

TGG: JCL: MMG: RF

XF: _____

August 2, 1988

VISTA

Mr. Steve Freese
B. F. Energy
359 San Miguel, Suite 204
Newport Beach, CA 92660

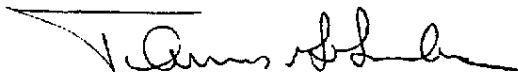
Dear Steve:

Enclosed is a copy of the skin irritation study on Vista 47 Ink Solvent. The testing produced a maximum irritation index of 3.7 out of 8, which classes it as a moderate irritant. This study is done by causing constant contact on the skin of rabbits for four hours.

From a regulatory standpoint an irritation score of 5.0 or greater as defined in the Hazard Communication Standard is considered a skin irritant by OSHA. However, proper precautions should be taken by users of this product to avoid prolonged or repeated skin contact.

Please contact me if you have any questions regarding the details of this study.

Sincerely,



Thomas G. Grumbles, C.I.H.
Environmental Quality Manager

dlj

Enclosures

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Thomas G. Grumbles

VISTA

To Environmental
Coordinators

Date 8/1

Attached is a good booklet on
cancer rates, etc. It may
have some helpful information
for your communication
programs.

TGG

CJRD

VVV 000008718

Is There An Epidemic?

**A Report
by the American Council
on Science and Health**

VVV 000008719

The American Council on Science and Health (ACSH) is a national consumer education association directed and advised by a panel of scientists from a variety of disciplines. **ACSH** is committed to providing consumers with scientifically balanced evaluations of issues relating to food, chemicals, the environment, and health. **ACSH** is a nonprofit association exempt from federal income tax under Section 501 (c) (3) of the Internal Revenue Code. All contributions are tax-deductible as provided by law.

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June 1988

American Council on Science and Health
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CANCER IN THE UNITED STATES: Is There an Epidemic?

This report was written by **Alan C. Fisher, Dr.P.H.**, and **Wendy Worth, Ph.D.**

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American Council on Science and Health

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Part I: Cancer Statistics and Trends

Introduction

Cancer is a disease that should concern all Americans. Actually, cancer comprises a group of more than 100 different diseases. Although their form and symptoms vary, all cancers begin with an alteration in a normal cell that in turn replicates itself millions of times resulting in the unregulated growth and spread of abnormal cells. Our concern over the disease is justifiable. Approximately 22 percent of all deaths in this country are now due to cancer. It is estimated that in 1987 there were 483,000 cancer deaths and 965,000 new cases of cancer in the United States.

Many Americans believe that we are currently experiencing a cancer epidemic. With the notable exception of lung cancer, this is not true. An "epidemic" is a significant increase in the frequency of a disease. In the case of cancer, what is excessive can be determined by a comparison of cancer rates over time, among different countries, and even among different groups within the same country. As will be seen, such comparisons do not reveal an overall cancer epidemic in the U.S. today.

Part I of this report focuses on these comparisons. Part II focuses on the known causes of human cancer and what one can do to reduce his/her risk of developing any of these many diseases. Before looking at these comparisons, however, it is necessary to have a working understanding of how cancer statistics are typically calculated and the limitations of the sources from which they are obtained.

Ways of Looking at Cancer Data: Terminology

Epidemiology is the scientific study of the distribution and determinants of disease frequency within and between human populations, and the application of this study to the control of health problems¹. Cancer epidemiology is a subspecialty of this discipline, which uses standard statistical techniques to compare cancer data from place to place, time to time, or group to group. This report will focus only on those techniques that are most useful for addressing the issue of a cancer epidemic in the United States.

An important concept in cancer epidemiology is *incidence*—the number of new cases diagnosed in a given geographic place during a specific period of time. To compensate for variations in number of inhabitants in different areas, incidence is divided by the total population

The United States

to yield the *incidence rate*. The measure thus formed is not influenced by population size and can be used for comparison.

Another important concept is cancer *mortality or death*. This is how many people in a given geographic place, during a specific period of time, die of cancer. Usually one wants to compare cancer deaths, and therefore calculates *mortality or death rates* by dividing the number of deaths by the total population of the geographic location.

Theoretically, epidemiologists prefer incidence rates to death rates. Incidence rates are more closely associated with the occurrence of a disease and its causes. By contrast, mortality data are the end result of the disease and are therefore further removed from its causes. Such data are also influenced by changes and advances in treatment. However, death rates for cancer are more readily available than incidence rates, because the cause of death appears on every death certificate. Thus, epidemiologists frequently use cancer death rates to indicate incidence rates. Death rates most closely resemble incidence rates when the average survival time for a form of cancer is short. Lung cancer is one instance where incidence and death rates are similar.

Despite the theoretically valid argument just outlined, some evidence points to unreliability of incidence rates for studying recent trends in cancer. With respect to some cancers, recent trends show either sharp increases or substantial fluctuations from year to year. The following section deals with the "biases" (or systematic departures from true values) believed responsible for these statistical artifacts (exception: lung cancer).

Occasionally, incidence rates and death rates are standardized or "adjusted" for an additional factor, namely age. The adjusted rate is composed of a weighted average of rates for specific age groups. The amount of weight given to each age group is determined by reference to a standard population. Since the risk of cancer increases with age and people in the United States are living longer today, failure to either adjust cancer rates by age or look at different age groups separately (age-specific rates) can convey the impression of an overall increase in cancer when in fact there is none. Therefore, when one compares cancer rates over time, between places, or among racial and ethnic groups, where the age distribution varies, one generally adjusts the rates statistically to a standardized age distribution so as to make them comparable. Thus, changes in cancer frequency over and above those influenced by a changing age distribution become detectable.

Incidence rates and death rates are often reported per 100,000 persons. Also, it is common to report rates for a one-year period corresponding to a given calendar year.

Sources of Cancer Data

In the United States there is one primary source of data for incidence rates and one for death rates.

The National Cancer Institute (NCI) has conducted surveys of cancer incidence in the past (1937, 1947, 1969-71). It was not until 1973, however, that it began to constantly monitor cancer incidence through the Surveillance, Epidemiology, and End Results (SEER) Program. This program involves collection of data from cancer registries in different geographic areas. The areas presently covered are the entire states of Connecticut, Hawaii, Iowa, New Mexico, Utah, and New Jersey, as well as the Commonwealth of Puerto Rico. Also included are metropolitan Atlanta, Detroit, and the San Francisco-Oakland and Seattle-Puget Sound areas. The areas covered by SEER have changed slightly over the years.

Mortality data on cancer come from the National Center for Health Statistics (NCHS) and are obtained from death certificates throughout the U.S. Such data have been available since 1933.

Every year the American Cancer Society (ACS) uses data from the NCI and NCHS to compile estimates of both cancer incidence and mortality for the upcoming year. The annual ACS publication presenting this information is called *Cancer Facts and Figures*.¹

A. Accuracy.

All three sources of cancer data have limitations, some of which are inherent in the data and can cause bias during examination of statistics for a specified period. In other circumstances, biases cause concern when we compare data from different time periods. Different kinds or degrees of bias may characterize each period.

The major limitation of the recent NCI data (SEER) is that the geographic areas covered in the Survey do not represent the United States as a whole, but were chosen because they represent epidemiologically diverse populations. Information included for incidence rates by site, age, race, and ethnicity allows epidemiologists to monitor changes in incidence over time and make comparisons among groups. In fact, mortality rates by cancer site, for the SEER areas and the U.S. as a whole, correspond closely, especially for whites. Thus, cancer incidence rates by site obtained from SEER may be representative for the white population.

In comparing incidence rates over time for NCI data, a number of important biases are relevant. First, there has been a change in the geographic areas covered. Although some overlap occurs among the first three NCI

studies and the areas covered by SEER, there is also a significant difference in the areas covered by the earlier studies compared to those covered by SEER. A second bias stems from differences in the accuracy of area population estimates at different time periods. Another important problem likely to have varied over time lies in the care taken to include each cancer patient only once in the study and to include only new cases of cancer for the period under study. Accompanying this is the bias introduced by changes in the effort and motivation of physicians in registering cases of cancer incidence. Furthermore, over the years there have been technical changes in the definition of exactly what constitutes a cancer as well as changes in the methods and ability to diagnose many forms of the disease. Many experts believe that current cancer incidence rates are being inflated by a significant number of cases of "overdiagnosis"—growths that are biologically malignant but do not spread quickly and threaten the individual's health. In a recent report,¹³ the National Cancer Advisory Board identifies this problem as inflating some cancer rates.

Data on death rates from the National Center for Health Statistics (NCHS) are subject to several kinds of problems. Since the source of these data (death certificates) has remained constant, biases in current data and those arising from comparisons with past data will be discussed together.

Rules governing the registration of deaths from one state to another may vary over time, thus introducing a source of bias and making comparisons more difficult. Classification systems used in the reporting of deaths have varied over time. Even when the exact type of cancer is detected while the patient is alive, the correct information may not appear on the death certificate. In addition, often only one underlying cause of death appears on a death certificate, and therefore some cancer deaths are incorrectly attributed to secondary causes or even missed entirely. Such errors are more likely to occur in elderly patients, and have been more of a problem in the past than now. In cases where the patient has widespread cancer, the primary site of the disease may not be known and/or properly recorded and thus the type of cancer is not specified on the death certificate. When cancer has spread, the death certificate may contain incorrect information as to its primary source. Sometimes a misdiagnosis of the cell type of cancer occurs. Many such errors can also affect incidence rates.

The annual *Cancer Facts and Figures* from the American Cancer Society (ACS) is a major source of cancer information disseminated to the public. It is thus fair to ask if the estimates of cancer death and incidence contained in this publication are accurate.

Because it takes time to compile nationwide information

from death certificates, the ACS uses death rates three to five years old to project trends for the coming year. In general, their track record has been very good. One study¹² found that when actual data become available and are compared to ACS estimates for specific sites, estimates are off by only two to four percent. The more common the type of cancer, the better the estimate tends to be. Estimates for sites with rapidly changing mortality (i.e., lung cancer in women) have been consistently low.

ACS estimates of incidence tend to be less accurate than death estimates, partly because actual rates from which estimates are made are limited to locations covered by SEER. Yet, estimates for incidence rates are made for the whole country. These estimates are also dependent upon the accuracy of population and mortality projections which may contain errors. Incidence estimates for states covered by SEER, and for cancer sites, have been in error by as much as 22.3 percent, though, in fairness, they have also been within one percent of actual figures. However, it is not uncommon for these estimates to be off by 15 to 20 percent.

One problem with all of the above sources of data is their comparability. For example, the ACS often reports data in actual numbers rather than rates, while the SEER and NCHS figures are reported as rates. In addition, when rates are adjusted by age they are not necessarily standardized to the same base population. This creates additional error. Thus, comparison of cancer statistics between publications or over time must be made with caution.

Time Trends in the U.S.: Incidence and Mortality

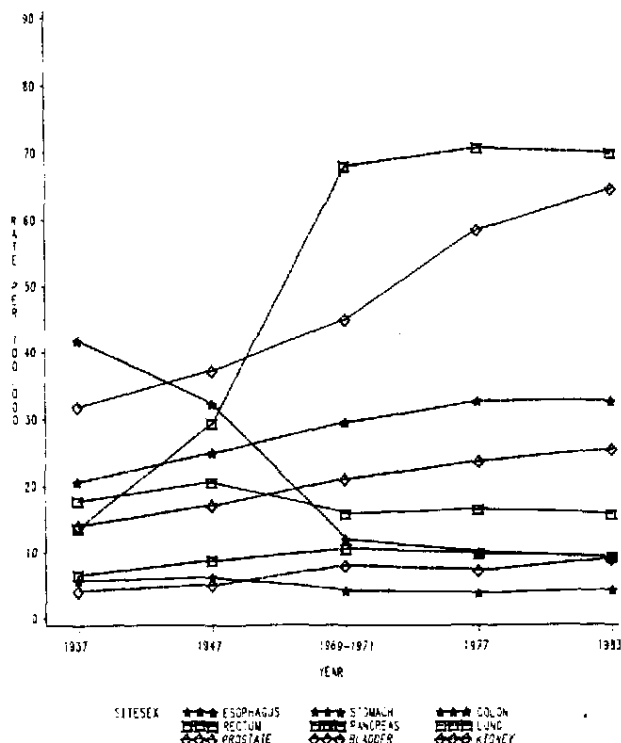
An important way of examining the issue of whether there is a cancer epidemic in the United States today is to look at patterns over time for cancers of specific sites. As mentioned above, incidence rates are theoretically preferable to death rates for this type of comparison. But, as noted, in recent years the incidence for some cancers has shown substantial fluctuations not easily explained by the nature of the disease. These variations seem likely to be statistical artifacts produced by a combination of overdiagnosis and improved reporting of cancer cases. Breast and prostate cancer are believed to be examples of this situation. In light of these biases, both incidence and mortality data over time are presented.

A. Incidence

The availability of data on incidence rates over time is limited. As mentioned earlier, there were three nationwide NCI cancer studies before the SEER program, covering the years 1937, 1947, and 1969-71. Using these three points in time and the SEER results, the following trends for cancer incidence emerge:

FIGURE 1

AGE-ADJUSTED CANCER INCIDENCE RATES FOR SELECTED SITES
MALE, UNITED STATES



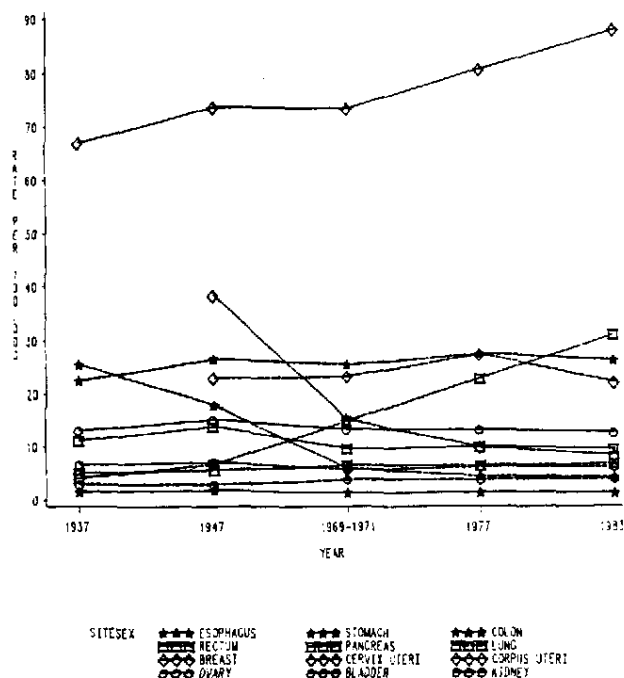
Source: Data from NCI cancer incidence surveys (1937, 1947, and 1969-71) and SEER (1977, 1983), adjusted to the age distribution of the 1950 U.S. Census population.

Figures 1 and 2 display incidence rates for the three national cancer surveys and for two years from the recent SEER studies. The data are available for white males and females from 1937 to 1983. We see that during this period there were sharp decreases in stomach and cervical cancer. Lung, breast, and prostate cancer all show increases. In males, bladder and kidney cancer increased.

The increase in breast cancer occurred between the 1969-71 and 1973-77 studies. As seen in Table 1, the

FIGURE 2

AGE-ADJUSTED CANCER INCIDENCE RATES FOR SELECTED SITES
FEMALE, UNITED STATES



Source: Data from NCI cancer incidence surveys (1937, 1947, and 1969-71) and SEER (1977, 1983), adjusted to the age distribution of the 1950 U.S. Census population.

incidence rate peaked in 1974, and until the last few years showed an erratic pattern. It is believed that the 1974 peak is due to early disease detection and over-diagnosis. In that year, greatly heightened public awareness of breast cancer followed the announcement that the wives of President Ford and Vice President Rockefeller had the disease. Because no known environmentally induced factor can explain this sudden rise and subsequent variations for breast cancer, it is reasonable at present to attribute this finding to a statistical artifact—a climate of increased public awareness. However, increases during the last few years suggest that such a conclusion may not apply to the most recent data.

TABLE 1

U.S. Incidence Rate per 100,000 for Female Breast Cancer, Whites, 1973-83*

Year	Rate
1973	79.8
1974	90.9
1975	84.6
1976	82.7
1977	80.8
1978	81.1
1979	81.3
1980	81.4
1981	85.0
1982	85.2
1983	87.8

* Age adjusted to the 1950 U.S. population.

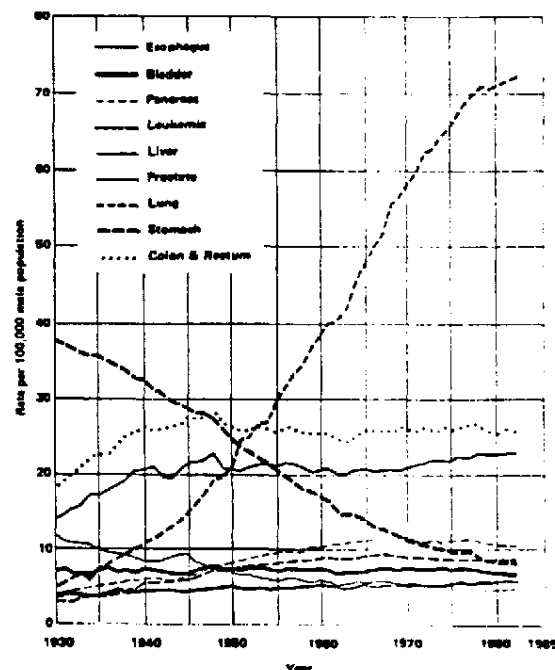
Source: SEER, unpublished computer run.

Cancer of the prostate shows a consistent increase in the period covered. As in the case of breast cancer, this may also be due to a statistical artifact. Doll and Peto² believe this pattern to be the result of a "vigorous search for lumps" resulting in overdiagnosis of the disease in cases where it is associated with old age and is not life-threatening. Frequently, this apparent increase is detected as a result of biopsies for noncancerous conditions. The average age at diagnosis of prostate cancer is about 73. Over the years the frequency and accuracy of autopsies have increased, and incidental findings of prostate cancer have been added to incidence cases. Thus, the recorded increase in prostate cancer may reflect increased biopsy and autopsy findings of cancers in men without any symptoms of the disease.

At this time, it is not known whether the increases in male bladder and kidney cancer are real or an artifact resulting from different methods of diagnosis and data collection in the different periods.

FIGURE 3

Age-Adjusted Cancer Death Rates* for Selected Sites
Males, United States, 1930-1982



Sources of Data: U.S. National Center for Health Statistics and U.S. Bureau of the Census
Adjusted to the age distribution of the 1970 U.S. Census population

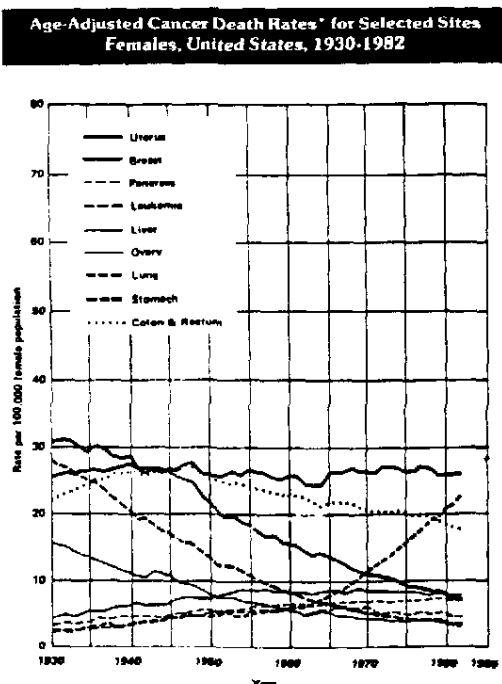
The conclusion from these incidence data is that there is an epidemic of lung cancer in the United States today. However, the most recent NCI data indicate that between 1982 and 1983 the incidence rate for lung cancer in white men decreased from 73.0 to 70.0 per 100,000, adjusted to the 1950 population distribution (82.7 to 79.3 per 100,000 adjusted to the 1970 distribution). This is the first such decrease in 50 years and corresponds to a substantial decrease in smoking patterns among men about 20 years ago. Among women the story is different. No such decrease is expected for 15 to 20 years because younger women are smoking more.

B. Mortality

Figures 3 and 4 present death rates for males and females from 1930 until 1982 (the latest data available as of this writing). An important advantage of these data is availability throughout the period. These data support the conclusions based on examination of incidence data.

For males, the sharp increase in deaths from lung cancer is apparent, as is the sharp decrease in deaths due to stomach cancer. The other forms of cancer tend to show steady death rates, especially in recent years. There is no

FIGURE 4



Sources: Cancer Rates and Risks, NIH publication 85-691 and Cancer Incidence and Mortality, NIH publication 85-1837.

marked increase in mortality from prostate cancer. This supports the argument that increases in incidence are largely artifactual. As for females, the increase in deaths due to lung cancer occurs later, as expected. Breast cancer mortality does not increase markedly, again suggesting that increases in past incidence data are artifactual. Death rates from stomach and cervical cancer also declined over the entire period, and those for colon and rectal cancer show a downward trend. Deaths from other cancers indicate a steady rate in recent years.

Current Cancer Patterns in the U.S.: Incidence and Mortality

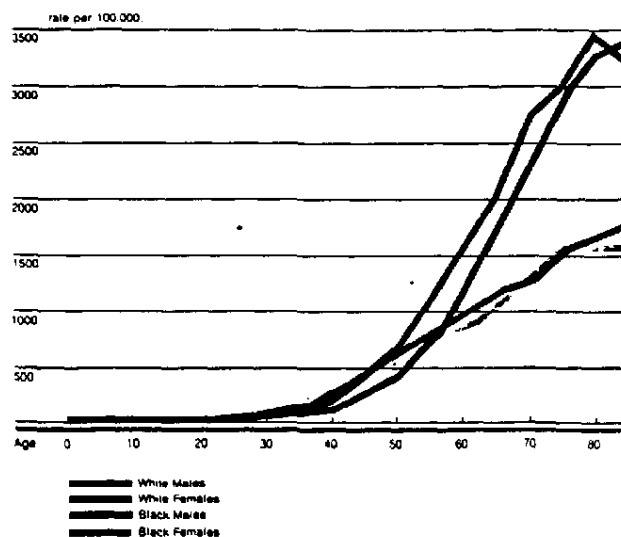
Examination of current cancer patterns is an important step in addressing the issue of whether there is a cancer epidemic. For example, by comparing racial and ethnic groups at the same point in time we can specify which groups have high rates for specific cancers. This may indicate trends toward an epidemic and further clarify the nature of the epidemic of lung cancer discussed in the previous section. These comparisons have been examined for men and women from the 1930s until the early 1980s. Comparing groups at the same point in time allows us to eliminate biases in the data attributable to

changes in recording procedures and diagnoses with the passage of time.

Before looking at comparisons of cancer rates for racial and ethnic groups, it is important to stress that cancer incidence (and mortality) increases significantly with advanced age. In fact, more than half of the cancer cases diagnosed today are among people 65 or older. Figure 5 displays this trend for white males, white females, black males, and black females. The sharp increase in incidence evident for all four groups after the age of forty is to be expected, as is the higher incidence of cancer among males. It does not indicate an overall epidemic, but rather explains how our mistaken perception of an epidemic may have come about.

FIGURE 5

**Average Annual Age-Specific Cancer Incidence per
100,000, U.S. Population, All Sites Combined, 1973-77**



Source: Unpublished data from SEER.

TABLE 2

**Average Annual Age-Adjusted Cancer Incidence Rates per 100,000
by Primary Site and Racial/Ethnic Group, U.S. SEER Program, 1978-81**

Primary Site	Whites	Blacks	Hispanics*	Japanese	Chinese	Filipinos	Native Hawallians	American Indians
All sites	335.0	372.5	246.2	247.8	252.9	222.4	357.9	164.2
Bladder	15.4	8.6	8.2	7.7	7.7	5.1	8.2	1.1
Breast, Female	86.5	71.9	54.1	53.1	54.0	43.4	111.1	28.5
Ages < 40	8.2	10.7	7.9	8.6	7.4	7.1	7.1	4.0
Ages 40 +	221.1	179.3	134.9	146.5	141.0	117.0	300.0	71.4
Cervix Uteri	8.8	20.2	17.7	7.6	11.2	8.8	14.1	22.6
Colon	34.6	37.9	15.8	34.0	27.7	17.7	18.4	8.0
Rectum	15.0	11.7	9.4	16.4	13.1	12.4	14.3	1.9
Corpus Uteri	25.1	13.4	11.1	18.6	17.6	11.7	27.1	2.6
Esophagus	3.0	11.5	1.6	2.4	3.4	3.6	6.4	2.4
Larynx	4.6	6.6	2.6	2.6	1.9	1.8	5.2	0.9
Lung, Male	81.0	119.0	34.3	45.1	62.6	38.1	100.9	14.6
Lung, Female	28.2	30.5	13.0	14.1	31.2	18.4	38.6	3.1
Multiple Myeloma	3.4	7.9	2.5	1.2	1.6	4.1	5.5	2.8
Ovary	13.6	9.5	10.4	8.7	9.1	9.4	13.5	3.2
Pancreas	8.9	13.6	10.8	7.4	9.3	6.7	10.0	6.0
Prostate	75.1	120.3	76.5	44.2	26.1	48.9	57.9	45.4
Stomach	8.0	13.8	15.7	27.9	9.0	7.0	32.4	19.3

* Cancer incidence data for Hispanics come from New Mexico only.

Source: Baquet, C.R. et al. *Cancer Among Blacks & Other Minorities: Statistical Profiles*. NIH Publication No. 86-2785. Bethesda, Md., National Cancer Institute, March 1986, page 9.

TABLE 3

**Average Annual Age-Adjusted Cancer Mortality Rates per 100,000
by Primary Site and Racial/Ethnic Group, U.S. Total United States, 1978-81**

Primary Site	Whites*	Blacks	Japanese	Chinese	Filipinos	Native Hawaiians	American Indians
All sites	163.6	208.5	104.2	131.5	69.7	200.5	87.4
Bladder	3.9	3.8	1.8	1.7	1.5	1.6	1.0
Breast, Female	26.6	26.3	9.9	13.0	8.0	33.0	8.2
Ages < 40	1.6	2.5	1.1	0.8	0.9	1.2	1.1
Ages 40 +	70.2	68.1	25.2	34.6	20.6	88.7	20.6
Cervix Uteri	3.2	8.8	2.7	2.9	1.6	4.2	5.8
Colon	18.1	18.8	13.6	15.5	5.8	11.4	6.8
Rectum	3.5	3.5	3.6	3.8	2.3	3.6	1.8
Corpus Uteri	3.9	6.6	3.9	4.3	2.0	3.0	1.8
Esophagus	2.6	9.2	1.9	3.3	1.9	6.5	2.1
Larynx	1.3	2.5	0.2	0.7	0.4	1.4	0.9
Lung, Male	69.3	91.4	32.7	48.2	20.0	88.0	28.0
Lung, Female	20.2	20.1	8.6	21.2	6.8	31.5	8.6
Multiple Myeloma	2.4	5.0	1.2	1.2	1.2	2.8	1.9
Ovary	8.1	6.4	4.3	4.2	2.8	7.0	3.3
Pancreas	8.4	11.0	7.0	7.4	3.3	10.9	4.5
Prostate	21.0	43.9	8.8	7.5	8.2	11.6	15.5
Stomach	5.3	10.0	17.5	7.8	3.3	25.3	6.2

* The National Center for Health Statistics from which these data are derived does not code ethnicity for Hispanics.

Source: Baquet, C.R. et al. *Cancer Among Blacks & Other Minorities: Statistical Profiles*. NIH Publication No. 86-2785. Bethesda, Md., National Cancer Institute, March 1986, page 10.

VVV 000008728

People are living longer than ever. Earlier in the century infectious diseases were the big killers but modern medicine and improved health measures have eliminated them as major causes of death and allowed many people to live long enough to get cancer, a disease that often takes several decades to develop. Also, the population has not only grown, but grown older. Therefore, if one looks only at the "crude figures," i.e., the number of cancer cases and impressions gained from media reports and personal knowledge of cancer victims, it may appear that there is an epidemic. However, if one converts these figures into rates (thus controlling for the size of the population) and statistically adjusts them to a standardized age distribution, they become more meaningful. As seen in the previous section, with the exception of lung cancer, they do not reveal any evidence of an epidemic.

Incidence and Mortality among Racial and Ethnic Groups

As discussed, SEER data are collected so as to include different racial and ethnic groups and thus allow for comparisons of cancer rates among these groups. Table 2 presents incidence rates for eight different groups and includes information by sex.

Overall cancer rates vary considerably among the groups. Blacks have the highest cancer incidence rate, followed by native Hawaiians. American Indians have the lowest. Blacks have the highest rates for cancer of the breast (women under 40), esophagus, colon, larynx, lung (male), pancreas, prostate, and multiple myeloma. They also have a high rate for cancer of the cervix.

Based on SEER data, whites have the highest rate for cancer of the bladder and ovary. American Indians have the highest rate for cervical cancer and native Hawaiians the highest rate for cancers of the breast in women over 40, and for cancers of the uterine corpus (cancer of the endometrium or lining of the uterus), stomach, and lung (female). Japanese Americans have the highest incidence rates for cancer of the rectum.

These data indicate that there is a cancer epidemic among blacks in this country as compared to other subgroups of the population. They further specify which types of cancer account for this epidemic. The explanation of this epidemic is beyond the scope of this report but the causes of cancer discussed in Part II will suggest some answers. Of particular relevance to blacks may be the risks associated with diet and smoking as well as other aspects of lifestyle.

Mortality data confirm this demographic pattern (Table 3). Blacks have the highest overall cancer mortality. Black mortality is highest for all cancers in which they have the

highest incidence rate: cancer of the breast (women under 40), esophagus, colon, larynx, lung (male), pancreas, prostate, multiple myeloma, cervical cancer and cancer of the uterine corpus. Thus, mortality data confirm a cancer epidemic among blacks in the U.S.

Another Way of Looking at Current Patterns: Gender

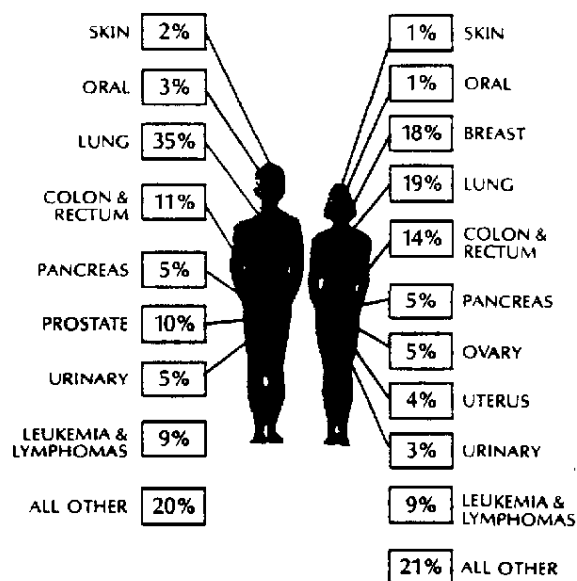
Figures 1-4 display differences in cancer rates between males and females. Incidence rates are from 1937, 1947, 1969-71, 1977, and 1983. Mortality rates are from 1930 through 1982. Another approach to current male-female cancer patterns is to look not at rates but at only the percentage distribution of deaths for the major forms of cancer among individuals who have the disease.

Each year, the American Cancer Society prepares estimates of the percentage distribution of cancer incidence and death by site and sex for the upcoming year. Since their projections for mortality tend to be much more accurate than those for incidence, only the mortality estimates are presented here (Figure 6).

The high percentage of expected death from lung cancer for men is readily apparent. Of the men expected to die

FIGURE 6

Estimated Percentage of Cancer Deaths by Site, Male and Female, 1986



Source: American Cancer Society

from cancer in 1986. 35 percent were projected to die from lung cancer! For women, the corresponding figure is 19 percent. The latter figure is of particular significance because it represents the first time that lung cancer will surpass breast cancer as the leading killer of women. (It has already done so in at least 15 states.) Thus, by looking only at cancer victims we have again confirmed the presence of a lung cancer epidemic.

International

Sources of Data

The major source of data for international disease incidence and mortality rates is the World Health Organization (WHO), specifically its affiliate the International Agency for Research on Cancer (IARC). Periodically, IARC publishes international incidence data, the latest volume being *Cancer Incidence in Five Continents*, Volume IV (1982). This volume contains data from 58 cancer registries, although it does not cover all countries on every continent. WHO also publishes updated data on mortality.

International Comparisons of Cancer Statistics

Comparison of cancer incidence and mortality among countries presents many potential sources of error. Data for each country are subject to the kinds of errors for incidence and mortality discussed earlier for the United States. Since the type of errors can vary by country and registry, comparisons can serve to magnify errors.

Of particular importance is the fact that incidence data are usually based on a small number of registries in a particular country. The data fall into specifically identified regions, states, counties, and metropolitan areas, and it is unclear how representative these registries are for the total population of the country. In addition, the extent to which people use medical services (often varying according to age), the availability and affordability of these services, the level of diagnostic ability, and the quality of data in the registry—compliance in reporting, careful checks and “cleaning” of the data—may all differ by registry and country.

A major problem with mortality rates is that the level of technology used in diagnosis and the medical treatment given to the patient can affect lifespan. Therefore, a high or low mortality rate may not truly reflect cancer mortality, but rather the influence of the level of diagnosis and treatment.

Because of these limitations we will primarily rely on the international data to help confirm the epidemics of lung

cancer and cancer among blacks already found in the time comparisons and racial and ethnic comparisons, respectively. Of course, there is also the possibility that the U.S. data for a particular site will not reveal an epidemic when in fact international comparisons do. Because of the serious limitations of international data, evaluation of these findings is beyond the scope of this report.

Internationally compared cancer incidence and mortality data are age-standardized to a world standard population. Sometimes different world standard populations are used for different continents. However, despite this precaution the influence of age can exert an effect on the data through such processes as differential use of diagnostic and treatment facilities by various age groups.

The IARC volume presents cancer incidence data by site and sex. For each site by sex appear high and low rates of incidence and the location of source registries. However, these comparisons are inappropriate for defining a cancer epidemic in the U.S. If a registry in a country is singled out as having the highest (or lowest) incidence for a particular cancer, it may or may not be representative of the rest of the country. Thus, international incidence data can best be used to help confirm patterns previously found. International mortality data may lend support to conclusions from the incidence data.

Table 4 presents international incidence data from “around” 1976 (the year of data collection varies slightly by country) for cancers discussed previously of which the United States has the highest incidence rate recorded. Among white males there is no evidence of an overall epidemic, but the rate for colon cancer is notably high. However, black males have the highest international rates for cancer of the pancreas, lung and bronchus, prostate, and multiple myeloma.

Among females there is also no evidence of an overall U.S. cancer epidemic and no evidence for an epidemic among black females.

Table 5 presents the other side of the coin: the international comparisons for incidence for which the U.S. has the lowest rates. The low rate of stomach cancer among males is consistent with previous findings.

Conclusion

Mortality rates are available for 47 countries. Table 6 presents the U.S. world standardized rate and rank for all sites combined for eight major cancers. The overall rates and ranks indicate no evidence of a cancer epidemic for either sex. While the mortality data are consistent with an epidemic of lung cancer for both sexes, there is nonetheless no conclusive evidence of an overall cancer epidemic in the U.S.

TABLE 4

**Comparison of International Incidence of Cancer per 100,000 for
Selected Sites by Sex: Highs for the United States, around 1976***

<u>Males</u>					
<u>Site</u>	<u>High</u>		<u>Low</u>		
	<u>Population</u>	<u>Rate</u>	<u>Population</u>	<u>Rate</u>	<u>Ratio H/L</u>
Colon	U.S., Connecticut	32.3	India, Poona	3.1	10.4
Pancreas	U.S., Bay Area, Black	18.3	India, Bombay	2.0	9.2
Lung & Bronchus	U.S., New Orleans, Black	107.2	U.S., New Mexico, Am. Indian	8.1	13.2
Prostate	U.S., Alameda, Black	100.2	Shanghai	0.8	125.3
Kidney	U.S., Hawaii, White	11.2	India, Bombay	1.3	8.6
Multiple Myeloma	U.S., Bay Area, Black	8.4	India, Poona	0.6	14.0

<u>Females</u>					
Colon	U.S., Bay Area, Japanese	27.4	India, Poona	2.8	9.8
Pancreas	U.S., New Mexico, Am. Indian	10.4	India, Bombay	0.9	11.6
Breast	U.S., Hawaii, Hawaiian	87.5	Japan, Osaka, Rural	8.9	9.8
Corpus Uteri	U.S., Alameda, White	38.5	Japan, Fukuoka, Rural	1.0	38.5
Bladder	U.S., New Orleans, White	6.5	Hungary, Szabolcs	0.5	13.0
Multiple Myeloma	U.S., Hawaii, Hawaiian	5.9	Poland, Katowice	0.4	14.8

* Age - standardized to the Standard World Population.

Source: Waterhouse et al., 1982.

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TABLE 5

Comparison of International Incidence of Cancer per 100,000 for Selected Sites by Sex: Lows for the United States, around 1976*

<u>Site</u>	<u>Males</u>			
	<u>High</u>	<u>Rate</u>	<u>Population</u>	<u>Low</u>
Stomach	Japan, Nagasaki	100.2	U.S., Atlanta, White	<u>Rate</u> 5.7
Lung & Bronchus	U.S., New Orleans, Black	107.2	U.S., New Mexico, Am. Indian	8.1
Esophagus	India, Bombay	10.7	U.S., Utah	0.4
				<u>Ratio H/L</u> 17.6 13.2 26.8

* Age - standardized to the Standard World Population.

Source: Waterhouse et al., 1982.

TABLE 6

U.S. Mortality Rate per 100,000 and Rank Among 47 Countries for Cancer, Selected Sites by Sex, 1980-81*

	<u>Male</u>		<u>Female</u>	
	<u>Rate</u>	<u>Rank</u>	<u>Rate</u>	<u>Rank</u>
All Sites	215.9	21	135.5	18
Oral	5.6	13	1.9	11
Colon & Rectum	25.1	17	18.2	17
Lung	71.6	9	21.4	6
Breast			26.6	15
Uterus			7.7	36
Stomach	8.2	45	3.9	45
Prostate	22.7	14		
Leukemia	8.7	3	5.2	10

* Age - standardized to the Standard World Population.

Source: World Health Statistics Annual, 1982-84.

Part II: What Causes Human Cancer?

While there is still an interest in the role of genetics in cancer risk, current research strongly suggests that external factors—aspects of our environment—cause most human cancers.

The importance of environment in cancer causation has been illustrated dramatically by studies of migrants from one part of the world to another. They or their descendants quickly take on the cancer patterns typical of their new home. It is also significant that dramatic changes in cancer rates sometimes occur in only a few decades. Genetic changes cannot account for this; environmental changes can.

Since 1970, professional and popular articles have frequently cited an estimate that "80 to 90 percent of all cancers are environmentally induced." This estimate was originally calculated by the International Agency for Research on Cancer (IARC), a World Health Organization affiliate. It has often been misinterpreted, leading to the mistaken impression that 80 to 90 percent of all cancers are due to *known* environmental factors such as air and water pollution, industrial chemicals or food chemicals and contaminants.

This IARC estimate was derived by comparing the highest and lowest cancer death rates from around the world. In assessing the meaning of this controversial statement, we must emphasize two points. First, saying that 80 to 90 percent of cancer is environmentally caused is not the same as saying that we know what these factors are and that we can control them effectively. The specific causes of most cancers are unknown.

For example, two well-known scientists who have addressed the risk posed by industrial pollution—a widely suspected cause of cancer—estimate that only about two percent of all cancers are attributable to such pollution. (Doll, R. and R. Peto, *The Causes of Cancer*, Oxford University Press, New York: 1981).

Second, our "environment" is not limited solely to our physical surroundings and man-made chemicals. Many scientists now believe that cultural and personal habits contribute more to cancer causation than environmental pollution or toxic chemicals. In its broadest sense, then, our environment encompasses such factors as smoking, diet, sexual and reproductive patterns, alcohol consumption, sunbathing, and other aspects of lifestyle, as well as our purely physical surroundings.

Tobacco

National incidence and mortality figures confirm the drastic and unprecedented rise in lung cancer in the United States. Yet before 1930, lung cancer was a rare disease. In 1912, a physician commenting on U.S. disease patterns wrote: "There is nearly a complete consensus of opinion that primary malignant neoplasms of the lung are among the rarest forms of disease." However, by 1950, clinical and statistical evidence clearly demonstrated a significant increase in lung cancer among men.

Since 1970, the same pattern of increasing incidence has also been observed among American women. Numerous clinical, epidemiological and laboratory studies have confirmed that the increase in lung cancer is directly related to cigarette smoking. It has been shown that lung cancer death rates are more than ten times higher among male cigarette smokers than among male nonsmokers. Although precise estimates of the number of lung cancer deaths caused by smoking vary, most experts agree that at least 80 and perhaps 90 percent of all lung cancer deaths are the result of cigarette smoking.

Smoking acts to increase cancer risks for body sites in addition to the lung. Tobacco smokers have greater risks than nonsmokers for cancers of the mouth, throat, bladder, pancreas and kidney. Cigarette smokers have two to three times greater risk for developing bladder cancer than nonsmokers. Scientists have estimated that cigarette smoking and other tobacco use is responsible for roughly 30 percent of all cancers in the United States.

Despite the clear association between smoking and cancer, the process by which cigarettes cause cancer has not been fully explained. Most research suggests that several chemicals found in tobacco smoke are the ultimate carcinogenic agents. However, other factors are equally important. These include the length of time a person has smoked, the total number of cigarettes consumed, how deeply the smoke is inhaled and, to a lesser extent, the brand of cigarette smoked.

Cigarette smoking can also act to increase or "promote" the cancer causing potential of other risk factors. It is known, for example, that drinkers of alcohol who also smoke heavily have greater risks for developing esophageal cancer than would be predicted by simply adding the two risks together. This synergistic effect has also been found among smokers exposed to certain hazardous materials such as asbestos and radon. These facts prompted the statement made in the 1979 U.S. Surgeon General's Report: "Cigarette smoking is the single most important environmental factor contributing to premature mortality in the United States."

Diet

There is substantial scientific evidence suggesting that diet may be an important factor in human cancer development. However, the exact roles played by specific aspects or components of the diet are still very uncertain.

The results of some scientific studies suggest that a high-fat diet may be associated with increased rates of cancers of the breast and colon-rectum. However, other studies have failed to support this conclusion. Still other studies point to increased calories from any source, rather than to fat per se.

There are similar discrepancies in the results of studies on the possible protective effect of dietary fiber against colon cancer. If there is such an effect, specific fiber components such as the pentosan fraction, rather than fiber as a whole, may be responsible. Different fiber components have different physiological effects in the intestine. Thus, attributing effects to fiber in general may be highly misleading.

Obesity has been associated with an increased risk of cancer at several body sites in some studies.

Vegetables, fruits, vitamins A, C, and E, and the mineral selenium are all being investigated for possible protective effects, but their benefits have not been shown in humans. Experts warn against the use of high-dose supplements of individual nutrients. Such supplements may be unsafe and have not been shown to be effective.

International epidemiologic studies have linked the smoked and cured foods popular in Japan, China, and Iceland with increased rates of stomach and esophageal cancers. The very different types of smoke-flavored and cured foods consumed in the U.S. have not been similarly implicated.

A large number of substances that occur naturally in foods have been found to be carcinogenic when evaluated by standard laboratory methods and criteria. Many additional carcinogens are produced in food by cooking or by the actions of microorganisms. The evidence currently available does not indicate that any of these substances has a measurable effect on cancer incidence in the U.S.

Food additives and agricultural chemicals have not been found to have an impact on U.S. cancer rates.

Alcohol

Although alcohol is at most a weak carcinogen, heavy alcohol consumption has been implicated in human cancers. Consumption of large amounts of alcohol in combination with heavy cigarette smoking synergistically increases the risks of cancers of the mouth and throat.

Heavy alcohol consumption alone may also have an indirect effect on liver cancer because it is capable of inducing cellular changes in liver tissue and this in turn may increase liver cancer risk. The effects of alcohol consumption are closely related to the extent and duration of use and the type of alcoholic beverage consumed.

Ionizing Radiation

Exposure to ionizing radiation, for example medical x-rays, increases the risk of developing leukemia and skin cancer.

As early as 1944, researchers noted that radiologists were more likely than other physicians to develop leukemia. Recent attention to this cancer risk has resulted in recommendations against the use of x-rays as a routine medical practice. However, experts agree that when x-rays are used prudently, their diagnostic and therapeutic benefits far outweigh their small cancer risk.

Drugs

In rare cases, certain drugs have contributed to an increase in cancer risk. The most prominent example is DES* (diethylstilbestrol), a drug that was widely prescribed to prevent miscarriages. In 1974 an obstetrician noted a rare form of vaginal cancer in women whose mothers had taken large doses of the drug during pregnancy. Although widely publicized, the actual number of cases of cancer caused by the use of DES as a human drug is small.

Postmenopausal estrogens are a risk factor for endometrial cancer. Extended use (more than two years) of these drugs has been shown to increase the risk of cancer of the lining of the uterus, the endometrium. This risk must be weighed against the potential benefits of the drug. For some women, the protective effect of estrogens against loss (osteoporosis) far outweighs their cancer risk. If estrogens are used as short-term therapy, they not only do not increase cancer risk and are effective in treating certain menopausal symptoms such as hot flashes and genital atrophy.

Other drugs used to treat tuberculosis and, ironically, some forms of cancer have also shown an increased risk potential. Yet for these drugs, the risks of not using them are far more serious than their cancer risk. For this it is important to weigh carefully both the risks and benefits of any drug before use.

* Until recently DES was also used as a cattle growth stimulant, but was discontinued after trace amounts were found in the livers of some animals. There is no evidence that these trace amounts were a risk to human health.

Sexual and Reproductive Patterns

Intercourse at an early age and multiple sexual partners have been linked to an increased risk of cervical cancer. Strong evidence suggests that cervical cancer may be caused by a sexually transmissible disease.

AIDS, another sexually transmitted disease, is also associated with cancer. Kaposi's sarcoma, a rare type of cancer, occurs frequently among victims of AIDS. Other cancers, such as those of the lymphatic system, may also be unusually common among individuals who have AIDS.

Sunlight

The most widespread environmental carcinogen, accounting for the majority of superficial skin cancers and for some types of melanoma, is ultraviolet radiation from the sun. Superficial skin cancers are rarely fatal and are not included in most statistical estimates of U.S. cancer patterns. Melanoma is more serious, but overall, the outcome of treatment of this cancer is good when it is discovered early.

Among light-skinned people, sunlight-related cancers are more common in tropical areas than in colder climates because the sun's rays are most intense near the equator. There are also individual differences in susceptibility to the effects of the sun, with people who tan poorly and sunburn easily being most at risk.

Sun exposure can cause cancer in two ways. Total exposure, over many years, increases the risk of superficial skin cancer and at least one type of melanoma. Intense, occasional exposures, especially in childhood and adolescence, increase the risk of two other types of melanoma.

Limiting exposure to the sun and to other sources of ultraviolet light is the simplest and most effective means of reducing this cancer risk. It is best not to cultivate a suntan or to patronize sun tanning parlors. It is helpful to use sunscreen products and to wear protective clothing, especially when exposed to midday summer sun.

Occupation

High dose, long-term exposure to a number of industrial chemicals and manufacturing processes, including asbestos, vinyl chloride, nickel refining, and dye manufacturing, can increase risk for several types of cancer. To date, approximately 20 industrial chemicals have been confirmed as human carcinogens. An additional 200-300 are suspected carcinogens on the basis of animal evidence. As with other cancer risks, occupational risks depend on the length and degree of exposure, the potency of the chemical agent, possible interactions with other chemicals, and lifestyle factors such as cigarette and alcohol

consumption. Most experts estimate that between one and five percent of all cancers in the U.S. are related to occupation.

Air Pollution?

The findings of trace amounts of carcinogens in urban and suburban air samples and the differences in cancer death rates between urban and rural areas have led many to conclude that pollution is a serious cancer risk. However, the case for air pollution as an important cause of human cancer remains unconvincing.

A number of reviews of the relationship between air pollution and lung cancer have reported no evidence of an association. Differences in lung cancer death rates between urban and rural people can be explained by differences in smoking habits. A recent study of lung cancer by the American Cancer Society concluded: "General air pollution had little effect in a comparison between urban and rural people. Smoking is the key factor."

This is not to suggest that pollution should not be controlled. There may be other health and aesthetic reasons to minimize environmental pollution. Some preliminary evidence suggests that certain types of air pollution may exacerbate acute respiratory illnesses such as asthma and bronchitis. Yet at present, the threat of cancer does not appear to be associated with general pollution.

Recently, attention has focused on the possibility that indoor air pollution may pose a threat to health. Some scientific studies suggest that prolonged exposure to one indoor air pollutant, environmental tobacco smoke, may be associated with an increase in lung cancer risk. The possible role of radon is also under investigation.

Cancer and the Environment: An Overview

The age-adjusted death rates for most forms of cancer have decreased or remained constant for the past 50 years. Lung cancer is a notable exception. This disease, caused primarily by cigarette smoking, has increased dramatically in both sexes. Black Americans have higher cancer rates than other ethnic and racial groups; lifestyle factors, particularly smoking, may be responsible.

The exact number of cancers that can be eliminated by sound preventive methods is unknown, but the percentage is undoubtedly substantial. Eliminating the effects of cigarette smoking alone would eventually reduce cancer incidence and mortality between 15 and 35 percent.

Some experts believe that when the dietary factors that may influence cancer risk are fully understood, a similar reduction might be achieved by changes in eating habits. However, other authorities consider this much too optimistic. Some cases will likely be prevented as a result of

reductions in occupational exposures to toxic chemicals. If it became unfashionable to cultivate a suntan, the rates of skin cancer and melanoma might be substantially reduced.

Effective preventive measures can successfully reduce our cancer burden or at least postpone cancer until very late in life. To this end, current scientific knowledge suggests that our preventive efforts should be focused on personal activities, especially smoking, sunbathing, and excessive alcohol consumption.

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