Tobacco and Health: a Review of the History and Suggestions for Public Health Policy

ERNST L. WYNDER, MD

Dr. Wynder is Founder and President of the American Health Foundation in New York City and its research institute at Valhalla, NY. This paper is based on his presentation at the National Cancer Institute's Workshop on the Smoking, Tobacco, and Cancer Program and its Goals for the Year 2000, which was held in Bethesda, MD, April 27-29, 1987. Tearsheet requests should be sent to Dr. Wynder at the American Health Foundation, 320 East 43d St., New York, NY 10017.

At the workshop, Dr. Wynder had this to say about his lifework: "The challenges of the scientific investigations on the tobacco and cancer question have led me to conclude that the human body is a nearly faultless organism if we do not overload it with toxic and carcinogenic burdens. Studies of the epidemiology of lung cancer and of our biochemical fitness to handle xenobiotics have taught us that lung cancer and other cancers are not an inevitable consequence of life and aging but have identifiable causes and thus are preventable."

Dr. Wynder began his research career in 1948 with the question, "Does cigarette smoking cause lung cancer?" A summer student at New York University that year, impressed with the rise in the prevalence of lung cancer, principally among men, he designed a questionnaire and began to interview patients on the wards of Bellevue Hospital about their smoking habits. With this approach, Dr. Wynder initiated the landmark case-control study that established him as an early leader among investigators of the cancer-tobacco question.

Synopsis

The suspicion that the use of tobacco adversely affects health existed for some time before a case-control study appeared in 1950 of 684 cases of lung cancer strongly associated with cigarette smoking. This paper, a brief history, describes the background of the 1950 landmark study as well as other pertinent epidemiologic and experimental studies of the 1950s and 1960s. This body of research provided evidence for the causative association between tobacco use and lung cancer and other chronic diseases such as several other types of cancer and coronary heart disease.

Despite this body of evidence, support from scientists, health professionals, and government officials on the issue of smoking and health came slowly. The scientific application of this discovery to prevent tobacco-linked diseases continues to be far more difficult than the discoveries themselves. Thus, although the low-yield cigarette has provided some assistance to smokers, smoking prevention is far more important, and greater efforts are needed to achieve cessation, particularly among women and minority groups.

Beyond this approach, efforts to prevent children and young people from beginning to smoke should stress State-mandated school health education beginning in the earliest grades. The Know Your Body School Education Program, which includes an annual screening with the results entered into a personalized Health Passport starting in first grade, has demonstrated reductions in onset of smoking as well as improved health behavior in nutrition.

To further reduce tobacco use, cancer prevention units staffed by health educators, behavioral scientists, and epidemiologists should practice prevention on a communitywide basis. The prevention of diseases as the ultimate aim of medicine and science can be demonstrated by the smoking and health issue that establishes that the prevention of many cancers is attainable.

THE IDEA that the use of tobacco can induce cancer—and otherwise adversely affect human health—was expressed well before the 1940s. As early as 1795 Soemmering had described an apparent association between pipe smoking and lip cancer (*1*). Abbe reported, in 1915, on a woman who applied snuff with a toothbrush; she developed cancer of the tongue (*2*). Although Adler stated, in a monograph in 1912, that lung cancer was relatively rare, he was one of the first researchers to suggest that tobacco might play a role in the induction of lung cancer (*3*). This suspicion was echoed by several clinicians and statisticians in the United States and Europe in the following three decades, but few investigators endeavored a systematic study (*4–13*). Most clinical histories taken from lung cancer patients in those days did not include a history of smoking. An increase in the prevalence of lung cancer was generally attributed to air pollution or improved diagnostic methods (*14–16*).

In 1933, Cook and coworkers in England described the isolation of cancer-producing hydrocarbons from coal tar (*17*). That the burning of

tobacco leads to similar products was first shown by Roffo in South America in 1939 when he reported the isolation of 3,4-benzopyrene from tobacco tar (*18*). In the late twenties, reports of sporadic attempts to bioassay tobacco extracts or distillates in laboratory animals began to appear in the literature, but most of these assays were inconclusive because of the toxicity of the test agent or because the experiment was stopped too soon (*19*).

From the knowledge that the burning of tobacco in pipes, or as cigars or cigarettes, could lead to the formation of cancer-causing chemical compounds, I thought it plausible that repeated inhalation of tobacco smoke could induce malignant transformation of the epithelial cells.

The Beginning

This was the state of knowledge in 1948 when, as a summer student at New York University, I began to conduct a case-control study. Having received permission to interview patients from Dr. Burns Amberson, Chief of the Chest Service at Bellevue, I developed a questionnaire and then interviewed sufficient persons in one summer to impress Dr. Evarts Graham, Chief of Surgery at Washington University School of Medicine. Dr. Graham permitted me to continue the interviews on his surgical service during my junior year in medical school, even though some of his associates considered such an exercise to be "futile."

In the winter of 1948, I visited Dr. Charles Cameron, Medical Director of the American Cancer Society. He appeared at first skeptical, but upon seeing our early results, suggested we apply for a grant to continue this study. Thus, with funding from the American Cancer Society in the spring of 1949, we hired an assistant, Adele Croninger, expanded the interview schedule, and started the first experimental studies with tobacco smoke condensate.

At the February 1949 National Meeting of the American Cancer Society on lung cancer, held in Memphis, TN, we were able to present a study of some 200 cases and controls that showed a high correlation between smoking and lung cancer. I recall with some dismay that not a single question or comment was voiced following the presentation, an indication that the issue of smoking and lung cancer was not in the forefront of research interests at that time. It is particularly ingrained in my mind that the subsequent speaker, presenting a talk on pulmonary adenomatosis in sheep and its viral etiology, received considerable attention.

The time until graduation in May 1950 was spent in conducting interviews of lung cancer patients nationwide, building apparatus for the collection of smoke condensates (tars), applying these tars to the skin of mice, and readying Dr. Graham's and my first report on smoking and lung cancer, which was published in the Journal of the American Medical Association on May 27, 1950 (*20*). The conclusion we reported was that "smoking, especially in the form of cigarettes, plays an important role in the etiology of lung cancer." In September of 1950, Doll and Hill in Great Britain reached a similar conclusion in their case-control study (*21*).

It has been 40 years since I began my scientific career in the wards of Bellevue Hospital with the question, "Does cigarette smoking cause lung cancer?" It has been 36 years since I started as an epidemiologist at Sloan Kettering Memorial Hospital, and 17 years since founding the American Health Foundation and becoming its president. During all that time, our investigations have continued in multiple studies on the epidemiology of other cancers and chronic diseases, on assessment of risk factors, and in the search for preventive strategies and their application. For me, the opportunities science offers to unravel nature's pathways have never ceased to be exciting.

The Discoveries

During the 1950s, my appointment by Dr. C. P. Rhoads as Head of the Section of Epidemiology, at the Sloan Kettering Institute for Cancer Research, provided the opportunity to conduct a variety of case-control studies of cancer sites that were found to be associated with tobacco usage: cancer of the larynx in 1956, cancer of the mouth in 1957, cancer of the esophagus in 1961, cancer of the bladder in 1963, cancer of the pancreas in 1973, cancer of the kidney in 1974 (*22–28*). Although Graham and I had already shown in 1950 that women, like men, were susceptible to tobacco smoke carcinogens, my group published a separate case-control study on lung cancer in women in 1956 (*29*).

One study conducted with Jerome Cornfield of the National Institutes of Health (NIH) in 1953 was unique because of its minimal cost, less than $100 (*30*). We sent questionnaires to families of physicians recorded in the Journal of the American Medical Association as having died from lung

cancer and compared their smoking habits with those of patients who had died from cancer of the large bowel. A high response rate to our letter from the families made the study viable, and the fact that physicians had no known occupational exposure to respiratory pollutants and were all of the same occupation gave special pertinence to this study. Physicians, like anyone else who smoked cigarettes, had a high risk of lung cancer. Several prospective studies—from England by Doll and Hill (*31–33*), from Canada by Best and coworkers (*34*), and from the United States by Hammond and Horn (*35*) and by Dorn (*36*)—lent strong support to the case-control studies.

After we had investigated the epidemiologic implications of tobacco smoking in the development of cancer and realized the experimental and biological limitations to model studies in laboratory animals with smoke inhalation, we turned to mouse skin and rabbit epithelium as test sites for tobacco tar, the solid particulate matter of the smoke. Cigarette smoke condensate induced cancer of the skin in both mice and rabbits (*37,38*). In 1957, together with Dr. George Wright of the University of Toronto, we presented further evidence of carcinogens in tobacco smoke condensate, as could have been predicted from our knowledge of the carcinogens present after incomplete combustion of any organic matter (*39*). These procedures were later refined by Dr. Dietrich Hoffmann. It is this chain of evidence—epidemiologic, biological, chemical, together with biological plausibility and common sense—that led us to conclude that cigarette smoking and, for that matter, tobacco use in general is indeed carcinogenic to humans.

We learned much about methodological techniques during our studies of tobacco carcinogenesis. Following the advice of Pasteur that "above all a scientist needs to know what *not* to do," we learned early that merely exposing mice, rats, and hamsters to tobacco smoke was not likely to induce lung cancer, because the nasal turbinals of rodents represent a highly developed defense system against foreign matter in the respiratory air as a consequence of their evolution in adapting to living so close to the dusty ground.

On the other hand, we knew then and know even better today that what makes tobacco smoke carcinogenic to man is its complex mixture of chemicals, which presented ample challenge for chemists. Dr. Hoffmann, who joined our research group at the Sloan Kettering Institute in 1957 and has remained my collaborator and friend for three decades, contributed much to our knowledge of biologically active smoke constituents through systematic chemical analytical and biological approaches (19). In the 1970s, at the American Health Foundation, Dr. Hoffmann and his associate Dr. Stephen S. Hecht documented the presence of nicotine-derived carcinogens in tobacco and tobacco smoke. Recently, they have shown that the metabolites of these tobacco-specific carcinogens form chemical lesions with guanine and thymidine in the DNA molecule (*40*). Dr. Hecht also demonstrated the importance of molecular configuration of chemical compounds in carcinogenic activity in an elegant study involving fluoro-substitution of the carcinogenic aromatic hydrocarbon S-methyl chrysene, followed by other important studies on structure-activity relationships (*41*).

Thus, through chemical and biochemical investigations, we have learned much about carcinogens, tumor promoters, and accelerators, as well as inhibitors of carcinogenesis. Our work in tobacco carcinogenesis, therefore, has purposefully contributed to our understanding of mechanisms of chemical carcinogenesis in animals and humans (*42*).

Whereas our 1950 publication stated that the use of tobacco, especially cigarette smoking, was "an important factor in the production of bronchiogenic carcinoma," in 1954 I titled an article "Tobacco As a Cause of Lung Cancer" with special reference to the infrequency of lung cancer among nonsmokers (*43*). The article reported that the available evidence presents "definitive proof that tobacco may act as a carcinogen to the human bronchial epithelium," and added that the word "cause" referred to the fact that a given cancer develops in proportion to the exposure to a given agent, and that "establishing tobacco as a cause of lung cancer does not deny the added significance of other factors." The paper also delineated 10 points that established tobacco as a cause of lung cancer. The dose-response relationship of tobacco smoke exposure to cancer incidence, which was evident in human studies and in laboratory work with animals, carried a major clue for preventive strategies that are discussed subsequently.

Yet, most of organized medicine and governmental agencies remained silent. At the National Cancer Institute (NCI), however, we obtained early support from its Director Dr. John Heller and from Mr. Cornfield. In fact, it has always been most satisfying that the leadship of the NCI, Dr. Heller, Dr. Kenneth Endicott, Dr. Carl Baker, Dr. Frank Rauscher, Dr. Arthur Upton, and Dr.

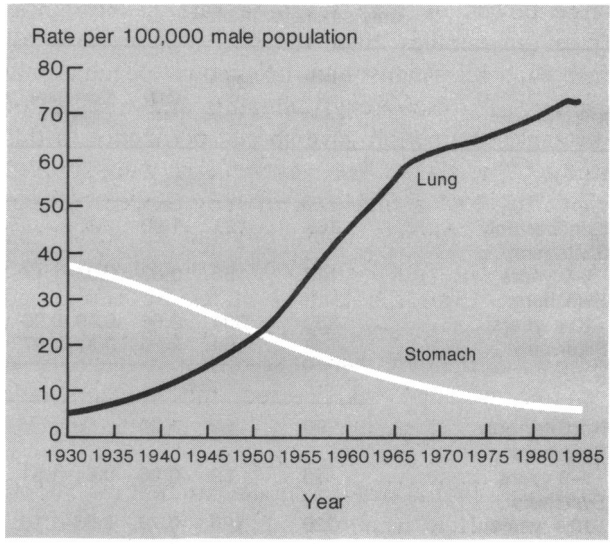
Vincent DeVita, have been most supportive of research related to the tobacco and cancer issue and of prevention in general.

In 1957, a study group (*44*) appointed by NCI, the National Heart, Lung, and Blood Institute, the American Cancer Society, and the American Heart Association examined the scientific evidence on the effects of tobacco smoke on health and arrived at the following conclusion: "The sum total of scientific evidence establishes beyond reasonable doubt that cigarette smoking is a causative factor in the rapidly increasing incidence of human epidermoid carcinoma of the lung." A similar conclusion was drawn by the Medical Research Council in Great Britain in the same year (*45*). Thus, by 1957 there appeared to be increasing consensus on the causative relationship between cigarette smoking and lung cancer. Strong scientific and political support came in 1962 with the Royal College of Physicians' report on smoking and lung cancer and particularly in 1964 with the first appearance of the reports on smoking and health from the Surgeon General (*46, 47*).

In retrospect, it is difficult to comprehend why it took health professionals and society so long to grasp the full extent of the causative association between lung cancer and smoking. As late as 1961, in a debate on this issue with Clarence Cook Little, Director of the Tobacco Research Council, we received little outside support. The New England Journal of Medicine, which published the debate, sided with our views on causation in an editorial entitled "The Great Debate," (*48-50*) but failed to be definitive in its conclusion.

Reflecting on the events of the 1950s and 1960s and the slow support received for our work on smoking and health, we ponder the reasons. The position of the tobacco industry is understandable as is its influence on groups depending on its financial support, such as the media, and even governments. But, it has been difficult to comprehend the benign neglect by the medical professions. Is it because physicians principally think of themselves as healers? Is it because only in therapy do they see academic and economic rewards? Is it that scientists are so concerned with fundamental research that they do not consider how findings can lead to preventive measures—measures that often can be effective without a finite understanding of all the basic mechanisms of causation? Is it because the departments of preventive medicine have always played a subordinate role in the activities of medical schools and universities? Or is it that the consumer who demands treatment when

Figure 1. Age-adjusted cancer death rates for lung and stomach, males, United States, 1930–84



SOURCE: Reference *51*.

disabled by disease does not with equal vigor demand preventive practice, particularly when lifestyle variables such as smoking are involved?

Clearly, application is more difficult than discovery. It continues to be so, except among certain segments of our society such as the upper income class and highly educated groups, who are more aware of the consequences of smoking and among whom cigarette smoking has become socially less acceptable. Although we have been aware of the primary cause of lung cancer for many years, death rates have continued to increase sharply. By contrast, those for stomach cancer have declined steadily, for reasons that are not entirely clear but undoubtedly related to diet (*51,52*) (fig. 1).

Where, then, do we stand in 1988 in applying the lessons from our knowledge of tobacco use as a major cause of excessive and unnecessary disease and disability in our society? In which direction should and can the "science of application" take us?

Application

Medical research strives for two endpoints: to cure disease and to prevent disease. Unless we accomplish one or the other, our efforts, important as they may be, represent only a prologue. In many instances, current knowledge calls for prevention through the application of known discoveries. To be successful, the science of application requires the best minds and adequate funding to achieve the place in the scientific hierarchy it

Table 1. Adjusted[1] odds ratios and 95 percent confidence intervals for lung cancer among filter only smokers and short-term and long-term switchers compared with nonfilter only smokers, male

Kind of smokers	Number of cases	Number of controls	Odds ratio	Confidence intervals
		Sample I		
Nonfilter only	165	121	1.00	...
Switchers: 1–9 years	120	122	0.83	0.59–1.17
Switchers: 10+ years	330	304	0.66	0.49–0.90
Filter only	36	54	0.69	0.37–1.27
		Sample II		
Nonfilter only	90	66	1.00	...
Switchers: 1–9 years	83	59	0.96	0.61–1.51
Switchers: 10+ years	226	195	0.79	0.53–1.18
Filter only	35	38	0.87	0.43–1.54

[1] Adjusted for number of cigarettes smoked per day, age, inhalation, and years of education.
SOURCE: Reference *61*.

Table 2. Percentage of high school students reporting daily use of cigarettes in the previous 30 days, 1975–84

Class	Total	Males	Females
1975	26.9	26.9	26.4
1976	28.8	28.0	28.8
1977	28.8	27.1	30.0
1978	27.5	26.0	28.3
1979	25.4	22.3	27.8
1980	21.3	18.5	23.5
1981	20.3	18.1	21.7
1982	21.1	18.2	23.2
1983	21.2	19.2	22.2
1984	18.7	16.0	20.5

SOURCE: Reference *64*.

deserves. Certainly, application of the discoveries made in the area of tobacco and health has been very difficult.

Initially, few organized attempts were made to deal with the smoking issue from the standpoint of public health, even though many investigators, particularly epidemiologists, had demonstrated time and time again the causative relationship between tobacco use and a variety of cancers and other diseases. The American Cancer Society and other voluntary health organizations were early in the forefront of tackling this issue. They were subsequently supported by large-scale activities at NIH, particularly NCI, and a succession of reports from the Surgeon General, culminating in the forceful intervention of Surgeon General C. Everett Koop.

Where should our policy be directed? The three broad areas that deal with the reduction of tobacco-related diseases in increasing order of importance are the low-yield cigarette, cessation of tobacco use, and prevention of the onset of tobacco habits.

Low-yield cigarette. There has been a good deal of controversy about whether the independent scientific community should give any attention to the low-yield cigarette—a cigarette yielding significantly less tar and nicotine in its mainstream smoke than the nonfilter cigarettes of the 1950s. It is properly argued that there can be no safe cigarette and that support of a so-called "less harmful" cigarette may cause more people to smoke. But, as long as society endorses the legality of smoking, many young people will develop a tobacco habit, and many people who are already smokers will continue to smoke. It seems that, especially when young, we suffer from an illusion of immortality, as we discussed in a thought-provoking symposium (*53*), which gives us the feeling that we are impervious to harm. As a matter of practicality, then, we must recognize that tobacco use will continue to some extent. Thus, continued reduction of the tar yield of cigarettes is a goal that should be pursued.

The tar content of American cigarettes has significantly decreased during the last few decades. However, sales-weighted average yields of nicotine (an industrial comparison standard which averages nicotine levels from total number of American cigarettes sold in the United States and presumably smoked) dropped initially but have remained fairly stable during the last 5 years. Some tumorigenic agents in cigarette smoke have been selectively reduced, as shown in a large series of studies by Hoffmann and his collaborators (*54*) (fig. 2). In turn, the carcinogenic potential of cigarette tar in terms of its tumorigenic activity on mouse skin has been reduced from its level in 1950 (*55,56*). More pertinently, the risk of lung cancer as well as bladder cancer has been reduced among persons who smoke only filter cigarettes, and in the case of lung cancer, among persons who have switched to filter cigarettes for more than 10 years (table 1) (*57–59*). Thus, reducing the tar content and the amount of tobacco in cigarettes has led to a reduced risk for certain types of cancer, although such reduction has not been shown for myocardial

infarction (*60*) where nicotine and carbon monoxide are likely to play a predominant role.

As far as future development is concerned, the average tar yields of cigarettes should not exceed 10 milligrams, with a tar to nicotine ratio of 10. To the extent possible, smokers must avoid compensating for lower nicotine yields by taking deeper and more frequent puffs or by smoking more cigarettes per day. The low yield and modified cigarette has been an advantage over the products smoked in the earlier decades up to the 1950s. However, because toxins and tumorigenic components in the smoke remain at levels that are harmful, we must emphasize the need for abstinence.

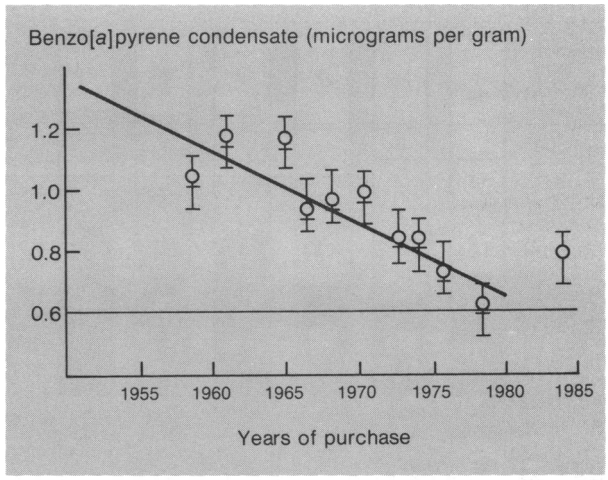
Cessation. A significant reduction in cigarette smoking has occurred in our population, principally among educated white males. This reduction is greater in the United States than in other industrialized societies, because of (*a*) public campaigns about the danger of smoking, (*b*) warning labels on cigarette packages and advertisements, and (*c*) an increased realization that cigarette smoking is no longer a socially acceptable habit. The prevalence of quitting (fig. 3) is dependent on education among men, though not among women (*61*). While this statistic is satisfying to a degree, it is important to recognize that a high prevalence of smoking continues among blue collar workers (*62*) (fig. 4) and among minority groups, particularly blacks (fig. 5). Women, particularly young women, appear to be less inclined to quit smoking, partially because of the fear of weight gain (fig. 6).

Table 2 indicates that while there has been some reduction in smoking among adolescent males, less has been achieved among adolescent females (*63,64*). High school dropouts smoke significantly more than students who complete high school. These findings suggest that tobacco-related diseases will decline first among educated white males and less among other groups of men. Among women, tobacco-related diseases will continue to increase.

In addition to general public education efforts, specific smoking cessation programs need to be implemented. The 1-year success rate in such programs averages about 25 percent, quite a good result for persons who cannot give up smoking by themselves. The great majority of quitters, however, have done so cold turkey (*65*).

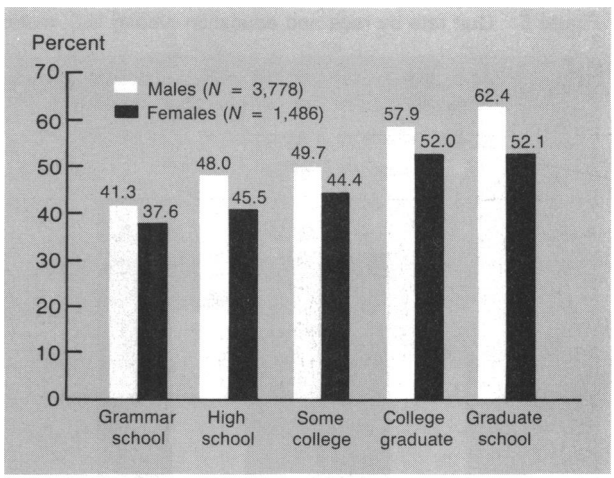
That so many people have quit on their own demonstrates that it can be done without any outside help; yet for those who are particularly habituated, outside help is necessary and needs to

Figure 2. Benzo[a]pyrene levels in the smoke of a best selling U.S. nonfilter cigarette (85 millimeters) monitored from 1958 to 1984



SOURCE: D. Hoffmann, et al., American Health Foundation, 1987.

Figure 3. Quit rate among smokers, males and females, by education



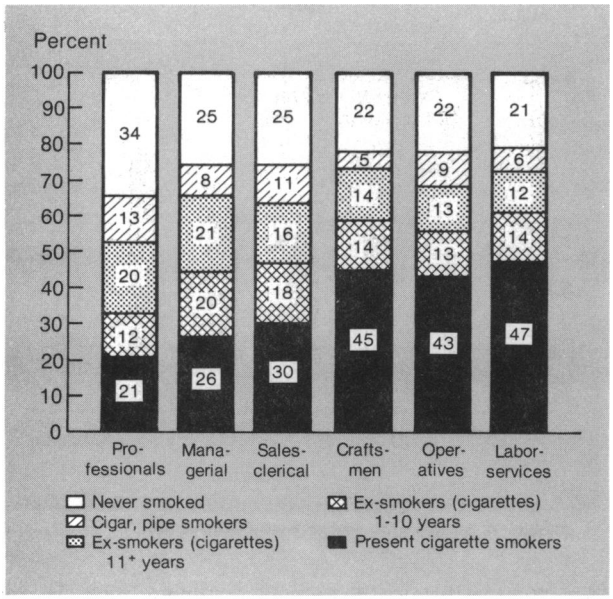
Note: Quit rate = $\frac{\text{number of ex-smokers}}{\text{number of ever-smokers}} \times 100$

SOURCE: Reference *61*.

be provided by health professionals. The cost of such programs should be reimbursed by health insurers. Hospitals should provide smoking cessation services. When one of our interviewers posing as a heavy smoker recently telephoned 28 hospitals in New York City, 24 said they could not help, 3 referred her to a local smoking cessation program conducted by the American Cancer Society, and 1 recommended a hypnotist. At a time when our hospitals have all kinds of medical specialty clinics, they should be willing to help heavy smokers who cannot stop smoking on their own.

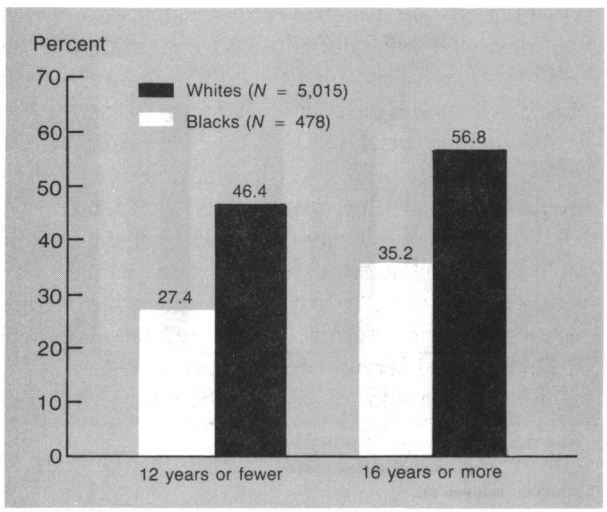
The worksite is also a good place to provide

Figure 4. Age-standardized smoking rates by occupational level, white male controls ages 41–70 years



SOURCE: Reference *62*.

Figure 5. Quit rate by race and education among U.S. males



NOTE: Quit rate = $\frac{\text{number of ex-smokers}}{\text{number of ever-smokers}} \times 100$

SOURCE: Reference *61*.

smoking cessation programs. Smoking cessation programs should be targeted in our school systems to students who smoke. Clearly, the earlier we can provide smoking cessation messages, the easier it should be to break the habit. Physicians can also make an important, cost-effective contribution to smoke cessation by providing a strong antismoking message to their patients who smoke.

Health behaviorists should provide services on the basis of existing knowledge as well as conducting research. Their work in smoking cessation should be conducted in hospitals, at worksites, and in schools. Thus, they should "practice" in the field as well as study the aspects of smoking cessation.

Prevention. The ideal approach for eliminating smoking in our society is through age-appropriate education of children. Tobacco use is an acquired habit that if not initiated early in life does not come "naturally." A smoking prevention program should be introduced in the first grade of the school curriculum, if not earlier. It should be an integral part of a health behavior program that encompasses all risk-taking behavior such as the use of illicit drugs and alcohol abuse. It should also teach sexual hygiene, as well as the benefits of good nutrition and physical exercise. The program should relate to the children's self-perception and self-esteem rather than merely provide knowledge, so that ultimately the children can make appropriate health decisions based on a value system they have established for themselves.

In line with these considerations, we have developed over the last 10 years the Know Your Body School Health Education Program (KYB). This program includes an annual health screening; the results are entered in a Health Passport, which personalizes the screening. Workbooks for the children and guides for the teachers are essential components of this program, which is delivered during 30 hours of the school year. Behavioral strategies include modification of the school cafeteria menus and conclude with a yearly testing for attitude, knowledge, behavior, and clinical indicators.

Dr. Heather Walter followed a single class for 5 years. KYB demonstrated reductions in the onset of smoking and in fat intake and serum cholesterol levels, and a general improvement in health knowledge and behavior (unpublished data, American Health Foundation, 1987). Smoking prevalence, verified by nicotine to cotinine measurements, was 7 percent in the control group and 4 percent in the group that had received KYB instruction from the 4th through 10th grades ($P = 0.05$). Another study among first and second graders showed a 50 percent improvement in intention to refrain from eating potato chips, ice cream, or chocolate cookies, which are particularly good indicators for the use of snack foods among children (*66*). We are currently promoting KYB in a number of cities in the United States as well as in China, Germany, Italy, and Israel.

If our children are to grow up with good health

behavior, including abstinence from smoking, we must have mandated school health education programs that include periodic health screening; an all-inclusive curriculum; training for health education teachers; yearly assessment in terms of attitude, behavior, and knowledge; and course grades based on knowledge and writing of a health education-related essay. Funding for such efforts should not exceed 1 percent of the total school budget. Not only will mandated school health education of the type described improve the overall health behavior of our children but also it will improve self-esteem, which, in turn, could lead to a lower dropout rate and greater learning capacity. At a time when we are consumed by the fear that our children will succumb to the use of drugs or contract AIDS, we must regard health and behavioral education in our school systems as one of the great challenges. Children have the right to expect leadership in this regard from an adult society that cares for the future of its children.

Cancer Prevention Units

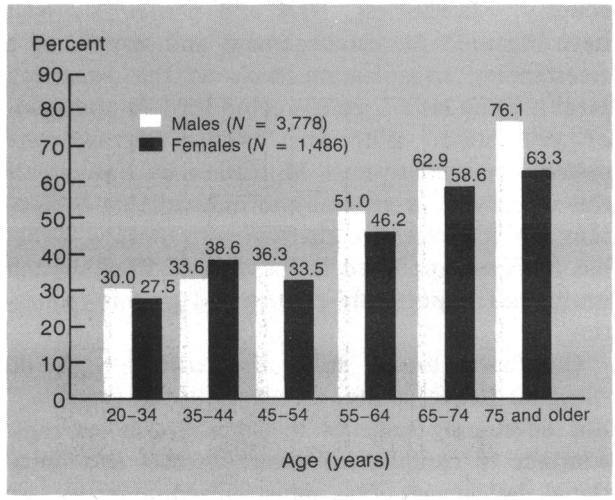
Since the use of tobacco is accepted as a causative factor in a variety of cancers and other diseases and is, in fact, the single-most preventable cause of death in industrialized societies, the question remains: What can we do further to reduce tobacco use? We have indicated our strategies as they relate to low-yield tobacco products, smoking cessation, and smoking prevention.

What is required is not just more research but rather greater application of existing knowledge. The Public Health Service needs to stimulate the scientific community to apply existing knowledge on smoking cessation and prevention, particularly for those smokers who cannot quit on their own.

Toward this end, we recommend the establishment of cancer prevention units (CPUs), either as part of an existing community health care establishment, or independently to practice cancer and disease prevention. We suggest that the targets of the CPUs be schools, worksites, hospitals, and communities and that the units be staffed with a director, a health promotion strategist, a behavioral scientist, a health educator, a smoking cessation facilitator, a nutritionist, an occupational hygienist, and an economist.

The major aim of the unit is to "practice" cancer prevention. We recommend that the NCI fund a limited number of such demonstration units for a 5-year period at the end of which the unit should be self-supporting. Just as the therapist

Figure 6. Quit rate by age, males and females



NOTE: Quit rate = $\frac{\text{number of ex-smokers}}{\text{number of ever-smokers}} \times 100$

SOURCE: Reference *61*.

serves the public in terms of curing disease, the CPU serves in terms of preventing disease, and just as the public pays for curative services, so too should it pay for preventive services. Unless society is willing to pay for prevention programs, preventive medicine will not flourish. In addition, the CPU can research how to effect changes in the public's lifestyle practices. Services to be offered would include smoking cessation clinics on site, at the workplace, and in the community. Similar programs in nutrition would also be available. The CPU would counsel school officials on the conduct of health education programs and would work with public health officials and corporate medical personnel to make preventive services available throughout the service area.

The CPU should establish associations with local hospitals, the Academy of Family Physicians, industrial physicians, and with other groups of health providers by establishing linkages with HMOs and insurance industries. The CPU would become a provider of services, a project resource, and a catalyst for the dissemination of prevention-oriented programs.

Epilogue

Throughout the history of medicine, the healing physician, both for academic and economic reasons, has been in the forefront of public and scientific recognition. If anything, the "magic bullet" philosophy of medicine has intensified in proportion to the sophistication of our equipment and skills. Preventive medicine has decreased in

stature in medical schools throughout much of the country. Nevertheless, NIH and particularly NCI have nurtured the establishment and growth of a freestanding organization such as the American Health Foundation, solely committed to the goals of prevention. With the existing disease care system expected to cost $1 trillion by the end of this century, it is generally recognized that prevention is a cost-effective alternative to therapy. What has been accomplished in the area of smoking and health has importantly contributed to this realization.

Our basic motto holds that medicine should "help us to die young, as late in life as possible." For developing societies this means that we must continue to conquer infectious diseases and nutritional deficiencies. For industrialized countries, we must concentrate on overcoming disorders relating to unhealthy lifestyles largely in respect to what we eat, smoke, and drink. We understand what has to be done to reduce the incidence of these disorders. The issue is whether we will make the commitment to do it. The risk of failing to proceed is great. The reward of being willing to proceed is even greater, for instance, the decline in mortality attributable to lung cancer that we are beginning to see among males in the United States.

These are the lessons learned from a lifetime of studying the epidemiology of cancer, particularly as it relates to tobacco use:

1. Epidemiology has established that cancer is not an inevitable consequence of life or aging.
2. We can always learn from history, be it in politics or in science.
3. A major obstacle of preventive medicine is human nature.
4. Epidemiology provides pertinent clues to the experimentalist.
5. Metabolic or biochemical and molecular epidemiology are vital components of epidemiology.
6. The science of application must attract our best and brightest talents because it holds the key to preventing disease long before the disease mechanisms are fully understood.
7. Interdisciplinary approaches to scientific research result in accelerated scientific progress.
8. Science, unlike many other aspects of life, follows exacting laws whose unraveling presents the scientist with frustration and challenges.
9. Active opposition and biases in time give way to scientific truth.
10. The pursuit of preventing avoidable illness with the cooperation, counsel, and friendship of colleagues is one of life's great pleasures. The most powerful force for successfully preventing behaviorally induced diseases involves the social support we receive from our families, friends, coworkers, and society as a whole. The self-esteem and the feeling of self-worth we receive from such support is no doubt the most important force to prevent lifestyle-induced diseases and premature death.

What we as behavioral scientists, as physicians, as citizens, do in this respect largely determines whether many of these diseases from which we suffer today will occur when our children reach adulthood. This view represents a challenge to all of us. The smoking and health issue exemplifies what must and can be done with our involvement.

The ultimate aim of medicine and science must be the prevention of disease. The lesson of tobacco carcinogenesis has shown that this goal is attainable for a large portion of human cancers. Simply said, the science of preventive medicine needs only to be practiced to succeed.

References

1. Soemmering, S. T.: De morbis vasorum absorbentium corporis humanai. Varrentrapp & Wenner, Frankfurt, Germany, 1795.
2. Abbe, R.: Cancer of the mouth. The case against tobacco. N Y Med J 102: 1-2 (1915).
3. Adler, I.: Primary malignant growths of the lung and bronchi. Longmans, Green and Co., Ltd., London, 1912.
4. Tylecote, F. E.: Cancer of the lung. Lancet 2: 256-257 (1927).
5. Lombard, H. L., and Doering, C. R.: Cancer studies in Massachusetts. 2. Habits, characteristics and environment of individuals with and without cancer. New Engl J Med 198: 481-487, Apr. 26, 1928.
6. Hoffmann, F. L.: Cancer of the lung. Am Rev Tuberc Soc 19: 392-406 (1929).
7. Lickint, F.: Tabak und Tabakrauch als Etiologischer Faktor des Carcinoms. Z Kresbforsch 30: 349-365 (1929).
8. McNally, W. D.: The tar in cigarette smoke audits possible effects. Am J Cancer 16: 1506-1514 (1932).
9. Arkin, A., and Wagner, D. H.: Primary carcinoma of the lung. JAMA 106: 587-591, Feb. 22, 1936.
10. Ochsner, A., and DeBakey, M.: Symposium on cancer. Primary pulmonary malignancy. Treatment by total pneumonectomy; analyses of 79 collected cases and presentation of 7 personal cases. Surg Gyn Obst 68: 435-451 (1939).
11. Mueller, F. H.: Tabakmissbrauch und Lungencarcinom. Z Krebsforsch 49: 57-84 (1939).
12. Ochsner, A., and DeBakey, M.: Carcinoma of the lung. Arch Surg 42: 209-258 (1941).
13. Schairer, E., and Schoeniger, E.: Lungenkrebs und Tabakverbrauch. Z Krebsforsch 54: 261-269 (1943).

14. Kennaway, N. M., and Kennaway, E. L.: A study of the incidence of cancer of the lung and larynx. J Hyg 36: 236-267 (1936).
15. Kennaway, E. L., and Kennaway, N. M.: A further study of the incidence of cancer of the lung and larynx. Brit J Cancer 1: 260-298, Sept. 30, 1947.
16. American Cancer Society. Statistics on cancer. Statistical Research Division, ACS, New York, 1949.
17. Cook, J. W., Hewett, C. L., and Hieger, I.: The isolation of cancer-producing hydrocarbons from coal tar. Pts 1, 2, and 3. Article No. 136. J Chem Soc (London): 395-405 (1933).
18. Roffo, A. H.: Krebseizeugendes Benzpyren gewonnen aus Tabakteer. Z Krebsforsch 49: 588-597 (1939).
19. Wynder, E. L., and Hoffmann, D.: Tobacco and tobacco smoke. Studies in experimental tobacco carcinogenesis. Academic Press, New York, 1967.
20. Wynder, E. L., and Graham, E. A.: Tobacco smoking as a possible etiologic factor in bronchiogenic carcinoma. A study of six hundred and eighty-four proved cases. JAMA 143: 329-336, May 27, 1950. [Republished as a landmark article, JAMA 253: 2986-2994, May 24/31, 1985.]
21. Doll, R., and Hill, A. B.: Smoking and carcinoma of the lung. Preliminary report. Brit Med J 2: 739-748, Sept. 30, 1950.
22. Wynder, E. L., Bross, I. J., and Day, E.: A study of environmental factors in cancer of the larynx. Cancer 9: 86-110 (1956).
23. Wynder, E. L., Bross, I. J., and Day, E.: Epidemiological approach to the etiology of cancer of the larynx. JAMA 160: 1384-1391, Apr. 21, 1956.
24. Wynder, E. L., and Bross, I. J.: Aetiological factors in mouth cancer: an approach to its prevention. Brit Med J I: 1137-1143, May 18, 1957.
25. Wynder, E. L., and Bross, I. J.: A study of etiological factors in cancer of the esophagus. Cancer 14: 389-413 (1961).
26. Wynder, E. L., Onderdonk, J., and Mantel, N.: An epidemiological investigation of cancer of the bladder. Cancer 16: 1388-1407 (1963).
27. Wynder, E. L., Mabuchi, K., Maruchi, N., and Fortner, J. G.: Epidemiology of cancer of the pancreas. J Natl Cancer Inst 50: 645-667 (1973).
28. Wynder, E. L., Mabuchi, K., and Whitmore, W. F.: Epidemiology of adenocarcinoma of the kidney. J. Natl Cancer Inst 53: 1619-1634 (1974).
29. Wynder, E. L., Bross, I. J., Cornfield, J., and O'Donnell, W. E.: Lung cancer in women: a study of environmental factors. New Engl J Med 255: 1111-1121, Dec. 13, 1956.
30. Wynder, E. L., and Cornfield, J.: Cancer of the lung in physicians. New Engl J Med 248: 441-444, Mar. 12, 1953.
31. Doll, R., and Hill, A. B.: A study of the aetiology of carcinoma of the lung. Brit Med J 2: 1272-1286, Dec. 13, 1952.
32. Doll, R., and Hill, A. B.: The mortality of doctors in relation to their smoking habit; a preliminary report. Brit Med J 1: 1451-1455, June 26, 1954.
33. Doll, R., and Hill, A. B.: Lung cancer and other causes of death in relation to smoking. A second report on the mortality of British doctors. Brit Med J 2: 1071-1081, Nov. 10, 1956.
34. Best, E. W., et al.: A Canadian study of mortality in relation to smoking habits. A preliminary report. Can J Public Health 52: 99-106 (1961).
35. Hammond, E. C., and Horn, D.: Smoking and death rates—report on fifty-four months of follow-up on 187,783 men. JAMA 166: 1159-1172, Mar. 8, 1958.
36. Dorn, H. F.: The mortality of smokers and nonsmokers. *In* Proceedings of the social statistic section of the American Statistical Association, annual meeting. American Statistical Association, Washington, DC, 1959, pp. 34-71.
37. Wynder, E. L., Graham, E. A., and Croninger, A. B.: Experimental production of carcinoma with cigarette tar. Cancer Res 13: 855-864 (1953).
38. Graham, E. A., Croninger, A. B., and Wynder, E. L.: Experimental production of carcinoma with cigarette tar. IV. Successful experiments with rabbits. Cancer Res 17: 1058-1066 (1957).
39. Wynder, E. L., and Wright, G.: A study of tobacco carcinogenesis. I. The primary fractions. Cancer 10: 255-271 (1957).
40. Hoffmann, D., and Hecht, S. S.: Perspectives in cancer research. Cancer Res 45: 935-944 (1985).
41. Hecht, S. S., et al.: Effects of methyl and fluorene substitution on the metabolic activation and tumorigenicity of polycyclic aromatic hydrocarbons. Am Chem Soc Symp Ser 238: 85-105 (1985).
42. Hoffmann, D., and Harris, C. C., editors: Mechanisms in tobacco carcinogenesis. Banbury Report 23. Cold Spring Harbor Laboratories, Cold Spring Harbor, NY, 1986.
43. Wynder, E. L.: Tobacco as a cause of lung cancer. With special reference to the infrequency of lung cancer among nonsmokers. Pa Med J 57: 1073-1083 (1954).
44. Smoking and health. Joint report of the Study Group on Smoking and Health. Science 125: 1129-1133, June 7, 1957.
45. Medical Research Council: Tobacco smoking and cancer of the lung. Brit Med J 1: 1523-1524, June 29, 1957.
46. Royal College of Physicians: Smoking and health. Pitman Medical, London, 1962.
47. U.S. Public Health Service: Smoking and health. Report of the Advisory Committee to the Surgeon General of the U.S. Public Health Service. PHS Publication No. 1103. U.S. Government Printing Office, Washington, DC, 1964.
48. Wynder, E. L.: An appraisal of the smoking-lung cancer issue. New Engl J Med 264: 1235-1240, June 15, 1961.
49. Little, C. C.: Some phases of the problem of smoking and lung cancer. New Engl J Med 264: 1241-1245, June 15, 1961.
50. The great debate [editorial]. New Engl J Med 264: 1234, June 15, 1961.
51. Silberberg, E., and Lubera, J.: Cancer statistics. CA 37: 2-19 (1987).
52. Howson, C. P., Hiyama, T., and Wynder, E. L.: The decline in gastric cancer: epidemiology of an unplanned triumph. Epidemiol Rev 8: 1-27 (1986).
53. Symposium: The illusion of immortality. Prev Med 5: 477-548 (1976).
54. Hoffmann, D., et al.: Chemical carcinogens in tobacco. *In* Cancer risks—strategies for elimination, edited by P. Bannasch. Springer-Verlag, New York, 1987, pp. 101-113.
55. Contenwill, U., et al: Experimental investigations on the tumorigenic activity of cigarette smoke condensate on mouse skin. VII. Comparative studies of condensates from different modified cigarettes (German). Z Krebsforsch Klin Oncol 89: 145-151 (1977).
56. Hoffmann, D., Schmeltz, I., Hecht, S. S., and Wynder, E. L.: Tobacco carcinogenesis. *In* Polyclyclic hydrocar-

bons and cancer, edited by H. V. Gelboin and P. O. Tso. Academic Press, New York, 1978, pp. 85-117.
57. Wynder, E. L. and Stellman, S. D.: Comparative epidemiology of tobacco-related cancers. Cancer Res 37: 4608-4622 (1977).
58. Hammond, E. C., Garfinkel, L., Seidman, H., and Lew, E. A.: Some recent findings concerning cigarette smoking. *In* Origins of human cancer, Book A. Incidence of cancer in humans, edited by H. H. Hiatt, J. D. Watson, and J. A. Winsten. Cold Spring Harbor Laboratories, Cold Spring Harbor, NY, 1977, pp. 101-112.
59. Kabat, G. C., Dieck, G. S., and Wynder, E. L.: Bladder cancer in nonsmokers. Cancer 57: 362-367 (1986).
60. Kaufman, D. W., et al.: Nicotine and carbon monoxide content of cigarette smoke and the risk of myocardial infarction in young men. N Engl J Med 308: 409-413, Feb. 24, 1983.
61. Kabat, G. C., and Wynder, E. L.: Determinants of quitting smoking. Am J Public Health 77: 1301-1305, October 1987.
62. Covey, L. S., and Wynder, E. L.: Smoking habits and occupational status. J Occup Med 23: 537-542 (1981).
63. National Institute on Drug Abuse: Student drug use in America, 1975-1981. University of Michigan Publications Division, Institute for Social Research, Ann Arbor, MI, 1982.
64. Koop, C. E.: The quest for a smoke-free young America by the year 2000. J Sch Health 56: 8-12 (1986).
65. Carmody, T. P., et al.: A prospective five-year follow-up of smokers who quit on their own. Health Educ Res Theory Pract 1: 101-109 (1986).
66. Krone, K., and Lieberman, L.: The Primary Grades KYB Health Modules Curriculum Study. American Health Foundation Report to the Kellogg Foundation, December 1986.

Population Characteristics and Health Care Needs of Asian Pacific Americans

JANE S. LIN-FU, MD

Dr. Lin-Fu is Acting Chief, Genetic Services Branch, Bureau of Maternal and Child Health and Resources Development, Health Resources and Services Administration, Rm. 6-17, 5600 Fishers Lane, Rockville, MD 20857. Tearsheet requests to Dr. Lin-Fu.

Synopsis

Asian Pacific Americans are one of the smallest but fastest growing minority groups in the United States. Between 1970 and 1980, this population increased 142 percent, from 1.5 million to 3.7 million. This dramatic growth is due largely to a change in U.S. immigration policies in the mid-1960s and the continuous influx of refugees from Southeast Asia since 1975. Despite such sharp increase, Asian Pacific Americans remain one of the most poorly understood minorities, and their health care needs have received relatively little attention. Health policy makers, planners, and service providers need to have a better understanding of the population characteristics of Asian Pacific Americans in order to address their needs properly.

Asian Pacific Americans are largely recent immigrants and refugees. They are extremely heterogeneous and bipolar in socioeconomic status and health indices. Because of their small numbers until the last two decades, many health workers have had little exposure to this minority, their culture, and health problems. Health workers need to be sensitive to the ethnocultural barriers that confront recent arrivals; be aware of the genetic disorders, infectious diseases, and mental health problems common in this population; and realize that anatomical and physiological differences may require attention in certain surgical procedures and medical management. Neglecting the health care needs of Asian Pacific Americans is not simply a violation of the principle of equality for all, but also an imprudent act that increases the mortalities and morbidities and health care costs of the nation.

ASIAN PACIFIC AMERICANS represent one of the smallest but fastest growing minority groups in the United States. In the decade between the 1970 and 1980 censuses, this population increased 142 percent, from 1,538,721 to 3,726,440 persons. This dramatic gain compares with an increase of 11