domized clinical trial under these circumstances. ysicians traditionally act in the best interests of patient under their care, and patients expect this eir physicians. If this commitment to the patient ttenuated, even for so good a cause as benefits to ure patients, the implicit assumptions of the doc-patient relationship are violated. I have no doubt t we would lose more than we would gain by adopt such an approach.

MARCIA ANGELL, M.D.

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CORRESPONDENCE

Letters to the Editor are considered for publication (subject to editing and abridgment), provided that they are submitted in duplicate, signed by all authors, typewritten in double spacing, and do not exceed 1½ pages of text (excluding references). They should not duplicate similar material being submitted or published elsewhere, and they should not contain abbreviations. Letters referring to a recent *Journal* article should be received within six weeks of the article's publication. We are unable to provide pre-publication proofs, and unpublished material will not be returned to authors unless a stamped, self-addressed envelope is enclosed.

CONTROVERSY ABOUT THE RISKS OF EDB

To the Editor: Ethylene dibromide (EDB) has been used for some 40 years in our country as a soil fumigant for nematode control, to protect stored grains, milling machinery, and a small percentage of citrus fruits from infestation, destruction, and contamination by insects. Of the total production of EDB, only 8 to 10 per cent is used as a fumigant; the remaining 90 per cent is used as an "antiknock" additive in leaded gasoline.

Recently EDB residues in some grain foods, citrus products, and well water have been added to the list of public-health nonproblems and nonemergencies, such as saccharin in diet drinks, mercury in swordfish, and nitrites in cured meats. EDB is a potent carcinogen according to a few experimental studies, the latest of which, as far as I know, was published in 1977.* This study was performed in rodents. The EDB was administered by gavage in doses from 10,000 to 50,000 times higher than those we may receive in our diets. By this procedure there was an increase in carcinomas of the stomach in rats and of the forestomach in mice. A few other studies in rodents in which EDB was administered by inhalation and dermally have produced similar results. But can the results of these studies, in which huge doses were used and which were nondietary, be extrapolated to people receiving tiny amounts in some foods and water? Interestingly, the incidence of carcinoma of the stomach in human beings has drastically decreased in the past 40 to 50 years — just the length of time that EDB in parts per billion (not million) has been in some of our foods. I am not implying any relation between the two, just pointing out an interesting association.

I know of no published or unpublished studies in which EDB was fed as part of the diet. Yet, predictions of risk assessment for cancer in human beings have been made by extrapolation from these rodent studies. The risk model used by the Environmental Protection Agency incorporates several hypothetical assumptions about the dose–response relationship and about dose equivalency in human beings and rodents. The risk model has not been subjected to peer review.

The levels of EDB for grains during various stages of storage and food production announced by the Environmental Protection Agency on February 3, 1984, and those for citrus fruits announced on March 2, are compatible with the best of health. Most of our present grain foods and citrus products meet these recommendations.

A few states have suggested that they may set levels of EDB appreciably lower than those suggested by the Environmental Protection Agency, but as of this writing only Massachusetts has done so and has taken action to compel removal from grocery stores of foods that contain more than these unnecessarily lower amounts of EDB. Consumers have even been encouraged to return products that do not meet these standards for lower levels. Such foods will be destroyed despite their safety. This is in a time of hunger for some. The Commissioner of Public Health of Massachusetts has taken this action despite the fact that EDB has been in some of our foods for over a generation and our health has never been better; longevity increases slightly every year; and as mentioned earlier, cancer of the stomach is at an all-time low. Where is the public-health emergency? There is none!

Continue to enjoy your blueberry muffins and oranges as you do your swordfish and diet drinks. Just don't eat (or drink) too much.

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*Weisburger EK. Carcinogenicity studies on halogenated hydrocarbons. Environ Health Perspect 1977; 21:7-16.

The above letter was referred to the Commissioner of Public Health of Massachusetts, who offers the following reply:

To the Editor: We appreciate Dr. Stare's concerns about this important subject. However, his letter demonstrates several serious misunderstandings about the central issues involved in setting limits for EDB in food. First of all, data on laboratory animals are widely considered to be relevant to human beings and to provide a suitable basis for regulatory action. If a substance causes cancer in animals, it must be considered to be a likely human carcinogen. EDB has been shown to cause cancer in animals at 12 different sites. EDB causes lung cancer in animals by three different routes of exposure, including dietary ingestion. EDB has been classified among the most potent carcinogens ever tested.

Secondly, the dosage in animal experiments is always much higher than typical human exposure. The purpose of this is to reduce the time and expense of the experiments. The vast majority of substances will *not* cause cancer, however, no matter how high the dose.

Thirdly, no safe threshold level of exposure below which cancer does not occur has ever been shown for a carcinogenic agent. Therefore, if a substance has been shown to cause cancer at any dose, then only in the absence of exposure will there be no excess risk of cancer. The Environmental Protection Agency, the Occupational Safety and Health Administration, and the National Cancer Institute all subscribe to this concept of no safe threshold for carcinogenic substances.

In addition, the population is not quite as healthy as Dr. Stare implies. Overall cancer rates are high and are rising somewhat both in Massachusetts and in the United States as a whole. Lung-cancer rates are rising especially fast. Under these circumstances, the data from animals on the carcinogenicity of EDB appear especially troublesome for human beings. These data strongly suggest that the levels of EDB in our foods are *not* "compatible with the best of health," as suggested.

There is a curious inconsistency between the Environmental Protection Agency's decision to suspend further use of EDB because of the dangers it poses to the public and their establishment of tolerance levels for EDB that allow over 99 per cent of products contaminated with EDB to remain on the market. This inconsistency suggests that economic considerations overwhelmed scientific, public-health considerations in setting the guidelines.

We believe that the only reasonable way to minimize cancer risks is to set levels much lower than the Environmental Protection Agency's and to remove EDB totally from the food system as soon as possible. Therefore, our department has recommended that levels of EDB be set well below the Environmental Protection Agency's tolerance levels. Other states, such as New York, have also set much lower levels than the Environmental Protection Agency's, and we anticipate that more states will follow. Prudent public-health policy allows no other alternative but to interrupt current exposure and prevent future exposure to EDB.

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CAUTION ABOUT CHORIONIC VILLI SAMPLING IN THE FIRST TRIMESTER

To the Editor: Chorionic villi sampling has been heralded by the media as a "painless procedure . . . done in a physician's office as early as the fifth week of pregnancy" with risks "probably . . . the same" as in midtrimester amniocentesis.[1] We have consequently received many inquiries from patients who would like to have chorionic villi sampling and from physicians who wish to do the procedure.

Experience with 240 cases in six centers (Lund, London, Paris, Milan, Philadelphia, and Chicago)[2] indicates that the fetal-loss rate averages about 12 per cent. This is far above the rate with midtrimester amniocentesis.[3,4] Although chorionic villi sampling is done in the first trimester, when the majority of spontaneous abortions normally occur, it is not yet clear whether this technique carries a sizable risk of excess fetal loss.

The most common indication for prenatal diagnosis is advancing maternal age. Even at 45 years of age, the risk of a chromosomal abnormality[5] is only about 5 per cent — well below the current fetal-loss rate with chorionic villi sampling. It would seem prudent now to reserve this procedure for disorders, such as the hemoglobinopathies,[6,7] with genetic risks sufficiently high to justify chorionic villi sampling.

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THE PREVALENCE OF CYTOMEGALOVIRUS VIRUR AMONG HOSPITALIZED CHILDREN AND THE RISK CYTOMEGALOVIRUS ACQUISITION BY NURSES

To the Editor: In the October 20 issue[1] Dworsky et al. report important study on the epidemiology of cytomegalovirus (C among health-care workers. However, the nurses studied all wo in newborn nurseries where there was a very low prevalence (1 cent) of CMV infections.

In a study to determine the prevalence of CMV viruria a children hospitalized at our medical center, 2198 children ha mission urine samples cultured for CMV within 17 months; 4 cent had CMV viruria (105 positive cultures), but importa differences were observed. In the intensive-care nursery the p lence of CMV viruria (congenital and acquired infections) w per cent (18 of 551 newborns with viruria). On the nursing un children up to two years of age, the prevalence of CMV virur 6.8 per cent ($P<0.01$, chi-square = 7.3 when values are com with those in newborns). The prevalence of CMV virur nursing unit for children two to five years of age was 7.2 per c of 382), similar to that for children up to two years of age (chi-square = 0.004). On a nursing unit for children 5 to 12 y age the prevalence of CMV viruria decreased to 4.2 per cen 476, $P = 0.01$, chi-square = 6.6 when values are compar those in children two to five years of age). For 12-to-18-year- prevalence of CMV viruria was only 1.2 per cent (3 of 245, P chi-square = 9.2 when values are compared with those in ch to 12 years of age).

Among 72 seronegative nurses with daily patient cont have acquired CMV, but only 20 have worked with the hig lence age group (up to five years). These observations sug in spite of constant exposure, CMV transmission occurs inf ly. However, previous studies[2-4] suggesting that pediatric n at increased risk for CMV acquisition included ward n opposed to nursery nurses alone. In one study[2] the highe seroconversion (7.7 per cent per year) occurred among 5 who worked on a general pediatric ward. Thus, additional ing of nurses and the prevalence of CMV infections an children for whom they care will be required before m conclusions can be drawn about the risk to pediatric nurses acquisition.

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GLUCOSE RESPONSE OF DIABETIC PATIEN DIFFERENT CARBOHYDRATES

To the Editor: The carbohydrate content of various f necessarily be used as a reliable predictor of the g sponse.[1,2] Although studies of this effect[2] have exam glucose levels over a four-hour period following a mea ization of the immediate postprandial response is of m