CHEMICAL MANUFACTURERS ASSOCIATION

May 23, 1995

Dear Vinyl Chloride Health Committee Members:

The May 4, 1995, response from the Agency for Toxic Substances and Disease Registry to our March 22 letter is enclosed. The response is favorable to the industry. Jonathan Ramlow, Bill Breslin (Dow), Caffey Norman and I talked to ATSDR representatives on May 16 to discuss the industry approach on the combined reproductive and developmental effects study protocol. The industry approach was acceptable to ATSDR. The Record of Conference Call is enclosed. Bill Breslin will prepare a draft protocol for the Committee's review prior to submission to ATSDR. The Agency has requested the final protocol by June 9. I anticipate distributing the draft protocol by June 2.

Jonathan Ramlow has asked me to explore the feasibility of scheduling a Committee meeting on June 7 or 8. There are many issues that need to be discussed as soon as possible. The most important among them is review of the combined 2-generation reproductive and developmental effects study protocol. The agenda for the meeting is enclosed.

The *Pesticide and Toxic Chemical News* report on EPA's vinyl chloride cancer risk assessment presented at the Conference on Risk Assessment for Sensitive Populations is enclosed. I will try to get more information from Jim Cogliano of EPA on his presentation prior to our next meeting.

I received the enclosed Cancer Facts & Figures - 1995 from Ed Beeler. The 1994 edition associated vinyl chloride exposure with lymphoma. The Committee filed comments with the American Cancer Society last year objecting to this association in the absence of scientific data to link vinyl chloride exposure to lymphoma. The ACS agreed with our comments and deleted reference to vinyl chloride from the lymphoma section in its 1995 edition.

Please complete the enclosed availability survey and return it to me by fax at (202) 887-5427 AS SOON AS POSSIBLE so that a meeting can be arranged. If you have any questions, please call me at (202) 887-1192.

Sincerely,

Has Shah

Hasmukh C. Shah, Ph.D.
Manager, Vinyl Chloride Panel

Enclosures

R&S152549

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CHEMICAL MANUFACTURERS ASSOCIATION

Vinyl Chloride Health Committee

Tentative Agenda

Date: To Be Announced

Time: To Be Announced

Place: CMA Offices
2501 M Street, NW
Washington, D.C.

1.0 Review of Committee Activities with Respect to EPA/ATSDR Data Needs for Vinyl Chloride Under TSCA Section 4

2.0 Review of Draft 2-Generation Reproductive/Developmental Effects Study Protocol

3.0 Discussion of Jim Swenberg's (University of North Carolina at Chapel Hill) Request for Tissue Samples from the Reproductive Effects Study - Jim Knaak

4.0 Discussion of EPA's Neurotoxicity Data Requirements

5.0 Update on Vinyl Chloride Epidemiology Study

6.0 Discussion of Carlo Tamburo's (University of Louisville) Proposal for a Brain Cancer Case Control Study

7.0 Discussion of EPA's Risk Assessment of Vinyl Chloride

8.0 Financial Statement

9.0 Set Date for Next Meeting or Conference Call

Hasmukh C. Shah, Ph.D.
Manager, Vinyl Chloride Panel

Subject to Approval

R&S 152550

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Agency for Toxic Substances
and Disease Registry
Atlanta GA 30333

MAY 4 1995

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Chemical Manufacturers Association
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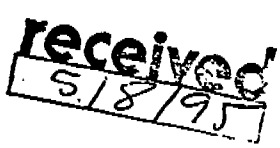

Dear Dr. Shah:

This is in response to your March 22 letter to Dr. William Cibulas regarding the intent of the Chemical Manufacturers Association (CMA) Vinyl Chloride Panel to address vinyl chloride data needs identified by the Agency for Toxic Substances and Disease Registry (ATSDR). In the letter, you enclosed a preliminary study protocol for a two-generation reproductive toxicity study of vinyl chloride by the inhalation route. You asked if ATSDR would consider including measures of developmental toxicity endpoints in the reproductive toxicity study protocol. You also submitted a review of the available data on the developmental toxicity of vinyl chloride.

After evaluating the available data on developmental toxicity studies for vinyl chloride, we determined that the CMA reproductive toxicity study protocol including examination of developmental toxicity endpoints provides an acceptable alternative to separately conducting a two-species developmental toxicity study. Generally, the Agency will consider proposals to simultaneously acquire reproductive and teratologic information in the absence of any reproductive or teratologic data. In view of the suggestive human data and limited animal data for developmental toxicity, as stated in the ATSDR Priority Data Needs Document for vinyl chloride, we believe it would be appropriate and efficient to simultaneously examine both toxicity endpoints in the same study.

In merging the study protocols for developmental toxicity and reproductive toxicity, we ask that you adopt the Environmental Protection Agency testing guidelines for inhalation developmental toxicity studies (Code of Federal Regulations, Part 798.4350, "Inhalation developmental toxicity study," July 1, 1992). For example, exposure duration shall be at least six hours daily and the exposure period shall cover the period of major organogenesis (days 6-15 for rats for this study protocol). At the time of sacrifice or death during the study, the dam shall be examined macroscopically for any pathological changes which may have influenced the pregnancy.

CHEMICAL MANUFACTURERS ASSOCIATION
Vinyl Chloride Health Committee
Record of Conference Call

Date: May 16, 1995
Time: 11:15 a.m.

List of Attendees:

Bill Breslin	Dow Chemical
Bill Cebulas	Agency for Toxic Substances and Disease Registry
Christopher DeRosa	Agency for Toxic Substances and Disease Registry
Caffe Norman	Patton Boggs
Jonathan Ramlow	Dow Chemical
Ivan Stevens	Agency for Toxic Substances and Disease Registry
John Wheeler	Agency for Toxic Substances and Disease Registry
Has Shah	CMA

1.0 The Agency for Toxic Substances and Disease Registry representatives emphasized that the studies conducted under the Memorandum of Understanding between CMA and ATSDR be conducted according to EPA's TSCA GLPs and Study Guidelines. Deviations from standard EPA guidelines for the reproductive and developmental effects studies should be documented and justified. For the combined reproductive and developmental effects study. ATSDR agreed that the study can be conducted in one species.

2.0 Bill Breslin will develop a protocol for the combined reproductive and developmental effects study, including dose levels, based on discussion with ATSDR representatives. The protocol will specify evaluating developmental effects parameters in the first generation pups only. CMA will try to submit the protocol to ATSDR within the next three weeks.

3.0 ATSDR will contact EPA on EPA's need for neurotoxicity information on vinyl chloride and identify specific endpoints of interest. ATSDR will inform CMA of the EPA needs. If there is a mutual agreement on the need for neurotoxicity data, an attempt will be made to expand the reproductive/developmental effects study protocol to include an evaluation of neurotoxicity parameters.

4.0 The conference call concluded at approximately 11:55 a.m.

Has Shah

Hasmukh C. Shah, Ph.D.
Manager, Vinyl Chloride Panel

Subject to Approval

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"We have to first confirm these health effects, and then see if they fit into a pattern," Goldman remarked. "After that, we can develop an appropriate response," she said.

In the consent agreement, DowElanco said it would:

— assist EPA by using its best efforts to review files of its parent companies for any information concerning health and environmental effects incidents for pesticide products registered under FIFRA, which the company obtained as a consequence of its 1989 formation;

— waive its right to request a judicial or administrative hearing on the matter, and

— pay the penalty within 30 days, or pay the total civil penalty originally proposed in the complaint, $1.635 million. (☎5PTCN 2712, 15 pages, $8)

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2-VINYL-1,3-DIOXOLANE EXPOSURE LINKED TO RABBIT DEATHS IN SECTION 8(e) REPORT

Rabbits exposed to 1,3-dioxolane, 2-ethenyl, also known as 2-vinyl-1,3-dioxolane, died after displaying a variety of neurotoxic symptoms, according to TSCA Section 8(e) submission #8EHQ-0495-13021, said Degussa Corp.

The test substance was used undiluted and applied at 6.61 mg/kg, 66.1 mg/kg and 208.6 mg/kg on male and female rabbits, according to the study. The results were intoxication characterized by hypokinesia, decrease in muscle tone, loss of righting reflexes and salivation, which appeared about two hours after treatment and lasted until death.

According to the study, deaths occurred between five and 24 hours after treatment, with the LD50 dose for males and females calculated at 25.1 mg/kg.

In TSCA 8(e) submission #8EHQ-95-13426, Cytec Industries reported the results of a guinea pig maximization study conducted with acrylamide, 50% aqueous, by Stockhausen Laboratory for Toxicology in Germany. The results indicated a sensitization rate of 85%, indicative of a positive sensitization reaction, the submission said. (☎5PTCN 2716, 2 pages, $5)

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VINYL CHLORIDE CANCER RISK LINKED TO EARLY-LIFE EXPOSURE

A new quantitative cancer assessment on lifetime exposures to vinyl chloride in animals suggests that the risk of cancer depends on the age at exposure, with higher risks attributed to exposure at younger ages, said Environmental Protection Agency's V. James Cogliano at a recent toxicology meeting.

Speaking at the April 25 Conference on Risk Assessment Issues for Sensitive Human Populations at Wright-Patterson Air Force Base in Ohio, Cogliano said newborn animal exposure studies demonstrate that a brief exposure can induce unseen tumors and higher incidence of apparent tumors following long-term exposure later in life.

Cogliano, chief, Carcinogen Assessment Statistics and Epidemiology Branch, EPA's Office of Research and Development, said new quantitative approaches reflecting early-life sensitivity have been developed to supplement conventional approaches to estimating the increased cancer risk from inhaling vinyl chloride.

Vinyl Chloride Studies May Have Superfund Implications

These studies may have implications for Superfund risk assessments as EPA must decipher the risk of inhaling vinyl chloride from nearby dumps, he said, as well as estimating early risks for other chemicals.

Cogliano told attendees of the Ohio meeting that studies on early-life effects of vinyl chloride exposure, especially in a species other than rats, would help resolve how the sensitive period corresponds with humans. But he encouraged more studies in young animals to identify other carcinogens that affect sensitive stages of development.

When asked whether other chemicals had been tested, Cogliano replied that vinyl chloride was the first tested and that he would not expect to see a dramatic result with every chemical. "We think it could happen with other chemicals, but we have no data yet," he added.

University of Mississippi's Harihara Mehendale also focused on young animals — specifically, why early postnatal development rats were resistant to a deadly combination of chlordecone (CD) + CCl4 at individually nontoxic doses. Mehendale found that young rats responded faster to tissue injury than adults, and were less susceptible to acute exposure since they were more efficient in stimulating tissue repair.

Mehendale focused on liver injury occurring in postnatally developing and adult male rats and found that prompt and exacting stimulation of tissue repair permitted efficient recovery from injury for the younger rats. Mehendale said that perhaps kidneys and lungs should be examined as well.

Past research has focused on qualitatively defining causal relationships, but the U.S. Army-sponsored conference focused on the need to better define sources and calculate the magnitude of quantitative estimates of risk to sensitive populations.

While the meeting showed a convergence on different methods in variability issues, "early efforts are just that in showing the feasibility of different analyses," said Dale Hattis, a research associate professor at Clark University's Center for Technology, Environment and Development. According to Hattis, there is pressure to better characterize uncertainties to determine whether population subgroups are being adequately protected and whether policymakers are getting the biggest bang for their buck. "There is a need in the current environment to count the bodies," he added.

According to Hattis, it is unlikely that the same factor of 10 rule is appropriate for all different kinds of noncancer risks and different kind of agents, despite the need for relatively straightforward and standardized treatment of variability in toxic risks.

Sociodemographic Factors Affect Susceptibility to Chemicals

Ken Sexton of the University of Minnesota's School of Public Health noted how sociodemographic variables, such as class and race, can affect exposure-related and susceptibility-related attributes which, in turn, can lead to the well-documented disparities in health status among populations.

"There has been a surprising lack of research" in occupational and environmental studies on this issue, said Sexton, who helped launch EPA's National Human Exposure Assessment Survey, an interagency initiative to conduct exposure surveillance of Americans.

Sexton defined individuals and groups at potentially greater risk when they are exposed above a health-related benchmark, more susceptible to the effects of exposure, or both.

Too much regulation also can render groups more susceptible to risk, Sexton said. Increasing regulation can affect socioeconomic status, in turn adversely affecting health, he said.

The congressional debate on risk assessment is raising important questions, such as the impact of cumulative risk, comparative risk and total risk, and bringing together epidemiology and toxicology into "one sphere," he said.

Multiple Chemical Sensitivity Syndrome Explored

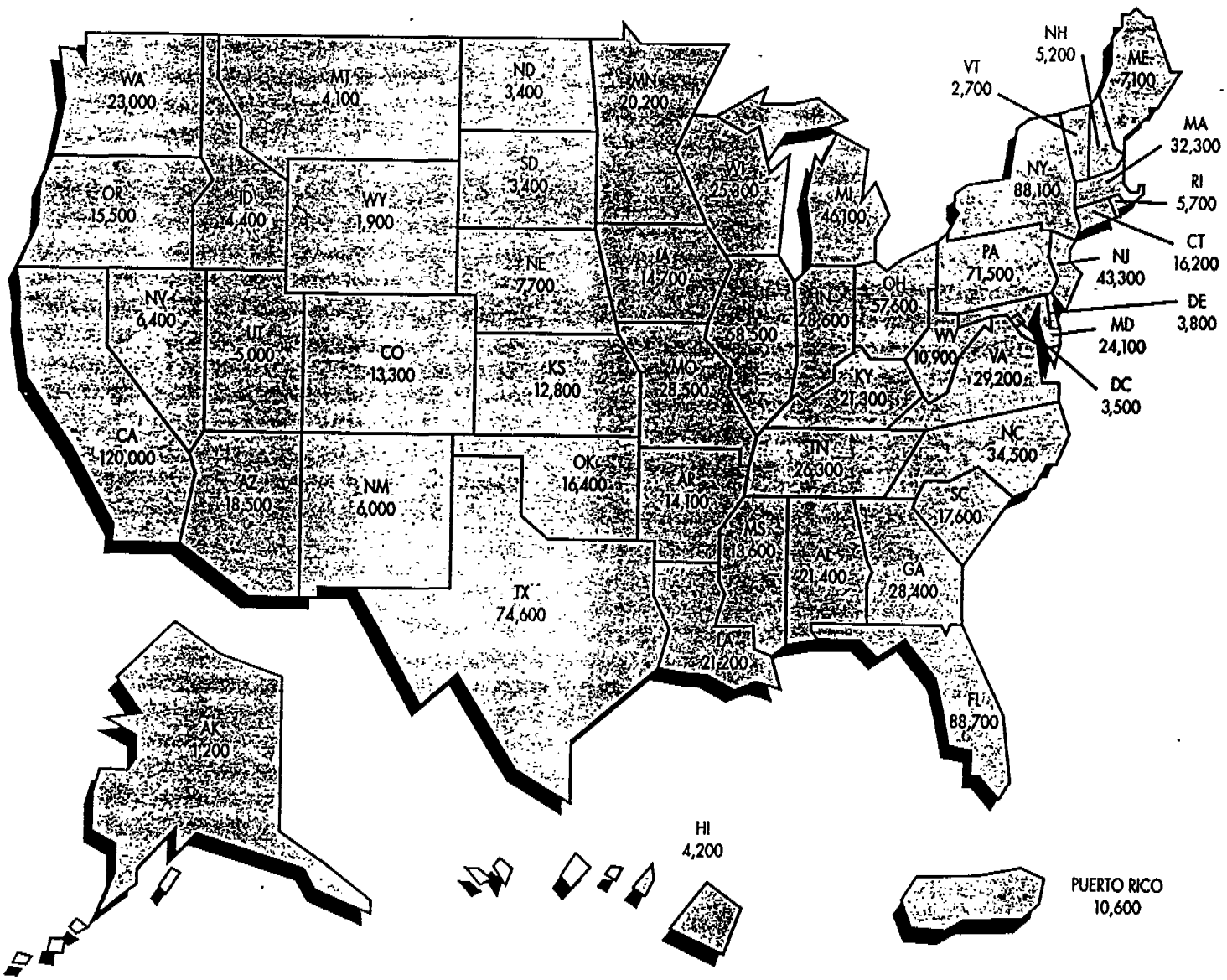
How multiple chemical sensitivity is defined will determine how many people are affected, said Claudia Miller, who for the past two years has been involved with Houston Veterans Affairs' Regional Referral Center for Gulf War veterans.

Miller outlined an emerging theory on chemical sensitivity. The idea that it "may be a mechanism for disease posits that a broad spectrum of chronic illnesses, ranging from asthma and migraine to depression and chronic fatigue, may be the consequences of environmental chemical exposures," she said.

First, an initial exposure event interacts with a susceptible individual, causing loss of tolerance to everyday, low-level chemicals, Miller said, adding that substances then trigger symptoms that perpetuate the illness. Seventy-one percent of Gulf War veterans surveyed said they have chemical intolerances, Miller said.

David Oberstreet, research associate professor, University of North Carolina at Chapel Hill, presented findings on his research on Flinders Sensitive Line rats, which are selectively bred to be more sensitive to organophosphate diisopropylfluorophosphate. These rats showed similar symptoms to MCS humans, including depression and reduced activity and appetite, he said.

CANCER FACTS & FIGURES–1995



AMERICAN CANCER SOCIETY®

Estimated number of new cancer cases in 1995 by state, total: 1,252,000 (excluding Puerto Rico).*

*Excluding basal and squamous cell skin cancer and carcinoma in situ.

REVISED 1/95

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mately 7,000 cases) and chronic lymphocytic (approximately 7,800 cases).

Mortality: An estimated 20,400 deaths in 1995.

Signs and Symptoms: Fatigue, paleness, weight loss, repeated infections, bruising easily, and nosebleeds or other hemorrhages. In children, these signs can appear suddenly. Chronic leukemia can progress slowly and with few symptoms.

Risk Factors: Leukemia strikes both sexes and all ages. Causes of most cases are unknown. Persons with Down syndrome and certain other genetic abnormalities have higher than normal incidence of leukemia. It has also been linked to excessive exposure to ionizing radiation and to certain chemicals such as benzene, a commercially used toxic liquid that is also present in lead-free gasoline. Certain forms of leukemia and lymphoma are caused by a retrovirus, HTLV-I (human T-cell leukemia/lymphoma virus-I).

Early Detection: Because symptoms often resemble those of other, less serious conditions, leukemia can be difficult to diagnose early. When a physician does suspect leukemia, diagnosis can be made using blood tests and biopsy of the bone marrow.

Treatment: Chemotherapy is the most effective method of treating leukemia. Various anticancer drugs are used, either in combinations or as single agents. Transfusions of blood components and antibiotics are used as supportive treatments. To illuminate hidden cells, therapy of the central nervous system has become standard treatment, especially in acute lymphocytic leukemia. Under appropriate conditions, bone marrow transplantation may be useful in the treatment of certain leukemias.

Survival: The 5-year survival rate for patients with leukemia is 38%, due partly to very poor survival of patients with some types of leukemia such as acute granulocytic. Over the last 30 years, however, there has been a dramatic improvement in survival of patients with acute lymphocytic leukemia; from a 5-year survival rate of 4% for people diagnosed in the early 1960s to 28% in the early 1970s to 52% in the mid-1980s. In children, the improvement has been from 4% to 73%.

Lymphoma

New Cancer Cases: An estimated 58,700 new cases in 1995, including 7,800 cases of Hodgkin's disease and 50,900 non-Hodgkin's lymphoma. Since the early 1970s, incidence rates for non-Hodgkin's lymphoma have increased over 65%. Incidence of Hodgkin's disease has declined over the same time period, especially among the elderly.

Mortality: An estimated 24,150 deaths in 1995 (non-Hodgkin's lymphoma, 22,700; Hodgkin's disease, 1,450).

Signs and Symptoms: Hodgkin's disease: enlarged lymph nodes, itching, fever, night sweats, and weight loss. Fever can come and go in periods of several days or week Non-Hodgkin's lymphoma: enlarged lymph nodes, anemi weight loss, and fever.

Risk Factors: Risk factors are largely unknown but part involve reduced immune function and exposure certain infectious agents. Persons with organ transplan are at higher risk due to altered immune function. Hum immunodeficiency virus (HIV) and human T-cell leukemi lymphoma virus-I (HTLV-I) are associated with increas risk of non-Hodgkin's lymphoma. Burkitt's lymphoma Africa is partly caused by the Epstein-Barr herpes vir Other possible risk factors include occupational exposur to herbicides and perhaps other chemicals.

Treatment: Hodgkin's disease: chemotherapy and radi therapy are useful for most patients. Non-Hodgki lymphoma: early stage, localized lymph node disease c be treated with radiotherapy. Patients with later sta disease often benefit from the addition of chemothera New programs using highly specific monoclonal antibodi directed at lymphoma cells, and improved techniques bone marrow preservation, are under investigation selected patients who relapse after standard treatment.

Survival: Survival rates vary widely by cell type and sta of disease. The overall 5-year survival rate for Hodgki disease is 79%. The overall 5-year survival for n Hodgkin's lymphoma has steadily improved, and in t past 30 years has increased from 31% to 52%.

Skin Cancer

New Cancer Cases: Over 800,000 cases a year of hig curable basal cell or squamous cell cancers. They are m common among individuals with lightly pigmented sk The most serious skin cancer is melanoma, which v be diagnosed in about 34,100 persons in 1995. Since 19 the incidence rate of melanoma has increased about per year. Incidence rates are over 10 times higher am whites than blacks. An additional 16,000 invas nonmelanoma skin cancer cases will occur in 1995, mo sarcomas, including Kaposi's sarcoma.

Mortality: An estimated 9,300 deaths this year, 7, from malignant melanoma and 2,100 due to other s cancers.

Signs and Symptoms: Any unusual skin conditi especially a change in the size or color of a mole or ot darkly pigmented growth or spot. Scaliness, oozi bleeding, or change in the appearance of a bump or nod the spread of pigmentation beyond its border, a cha in sensation, itchiness, tenderness, or pain.

Risk Factors: Excessive exposure to ultraviolet radiat fair complexion; occupational exposure to coal tar, pi creosote, arsenic compounds, or radium.

Prevention: The sun's ultraviolet rays are stron

Prevention

Researchers estimate that if everything known about the prevention of cancer was applied, up to two-thirds of cancers would not occur. The following are areas in which certain health choices might reduce an individual's risk of cancer.

Smoking

Cigarette smoking is responsible for 90% of lung cancers among men and 79% among women—about 87% overall. Smoking accounts for about 30% of all cancer deaths. Those who smoke two or more packs of cigarettes a day have lung cancer mortality rates 12 to 25 times greater than nonsmokers. (See Tobacco Use, p. 22.)

Nutrition and Diet

Research is showing the important role nutrition plays in preventing cancer. Evidence indicates that people may reduce their cancer risk by observing these nutrition guidelines:

1. Maintain a desirable weight. Individuals 40% or more overweight have an increased risk of colon, breast, prostate, gallbladder, ovary, and uterus cancers. Physicians can recommend a suitable diet and exercise regimen to help maintain appropriate weight and body fitness.

2. Eat a varied diet. A varied diet eaten in moderation offers the best hope for lowering the risk of cancer.

3. Include a variety of vegetables and fruits in the daily diet. Studies have shown that daily consumption of vegetables and fresh fruits is associated with a decreased risk of lung, prostate, bladder, esophagus, colorectal, and stomach cancers.

4. Eat more high-fiber foods such as whole grain cereals, breads, and pasta; and vegetables and fruits. High-fiber diets are a healthy substitute for fatty foods and may reduce the risk of colon cancer.

5. Cut down on total fat intake. A diet high in fat may be a factor in the development of certain cancers, particularly breast, colon, and prostate.

6. Limit consumption of alcohol, if you drink at all. Heavy drinking, especially when accompanied by cigarette smoking or smokeless tobacco use, increases risk of cancers of the mouth, larynx, throat, esophagus, and liver.

7. Limit consumption of salt-cured, smoked, and nitrite-cured foods. In areas of the world where salt-cured and smoked foods are eaten frequently, there is higher incidence of cancer of the esophagus and stomach. Modern methods of food processing and preserving appear to avoid the cancer-causing byproducts associated with older methods of food treatment.

Sunlight

Almost all of the more than 800,000 cases of basal and squamous cell skin cancer diagnosed each year in the US are sun-related (ultraviolet radiation). Epidemiologic evidence shows that sun exposure is a major factor in the development of melanoma and that incidence increases for those living near the equator. (See Selected Cancers: Skin Cancer, p. 15.)

Alcohol

Oral cancer and cancers of the larynx, throat, esophagus, and liver occur more frequently among heavy drinkers of alcohol especially when accompanied by smoking cigarettes or chewing tobacco. (See Selected Cancers: Oral Cancer, p. 17.)

Smokeless Tobacco

Use of chewing tobacco or snuff increases risk of cancer of the mouth, larynx, throat, and esophagus and is a highly addictive habit. (See Selected Cancers: Oral Cancer, p. 17.)

Estrogen

Estrogen treatment to control menopausal symptoms can increase risk of endometrial cancer. However, including progesterone in estrogen replacement therapy helps to minimize this risk. Consultation with a physician will help each woman to assess personal risks and benefits. Continued research is needed in the area of estrogen use and breast cancer. (See Selected Cancers: Uterus [Cervix] Cancer, p. 12.)

Occupational Hazards

Exposure to several different industrial agents (nickel, chromate, asbestos, vinyl chloride, etc.) increases risk of various cancers. Risk of lung cancer from asbestos is greatly increased when combined with cigarette smoking. (See Environmental Cancer Risks, p. 20.)

Ionizing Radiation

Excessive exposure to ionizing radiation can increase cancer risk. Most medical and dental x-rays are adjusted to deliver the lowest dose possible without sacrificing image quality. Excessive radon exposure in homes may increase risk of lung cancer, especially in cigarette smokers. If levels are found to be too high, remedial actions should be taken.

Environmental Cancer Risks

The environmental causes of cancer include exposures in the community or workplace settings, as well as exposures determined by individual lifestyle choices (smoking, diet, medications, etc.).

The degree of cancer hazard posed by such risks depends on the concentration or intensity of the carcinogen in the environment and the exposure dose a person receives. These factors in combination create a range of risk. For example, in situations where high levels of carcinogen are present and where exposures are extensive, significant hazards may exist, but where concentrations are low and exposures limited, hazards are often negligible.

Risk Assessment

To protect people against unsafe exposures, risks should be assessed so that appropriate environmental standards can be set. **Risk assessment** is a two-step process: identifying the toxic properties of potential oncogenic hazards and measuring the extent of human exposure.

The first step, **hazard identification,** evaluates the chemical or physical nature of hazards and their oncogenicity in observed clinical and epidemiologic studies and in laboratory tests using animals or cell systems. Special attention is given to any evidence suggesting that cancer risk may increase with dose (dose-response relationships).

The second step, **exposure measurement,** determines the levels of hazards in the environment (air, water, food, etc.) and the extent to which people are actually exposed (how much they eat of a particular food, use a particular water source, etc.). Knowledge of how the body absorbs, metabolizes, and excretes chemicals or is exposed to radiation sources is essential to determine accurately the actual carcinogenic dose delivered to humans.

Unfortunately, evidence of risk for most potential carcinogens usually rests on the results of high-dose animal experiments or on human observations where high-dose exposures have occurred. To use such information in setting human safety standards, scientists must extrapolate from animals to humans and from high-dose to low-dose conditions. Both extrapolations involve much uncertainty; therefore conservative assumptions are used so that risk assessment will err on the side of safety. For cancer safety standards, only increased risks of one case or less per million persons over a lifetime are usually accepted.

Safety standards developed in this way for chemical or radiation exposures are the basis for federal regulatory activities at the Food and Drug Administration, the Environmental Protection Agency, and the Occupational Safety and Health Administration. The application of laws and procedures by which standards are implemented and risks are controlled is called **risk management.**

Chemicals and Radiation

Not all chemicals or all forms of radiation cause cancer. Only a limited number of chemicals (for example, benzene, asbestos, vinyl chloride, arsenic, aflatoxins) show definite evidence of human carcinogenicity or are probable human carcinogens based on animal experiments (for example, chloroform, dichlorodiphenyltrichloroethane [DDT], formaldehyde, polychlorinated biphenyls [PCBs], polycyclic aromatic hydrocarbons). The only forms of radiation proven to cause human cancer are ionizing radiation (for example, x-rays, radon, cosmic rays) and ultraviolet radiation (principally UV-B radiation).

Unproven Risks

Public concern about environmental cancer risks often focuses on risks for which no carcinogenicity has been proven or on situations where known carcinogen exposures are at such low levels that risks are negligible. For example:

1. Non-ionizing radiation. Electromagnetic radiation at frequencies below ionizing and ultraviolet levels has not been shown to cause cancer. While some epidemiologic studies suggest associations with cancer, others do not, and experimental studies have not yielded reproducible evidence of carcinogenic mechanisms. Low-frequency radiation includes radiowaves, microwaves, and radar, as well as power frequency radiation arising from the electric and magnetic fields associated with electric currents (often called ELF or extremely low-frequency radiation).

2. Pesticides. Many kinds of pesticides (insecticides, herbicides, etc.) are widely used in producing and marketing our food supply. While some of these chemicals cause cancer at high doses in experimental animals, the very low concentrations found in some foods are generally within established safety levels. Environmental pollution by slowly degraded pesticides such as DDT, a result of past agricultural practices, can lead to food chain bioaccumulation and to persistent residues in body fat. Such residues have been suggested as a possible risk factor for breast cancer; concentrations in tissue are low, however, and the evidence is not conclusive.

Continued research regarding pesticide use is essential for maximum food safety, improved food production through alternative pest control methods, and reduced pollution of the environment. At the same time, banning any man-made chemicals with carcinogenic potential (as required for processed foods under the 1958 Delaney Amendment of the Food and Drug Act) is unrealistic, given the very low concentrations involved and the value of pesticides in sustaining our food supply. Scientists and consumer groups stress the important health benefits of a diet which includes many fruits and vegetables in contrast to the minimal risks associated with pesticide residues.

3. Toxic wastes. Toxic wastes in dump sites can threaten

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find out about status of Dicks paper

see if Dick can send copy of his model program

ask about other published Vmax + Km values

ask Dick about the for unit risk values