case that the EDB risks were far lower than risks due to malnutrition. Why not supply these food products to malnourished populations? However, such a decision would have been almost certainly impossible on purely political grounds.

### REFERENCES

1. USEPA, *Ethylene Dibromide (EDB). Scientific Support and Decision Document for Grain and Grain Milling, Office of Pesticide Programs, United States Environmental Protection Agency, Washington D.C., February 8, 1984.*
2. *The Merriam-Webster Dictionary defines “toxin” as “a poisonous substance that is a specific product of the metabolic activation of a living organism and is usually unstable, notably toxic when introduced into the tissues, and typically capable of inducing antibody formation.” Even the New York Times occa-*

sionally slips and calls simple chemicals like EDB and benzene “toxins.” They are “toxic substances” or “toxics” if you must. Rattlesnake and black widow spider venoms are “toxins.”

3. U.S. Consumer Products Safety Commission, CPSC Document #466: [www.cpsc.gov/cpscpub/pubs/466.html](http://www.cpsc.gov/cpscpub/pubs/466.html) accessed on June 20, 2011.
4. USEPA, *Natural Attenuation of the Lead Scavengers 1,2-Dibromoethane (EDB) and 1,2-Dichloroethane (1,2-DCA) at Motor Release Sites and Implications for Risk Assessment*, EPA/600/R-08/107, September 2008.
5. C.R. Cothorn, W.A. Coniglio, W.L. Marcus, “Estimating Risk to Human Health,” *Environmental Science & Technology*, Volume 20 (1986) pp 111-116.
6. USEPA, *Guidelines for Carcinogen Risk Assessment* USEPA/630/P-03/001F, March 2005, Washington, D.C. <http://epa.gov/cancer/guidelines/> accessed on June 17, 2011.
7. Ronald Altman, M.D., Chairman, Assistant Commissioner, Division of Epidemiology & Disease Control, New Jersey Department of Health; John C. Colmey, Ph.D., Vice President of Research and Development, Quality Assurance, Nabisco; Michael A. Gallo, Ph.D., Associate Professor of Toxicology- Robert Wood Johnson Medical School; Michael Gochfeld, M.D., Ph.D., Acting Chairman Department of Environmental and Community Medicine, Robert Wood Johnson Medical School; Arthur Greenberg, Ph.D., Professor of Chemistry, New Jersey Institute of Technology; Peter C. Kahn, Ph.D., Associate Professor of Biochemistry, Rutgers University; Donald B. Louria, M.D., Chairman, Department of Preventative Medicine and Community Health, New Jersey Medical School; Steven M. Marcus, M.D., Project Director, New Jersey Poison Information and Education System, Newark Beth Israel Medical Center; Russell McIntyre, Th.D., Robert Wood Johnson Medical School; Myron A. Schmutzer, Vice President, Quality Assurance, Wakefield Food Corporation; Inderjit Thind, M.D., Ph.D., Professor of Preventative Medicine, Director of Epidemiology, New Jersey Medical School.