CHEMECOLOGY
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# CANCER INCIDENCE DECLINING, SPECIAL REPORT INDICATES



WASHINGTON-- Contrary to widespread impressions, there is no "cancer epidemic" taking place in America today -- indeed, many scientific authorities believe that cancer incidence is dropping, according to a special report in ChemEcology, a publication of the Chemical Manufacturers Association.

Analysis of mortality trends over the past decade show that, apart from increases in lung cancer deaths linked to cigarette smoking, there has been little overall change in cancer death patterns. For people under 65, recorded mortality is dropping. For those under 45, the picture is even brighter.

Lifestyle abuses -- including what we eat and how much we drink and smoke -- are the single biggest factor in cancer causation. Many scientists believe these behavior factors may account for more than two-thirds of cancer incidence, ChemEcology explains.

Factors such as occupational exposure to cancer-causing substances and environmental pollution account for a small portion of the cancer burden. One recent analysis of cancer deaths attributes four percent to occupational exposure and two percent to pollution.

The report cites the American Medical Association's Council on Scientific Affairs, which stated: "There is no

(more)

definitive epidemiologic evidence that the United States has experienced an overall increase in the incidence of cancer related to high levels of pollutants or contaminants in the environment."

This view is shared by the American Cancer Society. Its report on a 20-year study of more than one million Americans finds that air pollution was not a major factor in causing lung cancer. "Smoking is the key," the Society concludes.

Prevention is likely to be an increasing direction for cancer research, according to one cancer authority cited in the report. Dr. John Cairns, a Harvard professor and chairman of the Scientific Advisory Board for the International Agency for Research on Cancer, thinks epidemiology will provide the key that unlocks cancer's mysteries.

"The practical experience is to try to work out what factors in our lifestyles, our customs and our habits are the cause of common cancers," Dr. Cairns explains.

In seeking cures, chemotherapy is effective with rapid-spreading and generalized forms of the disease. It is also used to supplement treatment by surgery or radiation therapy of clinically localized cancers.

Today, about 50 chemical compounds out of thousands tested have been found effective against some forms of cancer, and more are being investigated.

The American Cancer Society lists 14 cancers that a few decades ago had poor prognoses. Today, ACS states they are being cured in many cases, largely because of advances in chemotherapy.

Dr. Philip Handler

# No Cancer Epidemic, NAS President Says

*These comments on risk assessment by Dr. Philip Handler, president of the National Academy of Sciences, are excerpted from "The National Research Council in 1979."*

*Dr. Handler, a biochemist, has been president of the academy since 1969.*

Concern for the possibility of environmentally induced cancer has become the predominant theme of much risk assessment.

The assertion that virtually all cancer derives from environmental factors was made in consequence of the disparate geographic distribution of diverse forms of cancer combined with the absence of any evidence that predisposition to a locally frequent form of cancer is associated with a specific ethnic group.

The environmental postulate has been confused with the possibility of carcinogenesis due to the thousands of man-made chemicals recently introduced into the economy and hence into the environment. It is, perhaps, conceivable that a "cancer time bomb" has been implanted into human affairs. But if so, it has not yet erupted.

On the evidence, the environmental postulate must refer to the natural, not to the man-made, world. Only a very tiny fraction of all current deaths due to cancer, in the United States or elsewhere, could be due to man-made chemicals.

Moreover, only one or two percent of cancers can be traced to occupational exposure in such workplaces as coal mines, asbestos mines, and factories; "pollutants" of all sorts may contribute to — rather than cause — perhaps five percent of all cancers. Radiation exposure, principally medical use of X-ray, occasions no more than one percent of all cancers.

Indeed, the United States is not suffering an "epidemic of cancer," it is experiencing an "epidemic of life" — in that an ever greater fraction of the population survives to the advanced ages at which cancer has always been prevalent.

The age-corrected incidences of only two forms of cancer have changed significantly in the last half-century: The incidence of cancer of the lung has increased markedly due to cigarette smoking, which has less dramatically contributed to cancer of other organs. Meanwhile, the incidence of cancer of the stomach, formerly as prevalent in the United States as today in Japan, has declined dramatically for reasons that are quite unknown. (We've been doing something right and don't know what it is!) The overall, age-corrected incidence of cancer has not been increasing; it has been declining slowly for some years.

Those who first directed attention to the role of "environmental factors" in influencing the geographic distribution of the various forms of cancer — notably John Higginson of the International Agency for Research on Cancer in Lyons, France — are well aware of these circumstances.

However, they associate that varied distribution with neither man-made chemicals, radiation, industrial pollutants, nor natural "initiators" but, rather, with differences in regional "lifestyles" — for example, the relative consumption of such items as tobacco, coffee, alcohol, dietary fat, and fiber, or voluntary exposure to the sun. Indeed, neither any specific cancer nor total cancer incidence is correlated with the degree of industrialization or energy consumption per capita of different world regions.

Nevertheless, regulatory policy in the United States has heavily emphasized measures to minimize exposure to man-made chemicals which laboratory tests have proved carcinogenic in rodents. Quite apart from the social and economic costs of such measures, they are addressed to a relatively minor fraction of the current mortality rate from cancer of all sorts.

To quote Higginson, "There is no justification for ignoring minor hazard because a greater evil exists, but, conversely, there is no justification for overemphasizing a minor risk to the detriment of control of a major hazard."

It should be clear that man-made chemicals and radiation are, in this sense, a relatively minor hazard that must not distract the scientific community from the task of understanding fundamental cancer biology and addressing the difficult, complex problem of the influence of "lifestyle" factors on the incidence and tissue site distribution of cancer.

# Pathologist Analyzes Cancer Theories

The urban environment, not air pollution or occupational exposure to chemicals, is a major factor in causing cancer, Dr. Harry Demopoulos, Associate Professor of Pathology at the New York University Medical Center believes.

Data from the National Cancer Institute's Third National Cancer Survey support his hypothesis, he told a meeting of the Synthetic Organic Chemical Manufacturers Association.

That survey examined seven cities with combined populations of 16 million. Four cities — San Francisco, Dallas, Minneapolis, and Atlanta — are "clean" cities without heavy, polluting industry. Three cities — Detroit, Pittsburgh and Birmingham — are "dirty," heavily industrialized cities.

Overall cancer rates in white and black males were eight percent lower in "dirty" than in "clean" cities, the survey found.

"If the hypothesis was correct that industrial air pollution or urban air pollution were a major factor in cancer causation, we would have expected to have seen that four "clean" cities had lower cancer rates than the three "dirty" cities," Dr. Demopoulos said.

In addition, the incidence of certain site-specific cancers such as lung, larynx, bladder and stomach, which have been linked with industrial worker exposure, were no higher in the industrialized cities than the nonindustrialized ones, the survey showed.

The existence of thresholds for cancer-causing substances explains why the cancer incidences in the "dirty" cities were no higher than those for the "clean" cities, Dr. Demopoulos claimed.

"Now, clearly the exposures, although seemingly horrendous in the three "dirty" cities, were well below threshold levels because there was no effect. Recall, the Third National Cancer Survey showed eight percent less cancer in the "dirty" cities," he noted.

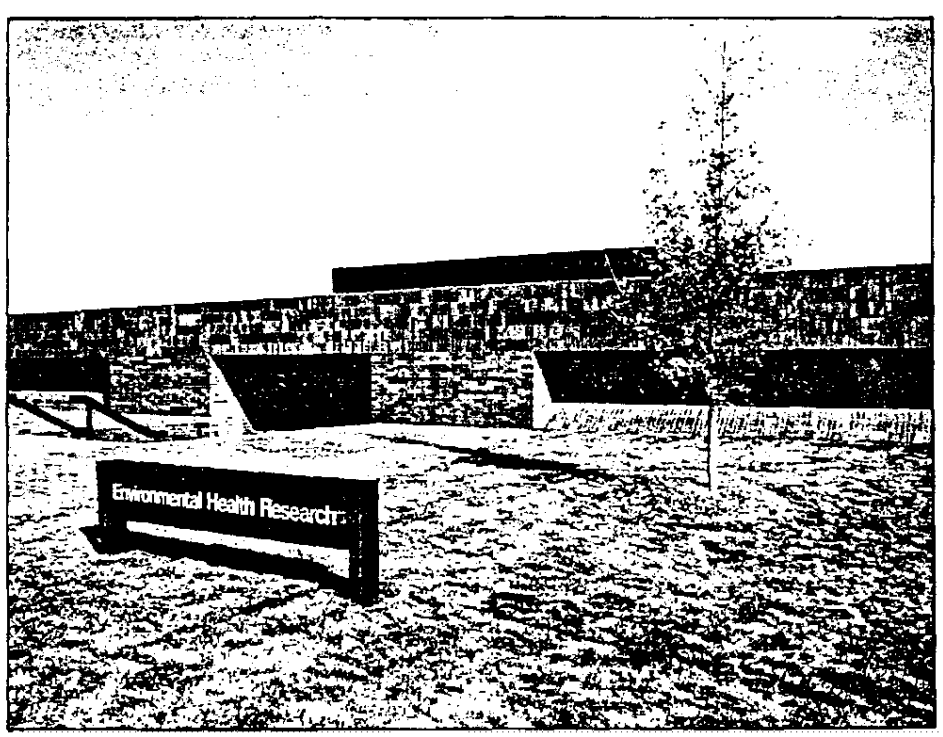
University medical researchers are disturbed by the federal government position concerning cancer causes, Dr. Demopoulos said. If society follows the federal government approach that blames industry and pollution of air and water for cancer incidence, it will be led away from answers that already are being defined through laboratory and epidemiological research.

"Whoever attempts to lead the nation toward less cancer had better be correct, because the selection of the wrong path is the equivalent of leading millions of Americans to certain death," Dr. Demopoulos warned.

Some predominant causes of cancer were analyzed and ranked at a series of symposia sponsored by the New York Academy of Sciences and the American Health Federation, with cooperation of the American Cancer Society. Looking at these causes, we do know how to avoid certain potentially cancer-causing situations, Dr. Demopoulos said.

Approximately 35 percent of cancer deaths are attributable to smoking high-tar cigarettes and excessive consumption of distilled liquor; 45 to diet; five percent to occupational exposure, with the remainder related to factors such as exposure to background radiation, pre-existing medical conditions and certain drugs, he said.

There is evidence that the occupational exposure deaths may have peaked and are on the way down since controls have been imposed on previously unregulated chemicals. Certain types of cancer, such as those related to asbestos and vinyl chloride exposure, have reached a plateau, Dr. Demopoulos noted.




# Mobay Opens Environmental Health Lab

An environmental health laboratory for research and testing of existing and new chemical products has been opened by Mobay Chemical Corporation.

The 60,000 square-foot-facility will help meet the need for increased toxicology data required by government agencies about many chemical industry products.

"The rapid escalation of requirements for toxicology data has placed a strain on private and public facilities that conduct toxicology research. This made it obvious that, from a practical and scientific standpoint, the corporation's requirement in this area could be met only with a new facility," Dr. K. M. Weis, Mobay's president and chief executive officer, said.

The OCAW brief attacked three provisions contained in the present lead standard. The provision giving certain industries five years to achieve the airborne exposure limit for lead was attacked as contravening the language of the Occupational Safety and Health Act and undermining the Act's fundamental purpose. According to the OCAW brief, the Occupational Safety and Health Administration, by statute, is required to provide workers with the maximum protection that is economically viable, but the Secretary of Labor instead attempted to "minimize the costs to industry by seriously sacrificing worker health and safety."

The present medical removal provision is claimed to be deficient by OCAW, because it allows workers suffering adverse effects from previous lead exposure to continue to be subjected to high lead concentrations for long periods of time. OCAW blames this deficiency on the secretary's impermissible policy of minimizing the employer's economic costs rather than simply ensuring the feasibility of adequate protections.

The Secretary of Labor violated his "statutory obligation to constantly upgrade and improve occupational health standards" by exempting the construction industry from compliance with the lead standard, the OCAW brief asserted. The union requested that three provisions of the lead standard — the five year compliance delay, the medical removal provision, and the exemption of the construction industry — be set aside.

The Act imposes on the secretary the affirmative duty to make regulations that most adequately assure that no worker will be affected by lead exposure, the union's brief reasoned. The five-year delay in compliance and the medical removal provision both violate the Act by allowing continued exposure to harmful amounts of lead, according to OCAW.

Finally, OCAW contended that the secretary's decision to deny construction workers the protection of the lead standard was arbitrary, irrational and inconsistent with the Secretary's statutory obligations. OCAW claimed that there is no evidence that lead poses no hazard to construction workers or that the lead standard would be peculiarly difficult to implement in the construction industry. In the absence of such evidence, the secretary's decision to exempt the construction industry was arbitrary and irrational, OCAW concluded.

### Capital Legal Foundation

An amicus brief in the lead standard case was submitted by the Capital Legal Foundation, a nonprofit public interest organization organized in 1977 to engage in nonpartisan legal research.

The foundation's brief contended that the secretary does not have the requisite statutory authority to require medical removal protection. This argument was based on the fact that a provision authorizing the Occupational Safety and Health Administration to adopt standards similar to the medical removal protection provision was dropped from the Occupational Safety and Health Act during congressional debate. The amicus brief also attacked the medical removal protection provision as being an attempt to supersede or supplant state workers' compensation laws.

## Safety Hazards

### INDUSTRY APPROVES PROPOSED STANDARD FOR MULTI-PIECE RIM WHEELS AT HEARING

Industry groups voiced their approval of the Occupational Safety and Health Administration's proposed standard for servicing of multi-piece rim wheels at a June 19 hearing.

The proposed standard would regulate the tire servicing employees, would establish a s procedure for servicing multi-piece rim wheels quire mandatory use of restraining devices, develop criteria for interchangeability of rim co ponents (Current Report, April 26, p. 1689; text, p. 1702).

The American Trucking Association stated that the proposal is "acceptable," but that the wording should be clarified to reflect more accurately the nature of the hazard as it relates to the proposed requirement. The association suggested that the proposal's definition of a restraining device is "unduly restrictive" and does not allow the use of alternative protective measures.

The Firestone Tire and Rubber Company testified that the proposed standard is "superior" and reflects "a conscientious and in-depth consideration of the problems of truck and bus rim and wheel maintenance." In addition, Firestone noted that the economic cost is "modest."

Firestone suggested that the proposal should be modified to include 15 inch rims and wheels rather than only rims 16 inches and larger as currently proposed. The company also asserted that the standard should prohibit welding of a rim unless the tire is deflated and removed from the rim beforehand.

The National Wheel and Rim Association stated that it "enthusiastically supports" the proposal as did the Rubber Manufacturers Association.

The Rubber Manufacturers Association, which originally petitioned OSHA in 1976 for adoption of the standard, urged OSHA to act promptly in deciding on a final rule.

Private citizens, stated their approval of the proposal, but added that OSHA should require comprehensive training in vocational schools for the servicing of multi-piece rim wheels.

The comment deadline is July 6 and issuance of the final standard is expected by October 1, according to OSHA project officer John Klocko.

## Carcinogens

### NO CARCINOGENESIS THRESHOLD FOUND AT LOW DOSAGE LEVELS IN NCTR STUDY

The theory that there is no "threshold dose level" below which a carcinogenic substance would be safe was supported by a recently completed carcinogenesis study using more than 24,000 mice.

The study, conducted by the Food and Drug Administration's National Center for Toxicological Research (NCTR) "has provided a massive and overwhelming experimental profile and data base which lends support to regulatory policies," according to Thomas Cairns, NCTR acing director.

One such regulatory policy is the Occupational Safety and Health Administration's proposal for regulating carcinogens in the workplace, which states "there is presently no means to determine a safe exposure level to a carcinogen."

"Explanation of the dose-response relationship at low doses is fundamental to the regulatory agencies' posture that there is no threshold level below which a carcinogen cannot exert its carcinogenic effect," Cairns said at June 15 symposium in Washington, D.C., announcing the preliminary results of the study.

The study, using the carcinogen 2-acetylaminofluorine (2-AAF), was designed to determine with precision the effects of a known carcinogen at low dose levels, according to NCTR.

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Among significant findings of the study were the following:

► No threshold response effect was found at the lowest dose level used in the study.

► The mice in the study developed a significant number of liver tumors which did not appear until after 18 months, at which point many current bioassay tests are ended.

► The incidence of bladder tumors in the dosed mice dropped substantially when feeding of 2-AAF was stopped after the first nine months of the study. However, liver tumors continued to develop after 18 months, even when dosage was ended at nine months.

**800 Cans of Diet Soda**

The concept of a threshold dose level has been debated by regulatory agencies and industry groups concerned with cancer risk assessment.

The controversy arises from the standard practice of testing suspected carcinogens by administering massive doses of a substance to a relatively small number of test animals. The results of such experiments are extrapolated to predict the effect of very low exposure levels on human populations.

Such tests, Cairns said, "have been challenged on scientific grounds and are confused, even distrusted, by laymen who see only the differences between the experimental process and ordinary life situations."

"The '800 cans of diet soda' perception of toxicological research and its validity is undoubtedly a serious impediment to the credibility of our research and the regulatory decisions that may be based on it," Cairns said.

NCTR scientists are still examining the data generated by the study. Detailed results will be reported in scientific journals.

For further information, contact Jeffrey Staffa, Associate Director for Research Operations and Planning, NCTR-FDA, Room 9-39, 5600 Fishers Lane, Rockville, Md., 20857, telephone (301) 443-3155.

## Coke Ovens

### EMPLOYER MUST FURNISH CLOTHING FOR COKE OVEN PROTECTION, OSHA SAYS

The Occupational Safety and Health Administration June 19 reaffirmed its requirements that employers must provide protective clothing for workers exposed to coke oven emissions.

In response to a complaint by workers at the Jones and Laughlin Steel Corporation, Aliquippa, Pa., OSHA stated that the coke oven emissions standard at 29 CFR 1910.1029(h)(2)(i) (Reference File, 31:8461) requires employers to provide clean and dry protective clothing at least weekly.

The Jones and Laughlin workers petitioned OSHA for a waiver from the requirements to allow them to purchase and launder their own clothing because they feared the standard did not provide for enough changes of clothing to protect them adequately.

OSHA noted that the standard requires "appropriate" clothing and equipment and special changing rooms. The standards also prohibits workers from taking protective clothing home with them. It also stated that weekly changes of clothing are the minimum requirement. Nothing in the regulation prohibits more frequent changes, the agency added.

## Oregon

### STATE ROLLOVER PROTECTION RULE FACES TENTATIVE REJECTION BY OSHA

Oregon's job safety and health standards requiring rollover protection for agricultural tractors are not as effective as federal regulations and probably will be rejected by the Occupational Safety and Health Administration, OSHA announced June 22.

In its notice of tentative intention to reject the state rules (44 FR 36506), OSHA indicated that Oregon's exemption of track-type tractors was deficient. Although hearings established that there never had been a rollover of these types of tractors in the state, OSHA said evidence indicated that such rollovers had occurred elsewhere and had resulted in deaths and serious injuries.

OSHA noted that it will consider exceptions filed in quadruplicate by persons who participated in public hearings on the subject within 30 days. An additional 15 days will be allowed for objections to exceptions, also to be submitted in quadruplicate. Submissions should be made to the Assistant Secretary of Labor, OSHA, U.S. Department of Labor, Third St. and Constitution Ave., Washington, D.C. 20210.

The OSHA notice of tentative intent to reject the Oregon standard appears in the Full Text section of this Current Report.

## Program Directives

### OSHA DETAILS REVISED PROCEDURES FOR TEMPORARY LABOR CAMP INSPECTIONS

Revised procedures for the handling of temporary labor camp and migrant housing facility inspections were detailed by the Occupational Safety and Health Administration in a June 15 instruction.

OSHA Instruction CPL 2.37, from the Office of Field Coordination, clarified the procedures for temporary labor camp inspections in light of the Employment and Training Administration's migrant housing standard at 20 CFR 620 and OSHA's own regulations at 29 CFR 1910.142.

Before inspections are undertaken, employers are to be asked which standards, or variances therefrom, they prefer as applied to the temporary facilities. OSHA noted that, where the ETA standards govern, hazards which violate the ETA standards but comply with OSHA standards at 1910.142 are not to be cited. A possible exception would be serious violations of Section 5(a)(1) of the Occupational Safety and Health Act, the instruction noted. The ETA standards may not be cited by OSHA inspectors, the agency added, but where OSHA standards are selected or for facilities constructed after January 1, 1979, 1910.142 applies.

The OSHA instruction also recommended that compliance safety and health officers should focus on facilities and conditions which relate closely to employee safety and health during migrant housing facility inspections. Specific items of concern to the inspector include site location, adequate shelter, approved water supplies, adequate toilet facilities, first aid provisions, and facilities for laundry, handwashing, and bathing. Inspectors are required to document the age of the dwelling units; the number of units and number of occupants per unit; the size of the housing area; the distances between dwelling units and water supply, toilets, livestock, and service buildings; the identity of the employer; and the extent to which the provision of housing is related to employment, the instruction stated. If the operation of the camp is related directly to the employment of the occupants, OSHA

health, and the occupational health nurse must be able to relate the workers' environment to the community at large. The nurse must take a pro-active role rather than a reactive one by educating and encouraging employees to develop healthy lifestyles."

## DR. BUNDY RETIRES FROM U.S. STEEL, JOINS NAVY MEDICAL CENTER:

Dr. Merle Bundy has retired as Director of Industrial Medicine for United States Steel Corp. and will become Medical Officer at the Naval Regional Medical Center, San Diego. Dr. Joseph J. Schwerha is promoted to succeed Dr. Bundy.

Dr. James A. Mackay, former medical director of bankrupt White Motor Co., has joined the medical department of United States Steel's Duquesne (Pa.) Works.

Joel Broida and Jack Shad, both toxicologists at NIOSH headquarters in Rockville, Md., have joined the National Cancer Institute.

Hank Wilson has been appointed liaison with labor for NIOSH, headquartering in Washington (not Rockville).

E. Ross Buckley, from the Justice Department, has been appointed general counsel of the Occupational Safety & Health Review Commission.

## SCHWEIKER DISAVOWS CALIFANO'S OCCUPATIONAL CANCER ESTIMATES:

HHS Secretary Richard Schweiker has disavowed the report of his predecessor, Joseph Califano, on the estimates of cancer related to occupational factors. In a letter to the Asbestos Information Association, Schweiker called the estimates too high.

The Califano "Estimates Paper," prepared by a group of HHS scientists and published in 1978, calculated that more than 25 percent of all cancer deaths in the U.S. could be attributed to occupational factors and that 17 percent of current cancer deaths were associated with previous exposure to asbestos. The estimates, presented by Califano to an AFL-CIO Conference on Occupational Health and Safety, were widely publicized and caused counter-reaction by much of industry and a number of scientists.

B.J. Pigg, Executive Director of the Asbestos Information Association, had written to Schweiker to urge that the Estimates paper be formally withdrawn by the Department from the record in OSHA's current review of the genetic cancer policy. It had been previously used by OSHA in development of the cancer policy, although it has never been a formal HHS position.

In his reply of April 29, Schweiker said that "more recent estimates imply that the original estimates of cancer associated with asbestos exposure were too high. Current estimates for over-all workplace-associated cancer mortality vary within a range of five to fifteen percent. Recent estimates for the contribution of asbestos to cancer mortality range from one to three percent."

"In sum," he continued, "the current state of knowledge does not allow us to give a precise estimate; responsible scientists simply do not agree on a single estimate of cancers caused or contributed to by environmental factors, or on the contribution of asbestos in particular.

"Our mutual concern for public health dictates that we all take appropriate action to prevent exposures to hazardous substances. Whatever the exact contribution of occupational factors to cancer, these factors remain a source of concern to this Department. Thus, the central issue is that all of us must do what we can to make the workplace less hazardous, particularly for those who continue to work with materials like asbestos for which society has not yet found a substitute.

"As you know, OSHA has announced its intention to reevaluate and revise the Generic Cancer Standard. This will provide an opportunity to consider the most recent estimates and scientific judgment about occupationally-related cancer."

In his letter, Pigg said that the claims made by the original "Estimates Paper" have been repeatedly discredited by the scientific community. "Indeed," he said, "all but two of its nine co-contributors have expressed doubt as to the Paper's correctness. It nonetheless continues to receive prominent attention and is often referred to as the official government position on occupation and cancer.

"We write to you now because the 'Estimates Paper' has once again been comprehensively criticized in the June issue of the Journal of the National Cancer Institute. An extensive review of the causes of

ancer commissioned by the U.S. Congress' Office of Technology Assessment, and written by the eminent British scientists Sir Richard Doll and Richard Peto, concludes the Paper 'should not be regarded as a serious contribution to scientific thought and should not be used or cited as if it were'."

## EVERYONE WANTS TO CUT HEALTH CARE COSTS—BUT NOW?:

The name of the game today in occupational medicine is cutting health care costs—so it was appropriate that the American Occupational Medical Association meeting in Toronto have a panel on the subject for what turned out to be a commendable exchange of views—views not always known to occupational health practitioners.

For example, Steven Sieverts, vice president for institutional affairs and health care cost containment of Blue Cross and Blue Shield of Greater New York, described ways his organization—frequently criticized for providing open-ended hospital cost protection without questions—said that's all undergoing a change.

He declared that inpatient hospital utilization is going down, fewer people are admitted to hospitals and they are having shorter stays. In addition, many of the marginal short-stay cases are being treated outside the hospital.

"We are also doing a substantial number of things with a positive thrust. For example, we are in the midst of a substantial effort to promote ambulatory surgery as an alternative to inpatient hospital care," he said. "Other less costly and more appropriate alternatives to hospital care also are being encouraged by our plan, such as home health which Blue Cross and Blue Shield of Greater New York pioneered during the 1950s and 1960s, pre-admission diagnostic testing, and the like."

Dr. Thomas J. Doyle, assistant vice president and chief medical officer of Consolidated Edison of New York, said that business groups are being established in several communities and coalitions are being formed to investigate the causes of continuing cost increases and to exert, collectively, an influence on cost containment.

He cited the experience of the New York Business Group on Health, formed with the early assistance of the New York State Chamber of Commerce and Industry following successful programs of groups working together in Philadelphia.

Dr. Doyle, who is chairman of the New York Business Group on Health's Data Analysis Committee, reviewed extensive 1980 Blue Cross data and an annual comparison is planned to monitor trends and confirm progress. Each company member of the NYBGH will have access to a manual being written by the committee to assist them in evaluation of the data from their own company. The manual will also provide information for data comparison guidance with other groups.

"Benefit plan design can be modified as more cost effective alternatives are identified," he said. "The participation of physicians, health benefit administrators and others involved in health care planning through the NYBGH has been an exciting new activity. We believe the companies involved will have a more healthy workforce through more cost-efficient over-all health care as a result of this participation."

Dr. C. Craig Wright, Director of Health Services for Xerox, made the point that multiple health promotion programs in a company can contribute significantly to reduction of health care costs. Worksite and leisure time physical fitness activities, lifestyle and behavior modification programs are all part of a larger wellness concept which reduces illness and therefore health care costs, he said.

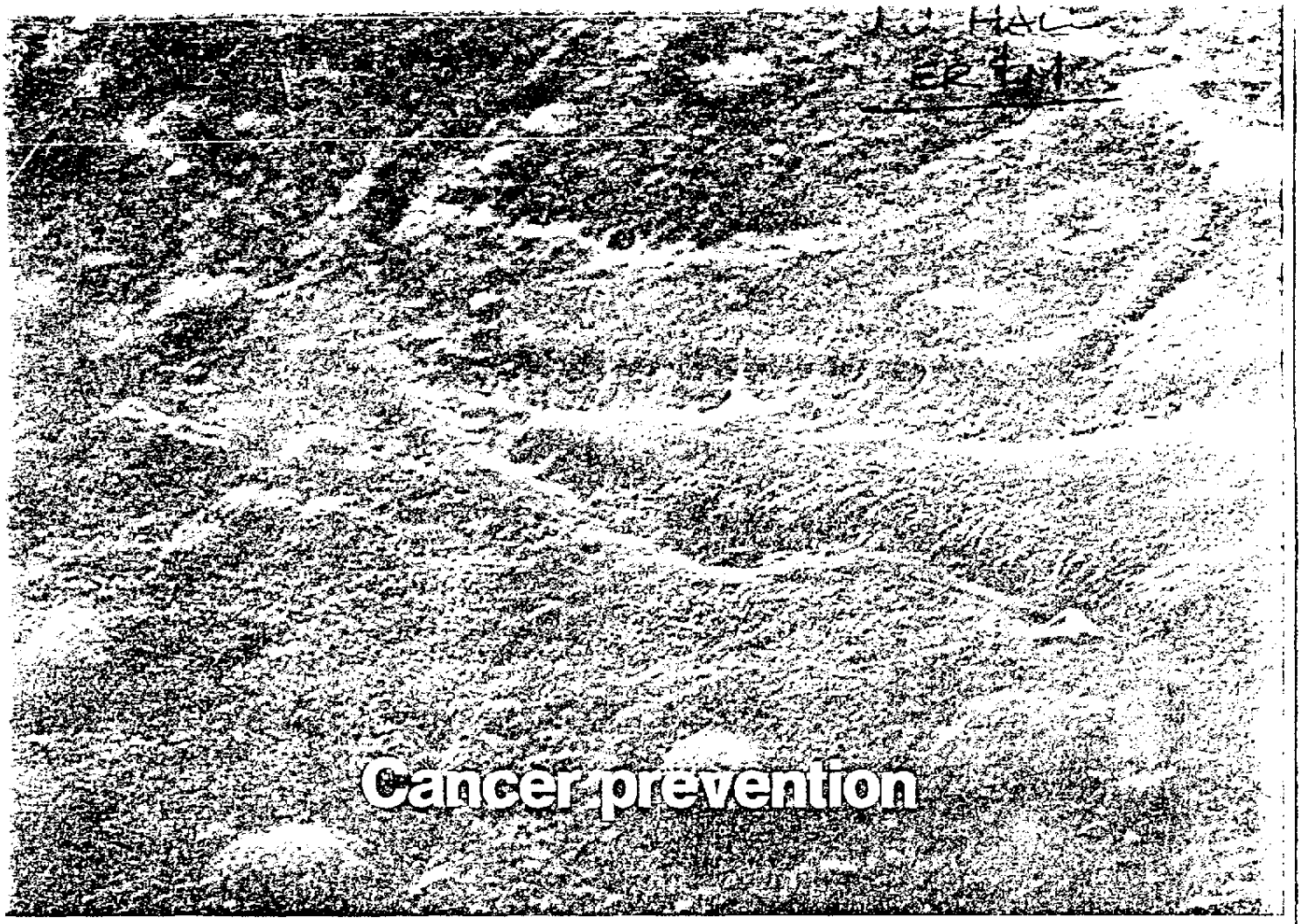
They also have a positive effect on employee self-image and sense of security, job satisfaction, company loyalty and identification, morale and productivity, Dr. Wright said.

Note: A seminar-workshop on private philanthrophy in health, to explore the private sector's support for health-related projects and services, will be held June 23 at the New York Telephone Co., under sponsorship of the New York Business Group in Health. Details available from the Group, 200 Madison Ave., 3rd Floor, New York, N.Y. 10016; (212) 561-2059.



## NIOSH SAYS COAL DUST AIR SAMPLING TECHNIQUES MAY BE FAULTY:

The possibility that coal miners may be exposed to more coal dust than the Mine Safety and Health Administration standard is suggested by a letter from NIOSH questioning MHSA's method of air sampling. The letter resulted from a request by MSHA to conduct a statistical study of a larger investigation of the



# Cancer prevention

## A full pound of cure is still a long way off

Cancer is a general name attached to a variety of diseases that afflict man and animals, and in which affected tissues enter a stage of uncontrolled growth that can lead to premature death. Cancer, in all its forms, accounts for approximately 17% of deaths in the United States (Table 1). Data derived from epidemiology, from migrant studies, and from occupational health suggest that 80 to 90% of human cancers are due to environmental factors, most of which are made by man and, thus, can be prevented by man.

We often note a fairly direct connection between exposure to certain environmental factors (e.g., radiation or chemical) and cancer. However, the time element frequently causes controversy about this relationship. For example, a workman exposed to carcinogenic dyestuff intermediates for 5 years may show bladder cancer 25–35 years later. A heavy cigarette smoker may have overt lung cancer only after 30–40 years of smoking. It is thus that cancer, with its long latent period between exposure and the actual occurrence of the disease, is quite different from more acutely toxic events.

Occupational exposure is responsible for a relatively small proportion of the annual death rate from cancer (Table 2). However, such cases have, over a period of time, received a lot of public notice. Hopefully, increased awareness, newer means of detecting carcinogens, and the introduction of preventive methods will eliminate both—occupational cancer risks and those caused by our life styles.

### Chemical carcinogens

In recent years we have learned a great deal about chemical carcinogens. We have data on harmful vs. innocuous dosages, dose-response curves, threshold levels, or threshold limit values, relationship to mutagenesis, and the like.

Many different types of chemical structures can induce a neoplastic change, but this property may differ in animal models and in man. Chemical carcinogens can be aliphatic or aromatic compounds, straight or branched chain, saturated or unsaturated, homo- or heterocyclic, and also inorganic chemicals. They can be gases, liquids,

**Table 1. Estimated cancer deaths by sex for all sites—1975** [a]

| Site | Both sexes | Male | Female |
|---|---|---|---|
| **All Sites** | **365 000** | **199 000** | **166 000** |
| Buccal cavity & pharynx (Oral) | 8200 | 5900 | 2300 |
| Lip | 225 | 200 | 25 |
| Tongue | 1950 | 1400 | 550 |
| Salivary gland | 650 | 400 | 250 |
| Floor of mouth | 525 | 400 | 125 |
| Other and unspecified mouth | 1250 | 800 | 450 |
| Pharynx | 3600 | 2700 | 900 |
| **Digestive Organs** | 101 700 | 53 800 | 47 900 |
| Esophagus | 6500 | 4700 | 1800 |
| Stomach | 14 400 | 8500 | 5900 |
| Small intestine | 700 | 350 | 350 |
| Large intestine (colon) | 38 600 | 17 900 | 20 700 |
| Rectum | 10 600 | 5900 | 4700 |
| Liver and biliary passages | 9800 | 4800 | 5000 |
| Pancreas | 19 500 | 10 900 | 8600 |
| Other and unspecified digestive | 1600 | 750 | 850 |
| **Respiratory System** | 85 700 | 67 150 | 18 550 |
| Larynx | 3250 | 2800 | 450 |
| Lung, bronchus, trachea | 81 100 | 63 500 | 17 600 |
| Other and unspecified respiratory | 1350 | 850 | 500 |
| **Bone, Tissue, Skin** | 8600 | 4900 | 3700 |
| Bone | 1900 | 1100 | 800 |
| Connective tissue | 1700 | 900 | 800 |
| Skin | 5000 | 2900 | 2100 |
| Breast | 32 900 | 300 | 32 600 |
| **Genital Organs** | 42 700 | 19 800 | 22 900 |
| Cervix, invasive (Uterus) | 7800 | — | 7800 |
| Corpus uteri (Uterus) | 3300 | — | 3300 |
| Ovary | 10 800 | — | 10 800 |
| Other female genital | 1000 | — | 1000 |
| Prostate | 18 700 | 18 700 | — |
| Other male genital | 1100 | 1100 | — |
| **Urinary Organs** | 16 500 | 11 000 | 5500 |
| Bladder | 9400 | 6500 | 2900 |
| Kidney and other urinary | 7100 | 4500 | 2600 |
| Eye | 400 | 200 | 200 |
| Brain and central nervous system | 8500 | 4800 | 3700 |
| **Endocrine Glands** | 1650 | 650 | 1000 |
| Thyroid | 1150 | 350 | 800 |
| Other endocrine | 500 | 300 | 200 |
| Leukemia | 15 200 | 8500 | 6700 |
| **Lymphomas** | 18 600 | 10 000 | 8600 |
| Lymphosarcoma and reticulosarcoma | 7800 | 4200 | 3600 |
| Hodgkin's disease | 3500 | 2100 | 1400 |
| Multiple myeloma | 5100 | 2700 | 2400 |
| Other lymphomas | 2200 | 1000 | 1200 |
| All other and unspecified sites | 24 350 | 12 000 | 12 350 |

Incidence estimates are based on rates from National Cancer Institute Third National Cancer Survey.

[a] After Silverberg, E., and Holleb, A., Major trends in cancer 25 year survey, ca., *Cancer Journal for Clinicians*, **25**, 2.21 (1975).

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or solids. But within each of these classes the capability to cause cancer is highly structure-specific. Such chemicals may also have other diverse pathologic or toxic effects, which may or may not be related to the carcinogenic effect.

With one exception, that of inorganic arsenical compounds, chemicals that cause cancer in man also cause cancer in animal models. It is highly probable that chemicals which reliably cause cancer in animals also constitute a carcinogenic hazard to man. In recent years, bioassay in animals to detect carcinogenicity is being complemented by in vitro tests through cell and organ culture, and also by examining mutagenicity in microbiologic systems. Considerably more research is required to validate the application of in vitro systems to detect carcinogens reliably, even if such tests are used only as selective prescreens.

## Types of carcinogens

There are several types of chemical carcinogens:

- Direct-acting or ultimate-chemical carcinogens have a chemical structure such that they can cause cancer without host-mediated enzymic activation. A number of synthetic chemicals, industrial products, and drugs fall into this class.
- Procarcinogens, which constitute the majority of chemical carcinogens, require host-mediated biochemical activation to be effective. Whether or not they will cause cancer depends on such factors as species, strain, sex, age, diet, intestinal microflora, and the presence of other agents that modify the enzymic capability. As a rule, the younger the individual, the more sensitive he is. Man is heterogeneous, and different individuals present distinct response patterns.
- Cocarcinogens do not cause cancer by themselves but potentiate the effect of a carcinogen, sometimes quite dramatically. Examples are tobacco smoke, coal and petroleum tars and oils, and some natural products, such as certain bile acids. In contrast to carcinogens, cocarcinogens

do not act irreversibly. They need to be present in sizeable amounts over long periods of time. Thus their total or even partial removal from the environment would assist in delaying or preventing the development of cancer. This is a fruitful area for additional research and development.

Current concepts hold that the ultimate carcinogen, like the mutagen, is a reactive molecule that modifies DNA. The thesis that properly activated carcinogens can be detected because they are also mutagenic in microbiological systems is now under active investigation.

This raises the further question as to whether chemicals that are carcinogens also represent a long-term mutagenic hazard to man. Theoretically, this may well be so. However, the activated carcinogen would have to reach the germ cells, rather than the somatic cells, or the germ cells would have to be able to activate a procarcinogen reaching it through the circulation. There is at present no good evidence for this; much more research is required. Chemical carcinogens are often quite toxic, especially to dividing cells, and thus at higher dose levels may induce sterility.

### Threshold levels

Because active chemical carcinogens interact with DNA and yield a long-lived adduct, we consider that their effect is largely irreversible. This is in contrast to other toxic agents and drugs, the effects of which are not heritable and are reversible.

There is some new evidence that an altered DNA can be repaired through a complex, multi-enzyme system (other point metabolic activation). This may explain why under some conditions with certain chemical carcinogens a single dose does not induce cancer. However, there are other chemical carcinogens that are active after a single dose. In any case, once a cell containing abnormal DNA has undergone DNA synthesis and mitosis, the repair system fails to recognize the abnormal DNA and thus is ineffective in restoring normal DNA structure and function.

Basically for this reason individual dosages of chemical carcinogens, although given at intervals, appear to have an additive carcinogenic effect. Therefore we must combine individual doses with a time element to answer the question as to whether a threshold can or does exist, a key difference and distinction from non-carcinogenic toxicants. Instances are known where intake of low levels of carcinogens gave no cancer, which may be evidence for no-effect levels. However, more research is required to delineate this important problem and also to evaluate synergistic effects. We should consider dosage and time together when selecting individuals where exposure to carcinogens might occur. In older persons, low total doses would be possible over the remaining years of employment since the total

**Table 2. Proportion (%) of cancers at selected sites for which there are sound etiologic hypotheses[a]**

| Site | Exogenous factors | | | Congenital / Familial or | | |
|---|---|---|---|---|---|---|
| | Cultural (a)[b] | Occupational (b)[b] | Iatrogenic (c)[b] | Misc. (d)[b] | Acquired (e)[b] | Unknown (f)[b] |
| Mouth (140, 141, 143, 144) | 90 | 1 | — | 5 | — | <5 |
| Salivary Gland (142) | — | — | — | — | — | 100 |
| Esophagus (150) | 80 | — | — | 4 | <1 | ±15 |
| Stomach (151) | 4 | — | — | 1 | — | 95 |
| Colon and rectum (153, 154) | — | — | — | 1 | <1 | 99 |
| Liver (155.0) | 70 | — | — | 1 | — | 30 |
| (155.0) Africa | — | — | — | — | — | 100 |
| Lung (162) | 80 | 1–2 | — | ±8 | — | <10 |
| Breast (170) | — | — | — | — | — | 100 |
| Cervix uteri (171) | — | — | — | — | — | 100 |
| Corpus uteri (172) | — | — | — | — | — | 100 |
| Ovary (175.0) | — | — | — | — | — | 100 |
| Other female genitals (176) | — | — | <1 | — | — | 99 |
| Prostate and testis (177, 178) | — | — | — | — | — | 100 |
| Penis (179.0) | — | <1 | — | 95 | — | <5 |
| Bladder (181.0) W. industrial | 50 | 10–20 | <1 | — | — | 30–40 |
| (181.0) Africa | — | — | — | 50 | — | 50 |
| Skin (190, 191) | — | 2 | — | 80 | 10 | <8 |
| Brain tumors (193) | — | — | 2 | — | <1 | 98 |
| Leukemia and lymphoma (200, 205) | | | | | | |
| Children | — | — | <7 | — | 1 | 92 |
| Adults | — | — | <1 | — | — | 99 |

After Higginson, J., and Muir, C. S., Epidemiology. In *Cancer Medicine*. J. F. Holland and E. Frei, Eds., Lea and Febiger, Philadelphia, 1973.

[a] Unless stated, these estimates refer predominantly to a Western-type industrial population.

[b] See letters in footnote for remarks. Mouth, (a) In India almost 100% caused by betel chewing. Alcohol and tobacco are main factors elsewhere. Esophagus, (a) Alcohol and tobacco major factors in France, U.S.A.; not in S. Africa, Kazakhstan, Iran. (d) Precise role of iron deficiency still to be clarified. (e) Tylosis. Stomach, (a) Role of tobacco at cardia. Colon and rectum, (d) Mineral oil possible factor. Liver (Western industrial), (a) Alcohol predominant factor. Liver (Asia), (d) Parasitic infection. Liver (Africa), (f) Role of aflatoxin and hepatitis still not known. Lung, (a) Doll estimates up to 50% of cigarette caused cancer may also be dependent on synergistic effect of atmospheric pollution. Breast, (f) Evidence concerning influence of maternal age at birth of first child, ethnic differences, familial tendency, and possible viral transmission make it difficult to assign etiology to the previous columns. Cervix uteri, (f) Role of age at first intercourse, herpes virus, circumcision, and Jewishness not sufficiently clear. Corpus uteri, (f) Role of height, obesity, multiparity, and other factors not sufficiently clear. Other female genitals, (c) Maternal stilbestrol causing vaginal adenocarcinoma in offspring. Penis, (d) Inadequate circumcision and cleanliness. Bladder, (a) Possible role of cigarettes; (W. industrial), (b) Chemical exposure; (Africa), (d) Schistosomiasis. Skin, (d) Includes outdoor workers and others exposed to sun. (e) Pigmented spots and malignant melanoma; albinism and xeroderma pigmentosum in other skin cancers. Brain tumors, (c) Maternal and other radiation. Leukemia and lymphoma (children & adults), (c) Ionizing radiation in embryonic and adult life.

life span available for cancer development would be limited.

### Background on occupational cancer

Chemical carcinogenesis actually was first observed in the last century in people who were exposed to cancer-causing environments: the chimney sweeps of Percival Pott, the chemical factory workers of Rehn, and the individuals with radiation-induced cancer.

Because the cause of occupational cancers, once established, is usually quite definitive, it is, as a rule, possible to recommend and institute successful preventive measures. In some instances, this involves total elimination of the carcinogenic factor by omitting production. Where the material appears essential for various reasons, systems can be designed to avoid exposure to staff. This includes not only a consideration of contact by production workers but also contact by maintenance workers, repair persons, and the like.

The types of cancer seen depend on a number of factors, including structure of the chemical, age and sex of the patient, interactions with other chemicals, length and periodicity of exposure. At this time, we have demonstrated only about 20 chemicals or mixtures that cause cancer in man (Table 3). Exposure to these chemicals may be due to occupational and environmental settings as well as personal habits. We must be concerned both with the interaction between chemicals and possible synergistic effects. For example, exposure to asbestos or certain minerals and ores in the course of mining operations has led to lung cancer. Analysis of this situation, however, revealed that lung cancer was most frequent in cigarette smokers while the risk in non-smokers was greatly reduced. The latter developed mainly mesotheliomas, not bronchogenic carcinomas. Occupational exposure to asbestos enhanced several-fold the risk of developing lung cancer in smokers. The risk can be lowered quite appreciably, not only by minimizing exposure at the work place, but also by actively discouraging personnel from smoking.

There is need to examine, in detail, other such interactions. For example, in animal models, which often mirror the human situation, we have observed interactions between exposure to chlorinated solvents such as carbon tetrachloride, and other carcinogens.

### Radiation-induced cancer

Various types of radiation are carcinogenic to man. For instance, excessive exposure to sunlight causes cancer of the skin, which actually has a high incidence but a low mortality. Early detection of the lesions results in successful treatment.

Skin cancer can arise occupationally in individuals who work outside, like farmers or construction workers, or voluntarily through sunbathing.

In the past, cancer incidence was also higher in staff using x-ray equipment. In modern times, much better control of emitted dosages has been effected through various modalities, including shielding. More sensitive emulsions permit shorter exposure times, and, thus, the risk is minimized appreciably.

Populations exposed to the atomic bomb explosions in Hiroshima and Nagasaki exhibit a slightly higher incidence of cancer, particularly of lymphomas and leukemia. Considering the latent period for cancer development, it is possible that the sequelae of radiation exposure in 1945 have not fully manifested themselves at this time.

**Table 3. Carcinogens in man[a]**

| |
|---|
| Polynuclear aromatic hydrocarbons: soots, pitch, coal tar and products, creosote, shale, mineral, petroleum and cutting oils; cigarette, cigar, and pipe smoke |
| Aromatic amines: 2-naphthylamine, benzidine and derivatives, 4-biphenylamine, 4-nitrobiphenyl, auramine and magenta |
| Alkylating agents: Chlornaphazine [bis(2-chloroethyl)2-naphthylamine], mustard gas [bis(2-chloroethyl)sulfide], melphalan 4-[bis(2-chloroethylamino)-L-phenylalanine)], busulfan (1,4-butanediol dimethanesulfonate), bis(chloromethyl)-ether, dimethyl sulfate |
| Nickel carbonyl, vinyl chloride, acrylonitrile |
| Isopropyl oil manufacture (process discontinued) |
| Betel nut, nass, tobacco chewing |
| Chromates, inorganic arsenicals, asbestos |
| Radiation ionizing, ultraviolet (solar), x-rays, nuclear fission products, uranium, radon, radium, thorotrast |
| Mixtures of agents |
| Benzene? |
| Mycotoxins, Senecio alkaloids, plant carcinogens? |
| Hormonal imbalance? |
| Viruses? |

[a] After Weisburger, J. H., Chemical carcinogenesis. In *Cancer Medicine*. J. F. Holland and E. Frei, Eds., Lea and Febiger, Philadelphia, 1973.

### Major causes of cancer

The incidence and mortality from various types of cancer stem mainly from factors other than occupational exposure. Broadly, they are the result of:

- use of tobacco products, especially cigarette smoking, and
- personal dietary habits.

There are interactions which, in some cases, potentiate the effect of these two major causes. For example, lung cancer is higher in cigarette smokers living in an industrial, urban environment with consequent air pollution, compared to a similar smoker in a rural, non-polluted environment. Certain cancers are the result of interaction between two distinct types of personal habits. Cancer of the esophagus is seen upon chronic intake of some alcoholic beverages and cigarette smoking. It is much less frequent in people who only smoke, and not seen usually in people who only drink.



### Smoking and cancer

All types of tobacco smoke have carcinogenic properties. Cigarette smoking, however, leads mainly to lung cancer because addicted cigarette smokers inhale while pipe and cigar smokers who don't inhale usually develop cancer in the oral cavity and the lip. Some smoking practices, like inverse smoking, lead to cancer in the oral cavity, both as a reaction to the smoke and the heat of the burning tobacco. The relationship between smoking of cigarettes and lung cancer has been well documented. There is a proportionality between the amount smoked, the length of time smoked, and the development of lung cancer. The age at which smoking is begun is important and inversely related to risk.

In addition to lung cancer, cigarette smoking is also a major cause, perhaps acting together with diet, of cardiovascular disease. (Diet alone in some individuals suffices to elicit coronary heart disease.) Smokers also present an increased risk to the so-called minor cancers, namely cancer in the urinary bladder, pancreas, esophagus, larynx,

and other portions of the upper respiratory tract. Emphysema is often an accompanying disease.

### Prevention of tobacco-related diseases

These important diseases are quite often fatal at relatively young ages. We must therefore develop information programs to persuade the public to give up smoking altogether. Such programs must be directed to sensitive and susceptible age groups, probably best late in elementary or early on in high school. This is important, because the younger the individual is at the beginning of his or her smoking career, the more likely he or she will develop lung cancer and other adverse sequelae.

For the already addicted smokers, two courses of prevention are open. One is individual and relates to enticing the smoker to participate in smoking withdrawal clinics or programs, as discussed in part by Dr. Paul White. Programs by the American Cancer Society and by the Federal Government, particularly the Cancer Control Division of the National Cancer Institute, are beginning to have an impact, mainly in males in the upper socioeconomic groups. Privately operated clinics that teach smokers to stop for a fee have varying success rates. It is important to motivate the smoking public to participate and to increase the success rate of permanent withdrawal.

Exposure to tobacco smoke does not seem to lead to irreversible damage, except after long excessive use. This is because this product consists of a mixture of small amounts of primary carcinogens with large amounts of cocarcinogens, the effect of the latter being reversible. There are now data to document that stopping habitual smoking often leads to a reversal of the risk so that ex-smokers, after about 10 years, have the same risk as if they had never smoked.

The second broad means of controlling the risk is through managerial preventive medicine. Basic research efforts have led to the periodic release of data on tar and nicotine contents of cigarettes. The public thus has a choice of selecting a smoke on the basis of such information. Cooperative research efforts of the Federal Government, the American Cancer Society, the American Heart Association, and the relevant industries, have led to progressively lower tar content of existing smoking products and to new materials with even lower content of potentially harmful products. It is important to continue this trend and to foster the development of even less harmful smoking materials. It would be useful to recommend also that some of the higher tar- and nicotine-containing products now on the market be progressively deleted and to encourage the greater use, if at all, of the lower risk materials. This approach has already started to yield results, for the dramatic documented increase in lung cancer rates in men in the United States for the last 40 years is beginning to plateau and, indeed, possibly to decline.

In women who started smoking more recently the increased cancer rate has not approached that of men and is not expected to reach the same high levels.

### Nutrition and cancer

Epidemiology and studies of migrant populations lead to the conclusion that a number of cancers with high incidence and mortality result from dietary habits. In most instances, this means that the macronutrients, and balances therein, are involved, rather than micronutrients, additives, or minor constituents. Again, we need more research on the contribution of micronutrients.

In the United States, cancers of the large bowel, endocrine-responsive organs (e.g., breast and prostate), ovary, pancreas, and kidneys are the result of Western-style dietary habits. These types of cancer are much less frequent in population groups with different eating habits, such as the Japanese. On the other hand, Japanese descendents living in the United States have rates comparable to those of other people living in the U.S. The key change in such migrants and their descendents is diet. Both Japan and the United States are industrialized countries with similar pollution problems, but their main cancer patterns, as regards types of cancer and incidence of each form, are different. This fact reinforces the concept that diet rather than industrial activity per se is responsible for the main human cancers.

We don't yet know by what mechanisms Western-style diets lead to the types of cancer mentioned above or to the development of cardiovascular disease. Thus a study of the key etiologic factors related to such diets and their alteration would have impressive consequences and significance for disease prevention.

The key difference between Western-style and Japanese diet is the amount of fat, particularly of animal origin, and the neutral unsaponifiable fraction, i.e., cholesterol. In the United States, 40 to 45% of calories are derived from fat, whereas in the Orient it is 15 to 20%. A number of White Papers on diet and chronic disease, including that of the American Health Foundation, the American Heart Association, and the Committee on Nutrition of the National Academy of Sciences, have recommended that Norman Jolliffe's "prudent diet" be adopted. This diet lowers the fat component to 30–35% of total calories, and recommends a daily intake of 300 mg cholesterol, or less. These adjustments in diets rely on persuading individuals or groups of individuals to alter their habits through voluntary action.

In addition, as with cigarettes, managerial prevention can be applied for nutrition-connected cancers. In this case, the success rate may be even better and possibly be achieved more readily. The U.S. food industries are an important component of industrial life and they have considerable impact on the public through their merchandizing procedures. It is possible to enlist the industry's research departments to assist in managerial prevention. Recently, in connection with a research program in the prevention of cardiovascular disease, the food and agricultural industries have begun to produce pilot quantities of beef with a lower level of saturated fat. Also, indirectly, the high cost of grain has resulted in having cattle coming to market with leaner meat. This trend should be encouraged for health rather than immediate economic reasons. In the long run, the maintenance of improved health has, aside from its moral and ethical aspect, an important economic impact.

In other areas of the world, such as Japan or Africa, the overall incidence of cancer is also high, but the distribution of cancer is quite different. Gastric cancer, the main type in Japan, has declined in the U.S. over the last 40 years. This decline may stem from more wholesome food practices, especially refrigeration and the lower consumption of meats (especially pork and ham) and fish treated with high levels of nitrate or nitrite-containing preservatives. At the same time, improved supplies of micronutrients, vitamins from fresh fruits, vegetables, and salads may counteract nitrite and provide better tissue integrity. Similar such trends are also evident in Eastern Europe,

and even in Japan. Other types of cancer (liver, esophagus) are likewise due to environmental and dietary practices prevalent in Africa or southern Asia, and would respond to a modification of habits or agricultural practices.

Micro-elements and contaminants in water consumed by man play, as yet, an undetermined role in the development of cancer. Workers in the asbestos industry not only have a higher risk of pulmonary disease, but may also have a higher risk of cancer in the large bowel, presumably because of ingestion of asbestos. These data need confirmation, and in fact more research is required to define the role of small amounts of contaminants such as asbestos, petroleum derivatives, hydrocarbons and halogenated derivatives thereof, phenolic components, and inorganic chemicals in public waters. Nonetheless, it would seem that assuring the purity of water, to which everyone is exposed from birth, would prevent possible synergistic interactions between environmental factors and such water impurities.

It thus appears that individual and managerial alteration of diet, of smoking habits, and products, and to a lesser but more feasible way, of occupational situations, would go a long way toward a necessary decrease in a series of neoplastic and other chronic diseases, which lead to untimely death.

### Personal hygiene and cancer

Cancer of the cervix and, to some extent, cancer of the penis, is seen in population groups with poor sexual hygiene. Recent implication of herpes-type viruses with these types of cancer may open the road to prevention. An immediately feasible preventive action is through the teaching of individual sexual hygiene and the managerial development of housing that permits proper hygiene practice. Also, early detection of neoplastic lesions by regular physical examinations, including "pap" smears, especially in high-risk groups, contributes to secondary prevention by reducing mortality caused by advanced disease.

### Mutagenesis

The relationship of carcinogenesis and mutagenesis is being used to develop a test for chemical carcinogens through mutagen screening in microbiologic systems, a much faster process than carcinogen bioassay in animals. Procarcinogens properly activated to ultimate carcinogens are usually mutagenic. However, not all mutagens in microbiologic systems are carcinogenic.

It is difficult to assess the potential mutagenic hazard from exposure to chemical and environmental carcinogens. There is a given incidence of births with genetic abnormalities. Some of these relate to a recessive trait that is expressed by combination of alleles. This problem is being studied extensively. Early detection in high-risk groups through genetic counseling and also aminocentesis provides the means of judicious decision-making.

Exposure of father or mother to mutagenic environmental agents may result in offspring with genetic abnormalities. This area requires further research. In fact, more data are needed just to define the limits of the problem.

### Teratogenesis

Teratogenic events are due to abnormal transcriptional features during differentiation of the fertilized ovum and as such are quite distinct from mutagenic or carcinogenic events.

Physically or mentally abnormal newborns constitute a portion of live births. As in the case of genetic abnormalities, a fair proportion of abnormal offspring are due to unknown causes. There is evidence that exposure of the fetus to radiation and to specific chemicals (e.g., thalidomide) results in malformed individuals.

It is true that some carcinogens can also act as teratogens. In the majority of instances, however, this is not so. Thus bioassay systems to detect carcinogens cannot be used as a rule to develop information on the possible teratogenic effect of a chemical or a mixture. The effect of teratogens is revealed usually in multi-generation studies, utilizing several species since not all chemical teratogens act alike in diverse species. Clearly, we need more research in this area.

### Recommendations

#### I. Life-style related cancers

**A. Tobacco.** Both human and animal evidence support the view that several types of cancer, especially lung, mouth, and larynx cancer, and non-neoplastic diseases such as emphysema and cardiovascular diseases are causally related to smoking

- Exert major efforts to develop smoking withdrawal clinics. Advise the public of the consequences of smoking, and the hazards of starting to smoke at an early age.
- Accelerate research on the development and appreciation of less harmful smoking products.
- Develop more information on the interaction between smoking and other environmental or occupational situations leading to synergistic augmentation of the hazard.
- Develop more information on conditions other than cancer that stem from smoking, such as emphysema and cardiovascular disease.

**B. Nutrition.** In various parts of the world, the main human cancers are associated with one of several dietary conditions. Efforts need to be made to perform more research to validate current concepts and to secure changes in the mode of living and dietary environment for the definitive prevention of several types of cancer.

- Breast, ovary, endometrium, and prostate cancer appear associated with high diet intake of fat. We must have more research to secure knowledge about the underlying mechanism. Inform the public of the need to lower the fat intake and adopt the "prudent diet" in which fat is at the most 35% of calories, and cholesterol is less than 300 mg per day. Inform the agricultural and food industries of the need to distribute and market products with lower fat and cholesterol content.
- *Colorectal cancer.* This disease is also associated with high fat intake and adoption of the "prudent diet" by the public is indicated. More research is needed to gather an understanding of the mechanisms involved in its development. We should encourage managerial prevention through work with the food and agricultural industries in lowering the fat content of foods, especially of meats and dairy products.
- *Gastric cancer.* Gastric cancer appears associated with the consumption of foods containing a high carbohydrate/fat ratio, low micronutrients, and sizable amounts of nitrite or nitrate. Detailed investigations are needed to document the underlying mechanisms. We must advise the public of the need to store food at low temperatures to

prevent the formation of nitrite from nitrate through microbiological reduction. Also, improved diets with respect to micronutrients, especially vitamin C, a nitrite antagonist, should be recommended. Managerial prevention includes control of the addition of nitrite and nitrate to foods. This area is currently under discussion by USDA and industry Advisory Groups.

### II. Occupational cancer

Exposure in an occupational setting to certain chemicals or mixtures has resulted in cancer in man, sometimes after a long, latent period. With few exceptions, the products responsible have also caused cancer in animal models. We must

- minimize exposure of workers, maintenance people, and allied personnel to known chemical carcinogens;
- avoid the release of chemical carcinogens into the environment through air, water, or solid effluents, including sewage, where the public at large might be affected;
- take advantage of the data generated by the National Cancer Institute, the National Institute of Environmental Health Sciences and other programs, on improved and more rapid methods of bioassay, including mutagen testing, to acquire information on, as yet, untested chemicals to which individuals may be exposed. Perform bioassays of chemicals revealed by such prescreens, and for which exposure parameters of specific groups or the population at large denotes the existence of potential risk;
- in view of a number of lines of evidence mentioned in this document on the lesser sensitivity of older individuals to chemical carcinogens, and considering the latent period required for overt cancer appearance, select older individuals, typically above age 45, for employment in situations where contact with carcinogens might occur. Of course, as noted above, systems should be designed to ensure minimal exposure to actual or potential carcinogens.

### III. Prevention of cancer

(1) Develop additional research efforts on the causes of all types of cancer and the mechanisms of carcinogenesis in order to provide a sound and reliable data base for the elaboration of rational preventive measures.

(2) Establish health-action groups to give wide publicity to currently available methods of cancer prevention.

(3) Through informational, managerial, and legislative efforts, lower the cancer risk of the work environment, and the risk from personal habits, such as smoking and poor dietary practices.

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OPERATION HALLEY'S COMET

From the Colonel

**"Tomorrow evening at approximately 2000 hours Halley's Comet will be visible in this area, an event which occurs only once every 75 years. Have the men fall out in the battalion area in fatigues, and I will explain this rare phenomenon to them. In case of rain, we will not be able to see anything, so assemble the men in the theater and I will show them films of it."**

Executive Office to Company Commanders

**"By order of the Colonel, tomorrow at 2000 hours, Halley's Comet will appear above the battalion area. If it rains, fall the men out in fatigues, then march to the theater where the rare phenomenon will take place; something which occurs only once every 75 years."**

Company Commanders to Lieutenants

**"By order of the Colonel in fatigues at 2000 hours tomorrow evening, the phenomenal Halley's Comet will appear in the theater. In case of rain, the Colonel will give another order, which occurs once every 75 years."**

Lieutenants to Sergeants

**"Tomorrow at 2000 hours, the Colonel will appear in the theater with Halley's Comet. If it rains, the Colonel will order the Comet into the battalion area in fatigues."**

Sergeants to Squads

**"When it rains tomorrow at 2000 hours, the phenomenal 75-year old General Halley, accompanied by the Colonel, will drive his Comet through the battalion area theater in fatigues."**

From The ACS Southeaster

# Indecent Exposure — Are You A Victim?

An ICWU Member in the plastics industry writes:

***Dear Doctor Cullen:***

***I have had breathing problems the last six months or so. I work with polyurethanes. Some of the drums are marked "irritant" and some are marked "sensitizer."***

***I want to see a lung specialist recently who said I may have job related asthma, but he wouldn't know for sure until he did some tests like a FEV1 and a RAST test. Could you tell me more about these tests?***

***The doctor mentioned the terms "sensitizer," "irritant," "allergic" and "antibodies." What is the difference between all these? Is there a cure for job related asthma?***

Dear ICWU Member:

As you already know from your own experience, exposure to polyurethanes from either their production or use can cause or worsen breathing problems. Polyurethanes products — foams, lacquers, coatings of all sorts — are used widely in the American economy. Polyurethane is a polymer, in other words, a chain of single urethane units. Polymerization of urethanes involves the use of isocyanates. Isocyanates are a group of chemical compounds that are some of the most "sensitizing" substances known to exist in the industrial environment.

What do we mean by sensitizing? Sensitization to a substance means that the body has developed the tendency to react in an excessive way when exposed to the offending substance. Exposure usually occurs either through inhalation or skin contact. Airways hypersensitivity or asthma occurs after exposure through breathing; contact dermatitis and/or hives occurs after exposure through skin contact.

To get back to isocyanates, approximately 5 percent of workers exposed to them on the job can be expected to develop sensitivity to them, usually in the form of asthma. Exposure to isocyanate occurs before the polyurethane foam or lacquer is fully polymerized. Once the polyurethane is completely polymerized or "set," there should be little or no free isocyanate present. Once sensitivity to an isocyanate has developed, the sensitized worker is often not able to work where there are even minimal concentrations present. Reaction can occur at exposure levels below the OSHA eight hour time-weighted average threshhold limit value (TLV). The current feeling of a number of occupational health experts is that isocyanate sensitivity may be a result, in many instances, of nonregular heavy exposure during accidental leaks and/or spills at worksites. In these cases the average exposure may be under the 8-hour time-weighted standard.

How does the doctor make the diagnosis of isocyanate sensitivity equal isocyanate induced asthma? Most important is a work history of exposure to isocyanates (don't expect your family doctor to know anything about them) and a medical history of recurrent shortness of breath, cough and possibly wheezing that occurs after exposure. In the case of isocyanate asthma in particular, or occupational asthma in general, shortness of breath, chest tightness and cough are more frequently found than wheezing. Wheezing is usually found in classic allergic asthma. Symptoms of occupational asthma won't occur immediately after exposure to the offending agent; they may not occur until 48 hours after exposure. In other words, after the worker is already at home. That is why you have to help the doctor make the diagnosis. He or she is unlikely to think of it without your talking up the relationship to work. An extremely important question is, do the symptoms go away on weekends and vacations?

### X-RAYS

Chest x-rays are almost always normal with asthma of any kind so they won't be of much use in diagnosing occupational asthma. Breathing tests are of much more use, but only during and perhaps immediately after an attack of asthma.

### LUNG FUNCTION TESTS

When an attack of asthma occurs the bronchi or breathing tubes become narrow so that it is hard to exhale after taking in a breath; there is an obstruction in exhalation. This obstruction usually reverses itself in time but the process can be speeded up with the use of certain medication. In other words, asthma is a reversible obstructive lung disease. If breathing tests are done in between asthma attacks, they will usually be normal.

### FEV1

The most important breathing test for occupational asthma is the $FEV_1$ (forced expiratory volume in one second) which is the measure of how much air a worker can blow out in one second with the maximum effort. $FEV_1$ can be measured both before and after a workshift to see if there is any decrease in breathing capacity. For the worker that has developed isocyanate sensitivity, there is usually such a decrease in the $FEV_1$ over the workshift.

### RAST TEST

Are there any blood or skin tests which can 100 percent confirm the diagnosis of isocyanate sensitivity? The answer at the present time is no. Obviously, it would be useful to have a simple blood test in order to screen for sensitized workers. However, the various blood tests that have been developed to measure "antibodies" or proteins made by the body to stick onto foreign substances such as isocyanate molecules, have thus far proved unreliable. The RAST test is one such test that unfortunately is not always positive when a worker has isocyanate asthma and is not always negative when an exposed worker is well. Skin tests have proved equally unreliable.

### IRRITANTS

You also asked about "irritant" substances in your shop. Many chemicals including some dusts, vapors and fumes are directly irritating to the lining of the respiratory tract. It is quite common for occupational doctors to see workers with chronic bronchitis or inflammation of breathing tubes due to long term exposure to irritating substances such as ammonia, sulfur dioxide, nitrogen dioxide, chlorine, ozone, manganese, chromium, silica, acid mists and fluorides.

It turns out that in high concentrations, isocyanates are quite irritating and will cause respiratory symptoms in most workers, not just those who are isocyanate sensitized. One clue that a substance is directly irritating rather than sensitizing, is the finding that most people on the job seem to complain of burning eyes, nose and throat when exposed to the airborne chemicals.

Workers with chronic bronchitis will usually improve with reduction of exposure. Exposure can be reduced through improved ventilation, enclosure of the work process, or adequate respiratory protection. Workers with occupational asthma, on the other hand, often have to change jobs to get completely away from the substance to which their airways are sensitized. Even if they are taking anti-asthmatic drugs, the sensitized worker may have to change jobs to avoid further illness.

Dr. Mark Cullen

ICWU HEALTH & SAFETY NEWS — APRIL 1982

# Cancer . . . Is A Six-Letter Word!

Cancer is a word that is frightening to most people. And a look at some statistics for this disease provides ample reason for such a reaction.

One out of every four Americans can expect to get cancer, and two out of every three families will have a member who develops some form of cancer. Although early diagnosis and improved treatment techniques increase the chances of survival, the rate of cancer deaths (and reported cases) is rising. In 1973, 351,000 Americans died of cancer, but by 1978 the numbers had increased to 364,000.

One reason more cases are seen has to do with our increased life expectancy. There is a long delay between exposure to a cancer causing substance and appearance of the disease. Since life expectancy is greater today, more people live long enough for cancers to appear. But the increased rate of cancer cannot be explained solely by longer life span. We must look beyond life expectancy and examine the evidence which points to potential occupational and environmental causes.

This article will explore what cancer is, potential and known causes, testing of cancer suspects, regulation of carcinogens, resources, and prevention of cancer. Because evidence strongly points to occupational and environmental exposures as major causes of cancer, the article focuses on this aspect.

### CANCER — WHAT IS IT?

The term "cancer" describes a number of different diseases with a variety of causes. But basically, in all forms of cancer the body's cell division system becomes damaged, which leads to a rapid, out-of-control growth of abnormal cells. Generally cancer starts off as a disease localized in one area (often on the surface of an organ such as the skin or liver). As growth continues, the cancer can spread deeper into the organ and eventually spread to other parts of the body (metastasize) by way of the blood or lymph systems.

### WHAT CAUSES CANCER?

There are many substances in our environment that are linked to development of cancer. In fact, environmental pollutants in air, water, soil, and the workplace are now estimated to cause between 75 and 85 percent of all human cancers. The increase in manufacture and use of petrochemicals is directly connected to the proliferation of cancer-causing substances in the environment.


Although we often hear that "everything causes cancer," this is not the case. From human studies, about 30 substances or agents have been identified as carcinogens, and about 50 others show evidence of carcinogenicity (ability to cause cancer) based on epidemiological evidence. Known cancer-causers (carcinogens) include chemicals such as benzidine and other organic amines, asbestos, nickel and chromium, benzene, and arsenic, as well as ionizing radiation and ultraviolet radiation.

Some cancers seem to have multiple causes, and exposure to more than one carcinogen may increase risk of cancer development. For example, both cigarette smoking and asbestos are linked to increased lung cancer risk. Asbestos workers who don't smoke have an eight times greater risk of developing lung cancer than an unexposed population, while asbestos workers who do smoke have a 92-times greater chance when compared to an unexposed population. On the whole, however, the potential for producing cancer when two chemicals interact is largely unstudied.

Although all occupational and environmental carcinogens have not been identified, there are numerous substances which are considered human carcinogens based on results of epidemiological studies as well as animal studies.

### TRACKING DOWN CARCINOGENS

Unfortunately, the majority of known carcinogens have not been discovered by any systematic method of testing chemicals before introducing them into the workplace or general environment. Instead, workers have been exposed to substances for many years, and after increased cancer rates are seen, confirming tests in animals may be conducted. Because most chemical carcinogens have a long latency period (time between initial exposure and the appearance of cancer), by the time a chemical is confirmed as a carcinogen large numbers of workers may have already been exposed.

Epidemiological studies are one of the major methods for determining that a

(continued on next page)

chemical is a carcinogen. In such a study, scientists follow a group of people who have been exposed to a certain substance. They compare the number of cancer cases and deaths with a similar group (control group) who were not exposed to the substance. Epidemiological studies are the best evidence that a substance is a human carcinogen. Their obvious drawback is that people continue to be exposed while waiting for the results. So we have a situation where, as Bill Lloyd of OSHA has said, "Almost everything we know now about occupational cancer comes from counting dead bodies."

Obviously other methods must be used to determine if a chemical is capable of causing cancer. The most widely used tests are long-term animal studies, although short-term tests in bacteria, insects, or plants are being used more frequently.

### ANIMAL TESTS

The basic aim of an animal test is to measure the effects of exposing a particular group of animals to a specific substance. Two groups of animals are used: experimental (exposed to the substance) and control (unexposed). These groups are further divided into subgroups, e.g., 50 animals of one sex and species. Generally, testing is performed in two strains of animals. Because of their short lifespan (about four years) rats, mice, and hamsters are the common test animals.

The animals are normally exposed in the way that most closely resembles human exposure (for example, inhalation of asbestos fibers). Generally, at least two doses should be used: the maximum tolerated dose, which is the largest dose that will not shorten the animal's lifespan by causing some other toxic effect; and either one-half or one-quarter of that dose. At the end of the test period the animals are killed and their organs examined for evidence of cancer. If the number of cancers in the exposed group is significantly greater than in the control group, the substance is considered a carcinogen.

Animal tests are considered appropriate in determining the cancer-causing potential of a substance because evidence of carcinogenicity in animal tests agrees with known human experience. One scientist who reviewed animal test data on 82 substances for which there was good epidemiological evidence of human carcinogenicity found that only one of the chemicals, arsenic, did not cause cancer in test animals.

Although most scientists agree that chemicals which cause cancer in humans will also cause cancer in animals, the reverse is not true. One major reason that people resist using animal test data to predict effects in humans relates to dose. The doses given to test animals are high, and this leads to the misconception that anything will cause cancer if the dose is high enough. Yet, numerous chemicals have been tested in animals at the normal high doses and have not been found to be carcinogenic.

High doses are used so that the possible effects will be easier to detect, since the group of test animals is relatively small when compared with the number of potentially exposed humans. For example, what if a low dose of a substance caused cancer in one of every 10,000 persons (or 10,000 tumors in 100 million Americans)? If you used the same corresponding dose in a group of 50 rats (as opposed to 10,000 rats) clearly the chance of cancer showing up would be minimal. Reliable conclusions can't be drawn from such a small test sample. In addition, the costs of using a large number of test animals would be prohibitive. When doses are high, the cancers are more likely to be seen in a small group of test animals because the cancer rate would be increased proportionately. And negative results at high doses increase the likelihood that the substance is not a carcinogen.

### OTHER TESTS

Because animal tests are expensive (up to $400,000 per test) and involve at least 3.5 to 4 years per test, it would be an impossible task not only to test all the new chemicals introduced into the workplace each year, but also to test those already in use. So there is a great deal of interest in short-term tests such as the one developed by Bruce Ames at the University of California, Berkeley. The test identifies substances which are mutagens (capable of changing genetic material). Ames and other scientists believe that most mutagens are also carcinogens. In fact, nearly 90 percent of those chemicals which are known carcinogens test positive as mutagens in the Ames test. Numerous other short-term tests are in use or being studied.

Despite problems with short-term and animal tests, some such testing program must continue. Past experience with asbestos, BCME, vinyl chloride, and numerous other carcinogens has clearly shown that we cannot wait for absolute proof of carcinogenicity in humans before regulating exposure to potential carcinogens.

### PREVENTION

Because the vast majority of cancers are caused by agents in the environment, it follows that they are potentially preventable — remove the substance, remove the risk. Experience has shown, however, that it is not that simple. In fact, this country has failed abysmally in controlling exposure to carcinogens. When other countries have banned substances (for example, Italy and England banning benzidine and other organic amines) the U.S. has continued its quest for "safe" exposure levels. So despite knowledge of carcinogenicity, workers continue to be exposed to benzene, coke oven emissions, asbestos, and other carcinogens. And in many cases they continue working without being told that substances to which they are exposed cause, or are suspected of causing cancer.

The reasons for this country's failure to regulate carcinogens and adequately protect workers and the general public include:

- Pressure from industry related to economic costs of control. This is evident in industry's support of cost-benefit analysis for new health standards. But, as Samuel Epstein points out in his book, *The Politics of Cancer*, the benefits of using a carcinogen go to one group of people (corporations and stockholders) while the real costs in terms of illness, death, lost wages, medical expenses, and so on, paid by workers and society are dismissed or ignored.
- Lack of a coordinated approach. The many government agencies which have authority in terms of research, control, prevention of cancer, or enforcement of carcinogen regulations have different approaches to these areas. Some efforts have been made to arrive at a uniform approach, but thus far such an approach has not been developed.
- The long latency period. Because of cancer's long latent period it's often difficult to identify cause with effect. And regulations are directed toward short-term, acute health effects.

### PROGRAM NEEDED

An effective program to prevent cancer is necessary. Such a program must also be aimed at those workers who have already been exposed. What are some of the aspects of such a program?

- Workers currently exposed to known carcinogens must be informed of their exposures and the hazards involved. In addition, controls must be instituted in those situations where carcinogens are used (for example, protective clothing, air-supplied respirators, closed systems handling carcinogens). Education and training programs should be developed for such workplaces. Labeling requirements should also be developed and enforced.
- All carcinogen use should be reported to government agencies for enforcement and compliance purposes. As part of the reporting system, workers should receive a copy of the carcinogen use report form.
- Efforts must be made to find and contact workers formerly exposed to carcinogens, so that they can be channeled into screening programs aimed at early detection of cancer, and receive medical attention if necessary.
- Research on reliable short-term tests to identify carcinogens should be performed.
- New chemicals introduced into industry and the environment must be tested for carcinogenicity. Chemicals already in use should also be screened. A method for prioritizing chemicals to be tested should be developed. (The Toxic Substances Control Act endorsed by the Environmental Protection Agency as well as OSHA's new cancer policy focus on this aspect of carcinogen regulation.) Efforts should also be made to identify effects of interacting substances.
- Tumor registries, where all cancer cases would be reported, should be developed throughout the country. Currently very few such registries exist.

### A SOCIAL DISEASE

The above are specific avenues for dealing with the epidemic of cancer related to occupational and environmental exposures. But implementing such programs is not enough. Cancer is not just a biological phenomenon. It is a disease with social, political, and economic aspects. Carcinogenic chemicals are produced and used despite their effect on humans because industry considers them necessary, and efforts to control such chemicals are met with the argument that in many cases it is not economically feasible to do so. Controls or substitutions would be costly, and increased product price would then be passed on to the consumer. These arguments caused delays in standards for substances such as coke oven emissions and arsenic, and will continue to do so as long as economic impact statements remain a crucial issue in standard setting.

Cancer is also a social disease. It robs the victims, their families, and society. And it heaps ever-increasing insurance and medical costs on society as a whole. Yet we demand certain goods that are manufactured using carcinogens — pesticides, hair dyes, dyes for other materials, plastics, and so on. So we must determine how important such things are in light of their terrible cost. Cancer is potentially preventable, but all sectors of society must be committed to prevention if we are to see an end to the cancer epidemic.

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## Common Chemicals and Cancer Risks

| Agent | Cancer Site |
|---|---|
| Arsenic (R) | Skin, lungs, liver |
| Asbestos (R) | Lungs; pleural and peritoneal mesotheliomas; rectum |
| Benzene | Bone marrow (leukemia) |
| Chromium | Nasal cavity, larynx, lung |
| BCME (bis-chloromethyl ether) | Lung |
| Benzidine (R) | Bladder |
| Alpha-napthylamine (R) | Bladder |
| Beta-napthylamine (R) | Bladder |
| 4-aminodiphenyl (R) | Bladder |
| 4-nitrodiphenyl (R) | Bladder |
| Wood dust | Nasal cavity and sinuses |
| Coke oven byproducts (R), shale, and mineral oils | Nasal cavity, larynx, lung, skin, scrotum |
| Vinyl chloride (R) | Liver, brain |
| Anesthetics | Bone marrow (leukemia) |
| Ultraviolet radiation | Skin |
| Uranium (radon daughters) | Lung |

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## *H&S Reports Available To Local Unions*

**There are numerous reports, books, pamphlets and other material for local union Health and Safety Committees, all of which hold a wealth of pertinent data.**

**From time to time, we will preview some of these and list addresses where ICWU Health and Safety Committees may write directly for them. Unless otherwise noted, these publications are free of cost.**

**"Taking Back Our Health: A Training Manual on Job Safety and Health for Black and Latino Workers"**

**The coming years do not hold great promise for Black and Latino workers. As the economy worsens, the rate of unemployment for these workers will be staggering; many will be displaced from their communities as employment opportunities evaporate.**

**Yet most adults from the Black and Latino communities will remain active workers. And as such, they will be confronted with discrimination and unhealthful working conditions on a growing scale, since the current administration has made it a policy to dilute the already inadequate number of standards and regulations promulgated by the Occupational Safety and Health Administration (OSHA), and to reduce OSHA's enforcement activity to near non-existent levels.**

**Job placement discrimination has inevitably guaranteed the dirtiest and most dangerous jobs for most Black and Latino workers with little prospect of advancement or transfer to less hazardous locations. As a result, Black and Latino workers are probably over-represented in the current occupational disease and injury epidemic that afflicts all American workers (available statistics indicate that each year as many as 100,000 workers die from job-related illness while another 2.2 million suffer disabling injuries; that 20-40 percent of all cancers are job-related).**

**Copies of report are available from:**

**The Urban Environment Conference Inc.**
**666 - 11th Street, NW, Suite 1001**
**Washington, DC 20001**

# Cancer and Lifestyle: An Expert View

*Does air pollution cause cancer? What is meant by the statement that lifestyle is responsible? What's the best way to tackle prevention and cure of this disease?*

*Questions like these are being asked by scientists, doctors, policy makers and private citizens. They must be answered to properly direct research and the regulatory process.*

*Dr. John Higginson is a senior scientist for a consortium of universities called Universities Associated for Research and Education in Pathology. He was founding director for the International Agency for Research in Cancer, an agency of the World Health Organization, and has been working in the cancer field for more than 30 years.*

*In this interview he comments on some issues that are part of the cancer controversy.*

Dr. John Higginson

**CE:** ***You have stated most causes of cancer are environmental. What is covered by that statement? What percentage of cancer does that include?***

**Dr. Higginson:** If one uses the term environment in a wide sense, the cultural environment, the work environment, the chemical environment, behavioral environment, diet and so on, it covers a wide amount of the cancer problem.

There's a lot of indirect evidence to indicate that these various factors influence about 80 to 90 percent of human cancers.

**CE:** ***When you talk about environmental causes, you name diet as one. Can you define a few of the others?***

**Dr. Higginson:** We have cigarette smoking, alcoholic beverages, sunbathing. In terms of the work environment, we have exposures in the workplace to chemicals, we have the environment that's related to medicine or radiation. Certain drugs which are used in fact to treat cancer are also highly carcinogenic in themselves.

**CE:** ***What does this imply then for cancer prevention or for cancer protection?***

**Dr. Higginson:** There are three major implications. One, where the carcinogenic agent is easily definable, it is controllable. The possibilities for prevention are almost 100 percent. In terms of cigarette smoking, cigarette-related cancer are almost theoretically completely preventable.

Secondly, in the work environment, removal of the highly carcinogenic substances or substitution by a less harmful substance may have a highly effective action.

Third, I don't think it has a major implication for detection, except in situations where you know people are at exceptionally high risk. Then you may wish to build into the system methods of trying to identify and diagnose those cancers earlier so that you are going to be able to treat hopefully at a curable level.

If your prevention is good, early detection in that particular cancer ceases to be very important.

**CE:** ***In your opinion, what direction should cancer research be taking? Where should research money and scientific effort be focused?***

**Dr. Higginson:** That is a question that everybody answers, and there is no universal agreement. Speaking personally, I believe it has to be tackled at four levels. One, the application of known knowledge such as the role of cigarettes, the role of occupational hazards, things like that.

Secondly, when we're dealing with those agents that are less clearly defined, we have to do the basic research to see if by understanding mechanisms we could possibly identify what for example is the role of late full-term pregnancy in causing cancer of the breast in females.

Thirdly, because we don't know the cause, obviously we have to do a lot more research and better treatment, because if you can't prevent a cancer, the only major hope is to be able to cure it.

Fourthly, these all add up to a common packet where each particular problem is investigated, and the most satisfactory approach developed. It's a mixture of applied and basic research. These are not contradictory, they're complementary.

**CE:** ***Has industrialization affected overall cancer rates?***

**Dr. Higginson:** The evidence at present that we have is that industrialization in the sense that we understand it of mass exposure to chemicals in the general environment has had very little impact.

Of course, where industrialization has led to exposure to compounds at a very high level, such as certain dyes that cause bladder cancer or asbestos that causes cancer of the lung and of the covering of the lung, it has had an impact.

But I think the question you're posing is the total community. There it doesn't seem to have had much of an impact.

***". . . industrialization in the sense that we understand it of mass exposure to chemicals in the general environment has had very little impact."***

**CE:** ***Why do some environmental groups keep trying to link cancer with industrial pollutants?***

**Dr. Higginson:** I think one must go back historically to the period when industrial cancer was ignored and many companies showed a high degree of insensitivity to the problems of the workplace. It wasn't only cancer but general working conditions, accidents in the workplace, and so on.

Now, this then was followed by a period in which many people believed cancer was due to viruses. But if it was due to a virus, there wasn't very much you could do about it. Then when one started moving back again to the chemical field, nearly all the chemical agents that have been defined as causing cancer in humans were first identified in the workplace. There was a kind of association—that environment meant chemical environment, and that the chemicals would be automatically man-made.

The reason it was discovered in the workplace was that these were high-risk populations of relatively modest numbers of individuals who showed very high frequencies. Because in those days the exposures in the workplace were often extraordinarily high and therefore you were really getting a very, very high frequency which is almost impossible to miss.

**CE:** ***The environmental groups are concerned as much about air pollution outside the plant as they are with workplace exposure.***

***There is still an attempt to link overall cancer rates to environmental pollution. Would you comment on that?***

**Dr. Higginson:** It started back in the early '50s, at a time when I entered the cancer research field.

At that time, many people said it wasn't cigarettes that were the major cause of lung cancer; it was air pollution due to industrialization. And this view was expressed by a considerable number of distinguished scientists.

Now Doll and Peto and myself and Winder all came up with these low figures for occupation. We were not able to identify significantly a large group of cancers due to general pollution. The most we could say was that possibly general pollution had an effect on lung cancer in cigarette smokers.

On the other hand, I can understand that not only environmentalists but many other people really don't believe that breathing dirty air full of soot is good for you. And certainly it had significant effect in terms of bronchitis and emphysema and other respiratory diseases in the United Kingdom.

There's a gut feeling that it must be carcinogenic. And certainly, dust contains cancer-causing agents. So people are very unwilling to believe something they can do something about in terms of air cleanness may have much less impact on the problem than they would wish.

**CE:** ***What role does workplace exposure to chemicals play in overall cancer incidence?***

**Dr. Higginson:** I feel that up to five percent is probably in reason. One tends to push the figure up rather than down because one doesn't want to miss something that you can do something about.

**CE:** ***What's the best way to lower even further the numbers of work-related cancers?***

**Dr. Higginson:** I think we would make considerable progress if we can get rid of cigarette smoking, because whatever you look at, whether it is lung, larynx, mouth, pancreas, bladder, many of which are work-related cancers, it's awfully difficult to unravel what proportion might be attributable to the workplace because of the overwhelming impact of the cigarette habit. This makes it almost impossible to identify other possible agents.

Certainly, for example, Dr. Selikoff has shown that in terms of asbestos workers and cigarette smoking, cigarettes so potentiate the effect of asbestos as to be enormous. And asbestos is a much less important problem from the public health point of view if you don't smoke cigarettes, because they do potentiate each other.

**CE:** ***Dr. Higginson, is there now or can we expect an epidemic of cancer in the United States that would be caused by industrial chemicals in the air?***

**Dr. Higginson:** In the 30-odd years I have been working in the cancer field, I have seen a number of situations called epidemics of cancer. The vast majority of them have faded out, and have obviously been a chance observation.

There has been a major epidemic in cigarette-produced lung cancer in the United States and elsewhere. A much smaller epidemic, but still very important for those concerned, has been asbestos.

The third possible epidemic, if one wants to use that term, is melanoma of the skin due to sunlight, due to the marked change of (sunbathing) habits among the community. That is the situation up to today.

***"We have not been able to define any group or individual group of cancers due to pollution of the general air."***

We have not been able to define any group or individual group of cancers due to pollution of the general air. There seems to be some effect of air pollution on smokers. Also, air pollution does have an effect on cardio-respiratory disease, chronic bronchitis and diseases of that type.

But it has had a surprisingly low or no effect on lung cancer. This has been looked at in great depth, especially in the '50s. When people started blaming cigarettes, many people stated that it wasn't the cigarettes, it was air pollution factor, due to industrialization.

Now, no one can ever deny that something couldn't happen in the future. But I think with what we know today it's most unlikely that any chemical could escape to the general environment in such quantities, except perhaps atomic war where radiation would occur, that would in fact lead to a major epidemic in the general population.

In fact, air in many countries such as the United Kingdom, is better today than it was in 1950. This is well documented. In the U.S. it's been somewhat slower. But I don't think we will see another epidemic due to air pollution, provided we maintain reasonable controls. I'm talking about a reasonable, balanced public health approach.

**CE:** ***You said we probably should not expect another epidemic from air pollution. Was there one before this?***

**Dr. Higginson:** There was a personal epidemic due to personal pollution of your own environment by the cigarette. Now it is possible that this is going to cause problems and that passive smoking may turn out to be much more dangerous than people believe.

There are two studies which suggest that people exposed to other people smoking, one in Greece, one in Japan, does lead to an increase in lung cancer. If that is the case, we might be able to say that some of the increase in lung cancer or some of the cases of lung cancer in ordinary individuals are produced due to passive smoking.

*To be Continued Next Month*