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Tobacco Use—United States, 1900-1999

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4 figures, 1 photo omitted

SMOKING—ONCE A SOCIALLY ACCEPTED behavior—is the leading preventable cause of death and disability in the United States. During the first decades of the 20th century, lung cancer was rare; however, as cigarette smoking became increasingly popular, first among men and later among women, the incidence of lung cancer became epidemic. In 1930, the lung cancer death rate for men was 4.9 per 100,000; in 1990, the rate had increased to 75.6 per 100,000.[1] Other diseases and conditions now known to be caused by tobacco use include heart disease, atherosclerotic peripheral vascular disease, laryngeal cancer, oral cancer, esophageal cancer, chronic obstructive pulmonary disease, intrauterine growth retardation, and low birthweight. During the latter part of the 20th century, the adverse health effects from exposure to environmental tobacco smoke also were documented. These include lung cancer, asthma, respiratory infections, and decreased pulmonary function.[2]

Large epidemiologic studies conducted by Ernst Wynder (see box) and others in the 1940s and 1950s linked cigarette smoking and lung cancer. In 1964, on the basis of approximately 7000 articles relating to smoking and disease, the Advisory Committee to the U.S. Surgeon General concluded that cigarette smoking is a cause of lung and laryngeal cancer in men, a probable cause of lung cancer in women, and the most important cause of chronic bronchitis in both sexes.[3] The committee stated that "Cigarette smoking is a health hazard of sufficient importance in the United States to warrant appropriate remedial action." Substantial public health efforts to reduce the prevalence of tobacco use began shortly after the risk was described in 1964. With the subsequent decline in smoking, the incidence of smoking-related cancers (including cancers of the lung, oral cavity, and pharynx) have also declined (with the exception of lung cancer among women).[4] In addition, age-adjusted death rates per 100,000 persons (standardized to the 1940 population) for heart disease (i.e., coronary heart disease) have decreased from 307.4 in 1950 to 134.6 in 1996.[4] During 1964-1992, approximately 1.6 million deaths caused by smoking were prevented.[5]

Smoking Trends During the Century

Early in the 20th century, several events coincided that contributed to increases in annual per capita consumption, including the introduction of blends and curing processes that allowed the inhalation of tobacco, the invention of the safety match, improvements in mass production, transportation that permitted widespread distribution of cigarettes, and use of mass media advertising to promote cigarettes.[6,7] Cigarette smoking among women began to increase in the 1920s when targeted industry marketing and social changes reflecting the liberalization of women's roles and behavior led to the increasing acceptability of smoking among women.[8,9] Annual per capita cigarette consumption increased from 54 cigarettes in 1900 to 4345 cigarettes in 1963 and then decreased to 2261 in 1998.[10,11] Some decreases correlate with events, such as the first research suggesting a link between smoking and cancer in the 1950s, the 1964 Surgeon General's report, the 1968 Fairness Doctrine, and increased tobacco taxation and industry price increases during the 1980s.

An important accomplishment of the second half of the 20th century has been the reduction of smoking prevalence among persons aged ≥18 years from 42.4% in 1965 to 24.7% in 1997, with the rate for men (27.6%) higher than for women (22.1%). The percentage of adults who never smoked increased from 44% in the mid-1960s to 55% in 1997. In 1998, tobacco use varied within and among racial/ethnic groups. The prevalence of smoking was highest among American Indians/Alaska Natives, and second highest among black and Southeast Asian men. The prevalence was lowest among Asian American and Hispanic women.[12] Smokeless tobacco use has changed little since 1970, with a 5% prevalence in 1970 and a 6% prevalence in 1991 among men, and 2% and 1%, respectively, for women. The prevalence of smokeless tobacco use is highest among high school males, with prevalence being 20% among white males, 6% among Hispanics males, and 4% among black males. Prevalence of use tends to be lower in the northeastern region and higher in the southern region of the United States. Total consumption of cigars decreased from 8 million in 1970 to 2 million in 1993 but increased 68% to 3.6 million in 1997.[13]

Reductions in smoking result from many factors, including scientific evidence of the relation among disease, tobacco use, and environmental exposure to tobacco; dissemination of this information to the public; surveillance and evaluation of prevention and cessation programs; campaigns by advocates for nonsmokers' rights; restrictions on cigarette advertising; counteradvertising; policy changes (i.e., enforcement of minors' access laws, legislation restricting smoking in public places, and increased taxation); improvements in treatment and prevention programs; and an increased understanding of the economic costs of tobacco.

The cigarette itself has changed. When cigarettes were first associated with lung cancer in the early 1950s, most U.S. smokers smoked unfiltered

cigarettes. With a growing awareness of the danger of smoking came the first filter, which was designed to reduce the tar inhaled in the smoke. Later, low tar cigarettes were marketed; however, many smokers compensated by smoking more intensely and by blocking the filter's ventilation holes.[13] Adenocarcinoma has replaced squamous cell carcinoma as the leading cause of lung cancer-related death in the United States. This increase in adenocarcinoma parallels the changes in cigarette design and smoking behavior.[13]

Changes in the social norms surrounding smoking can be documented by examining changes in public policy, including availability of Fairness Doctrine counteradvertising messages on television and radio and increased restrictions on tobacco advertising beginning with the ban on broadcast advertising in 1971. Cigarette advertising no longer appears on television or billboards, and efforts to restrict sales and marketing to adolescents have increased. Indoor air policies switched from favoring smokers to favoring nonsmokers. Smoking is no longer permitted on airplanes, and many people, including 12.5% of adult smokers with children, do not smoke at home.[14] Now 42 states have restrictions on smoking at government work sites and 20 states have restrictions at private work sites.

One of the most effective means of reducing the prevalence of tobacco use is by increasing federal and state excise tax rates. A 10% increase in the price of cigarettes can lead to a 4% reduction in the demand for cigarettes. This reduction is the result of people smoking fewer cigarettes or quitting altogether.[15] Studies show that low-income, adolescent, Hispanic, and non-Hispanic black smokers are more likely than others to stop smoking in response to a price increase.[17]

The November 1998 Master Settlement Agreement marks the end of the 20th century with an unprecedented event. Although admitting no wrongdoing, the tobacco companies signed an agreement with the attorneys general of 46 states. This agreement settled lawsuits totaling $206 billion; however, the agreement did not require that any of the state money be spent for tobacco use prevention and control. The American Legacy Foundation was established as a result of a provision in the Master Settlement Agreement that called for a foundation with a mandate to conduct effective tobacco education programs based on scientific research.

Ernst L. Wynder, MD

Although cigarettes were considered a symbol of popularity and social acceptability from the opening of the 20th century, critics warned of the dangers of what they called "coffin nails," or "little white slavers." They implicated cigarettes in cancer, heart disease, and other serious health problems; however, opposition to the cigarette would gain little ground until compelling scientific evidence linked smoking and disease. Researcher, educator, and activist Ernst Wynder, M.D. (April 30, 1922-July 14, 1999), dedicated his career to producing this evidence.

Ernst Wynder was born in Herford, Germany. His family emigrated to New Jersey in 1938 to escape Nazi persecution. He attended medical school at Washington University, St. Louis, Missouri, and received both a bachelor of science and a medical degree in 1950. Wynder began his lung cancer investigations when he was a medical student. While attending a summer internship at New York University, his curiosity was piqued during the autopsy of a two-pack-a-day smoker who had died from lung cancer. Wynder began collecting case histories of lung cancer victims, first in New York City and then in St. Louis. His research brought him to thoracic surgeon Evarts Graham, who, despite initial skepticism about Wynder's premise (Graham was a heavy smoker), granted access to his extensive case records, and agreed to sponsor the medical student.

In 1950, the *Journal of the American Medical Association* published Wynder and Graham's "Tobacco Smoking as a Possible Etiologic Factor in Bronchiogenic Carcinoma: A Study of 684 Proven Cases." Wynder and Graham's retrospective study was not the first to link smoking and cancer, but its sophisticated design, impressive population size, and unambiguous findings demanded attention and further research. During the next decade, hundreds of reports were published linking cancer and smoking, including large prospective studies, pathologic, and animal investigations. A second effect was to convince doctors that the health risks of smoking were serious. Many gave up the habit, including Graham, who quit smoking in 1952. Too late, it would seem, as he wrote to Wynder in 1957, weeks before the surgeon died from lung cancer.

Wynder devoted his career to the study and prevention of cancer and chronic disease, writing hundreds of scientific papers advocating further research and public education. Through the 1950s and 1960s he worked at the Sloan-Kettering Institute for Cancer Research; in 1969, he founded the American Health Foundation, serving as its medical director. In 1972, the foundation launched *Preventive Medicine*, with Wynder as editor. In 1999, the foundation employed approximately 200 researchers representing medicine, public health, biology, chemistry, nutrition, and behavior science. Wynder endured years of criticism from the tobacco industry and skepticism from many researchers, but he remained determined.

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Future Challenges

Despite the achievements of the 20th century, approximately 48 million U.S. adults smoke cigarettes; half of those who continue to smoke will die from a smoking-related disease. Tobacco use

is responsible for approximately 430,000 deaths each year—one of every five. Parallel to the health burden is the economic burden of tobacco use, which amounts to at least $50 billion in medical expenditures and $50 billion in indirect costs. If trends continue, approximately 5 million children living today will die prematurely because as adolescents they started smoking cigarettes.[16] Advances have been made in knowledge of tobacco use and its effect on health; intervention strategies to reduce these effects remain serious challenges.

First, trends from the 1975-1998 Monitoring the Future surveys[18] indicate that the 30-day prevalence of tobacco use (smoking on ≥1 of the 30 days before the survey) among high school seniors decreased from the late 1970s to the mid-1980s, and prevalence was approximately 30%; however, during 1991-1997 smoking prevalence increased to 36.5%. Prevalence among high school seniors today is highest among whites and lowest among blacks.[18] The recent increases in prevalence highlight the need for a nationwide comprehensive prevention program focused on this age group.

Second, decreasing prevalence among adults since the mid-1960s has not continued. Since 1990, prevalence among both men and women has remained constant (approximately 28.0% for men and approximately 22.5% for women). The stagnation emphasizes the need for policy changes that encourage quitting and for improved access to proven treatment interventions (e.g., Food and Drug Administration-approved pharmacotherapy and behavior counseling).

Third, large differences in tobacco use exist in the United States. For example, in 1997, smoking prevalence was 37.9% among American Indian/Alaska Native men, 32.1% among black men, and 27.6% among white men.[19] There are marked differences in deaths from malignant diseases of the respiratory system; the age-adjusted death rates per 100,000 U.S. residents in 1995 were 80.5 among black men and 53.7 among white men.[12] Age-adjusted death rates for cerebrovascular disease also reflect the disparity in health outcomes, with the rate being 53.1 per 100,000 among black men and 26.3 among white men.[12] No single factor determines the patterns of tobacco use among racial/ethnic groups; these patterns result from complex interactions among multiple factors such as socioeconomic status, cultural characteristics, acculturation, stress, biologic elements, targeted advertising, price of tobacco products, and varying capacities of communities to mount effective tobacco-control initiatives. These disparities in use and adverse health outcomes based on race/ethnicity and socioeconomic status need to be addressed.

Fourth, exposure to environmental tobacco smoke (ETS) at home and at work is a substantial problem. One study found that 87.9% of children and adult nonusers of tobacco had detectable levels of serum cotinine.[20] The distribution of serum cotinine levels is bimodal: one peak for nonsmokers exposed to ETS and a higher one for smokers. Both the number of smokers in the household and the hours exposed at work were associated with increased serum cotinine levels among nonsmokers.

Fifth, research is needed to determine whether new "highly engineered" products can reduce the harmful effects of tobacco or whether the mistakes associated with low tar and nicotine cigarettes will be repeated.[21] Several novel tobacco products, (e.g., bidis from India) appear to be increasing in popularity, but little is known about long-term health effects or about social and other factors associated with their use.[22]

Sixth, a dramatic increase in tobacco use has occurred worldwide. Because of the increase, the World Health Organization (WHO) established the Tobacco Free Initiative, and the World Health Assembly unanimously approved the development of a Framework Convention on Tobacco Control. This WHO effort will promote global cooperation on aspects of tobacco control that transcend national boundaries and will necessitate political action; mobilization of resources; and implementation of national, regional, and global strategies.

Much remains to be done despite the public health achievements in reducing tobacco use in the 20th century. The American Cancer Society has set goals for 2015 of a 25% reduction in cancer incidence and a 50% reduction in cancer mortality rates.[23] Approximately 50% of that goal can be achieved with a 40%-50% reduction in smoking prevalence by 2005. Commensurate with the cost of the harm caused by tobacco, resources must be expended, including programs preventing adolescents from starting to smoke, getting adults and young people to quit smoking, and eliminating exposure to ETS and disparities among population groups.

Reported by: Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.

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Surveillance for Acute Pesticide-Related Illness During the Medfly Eradication Program—Florida, 1998

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THE MEDITERRANEAN FRUIT FLY (Medfly) (*Ceratitis capitata*, Wiedemann) is an exotic insect that can damage approximately 250 fruit and vegetable plant species and is a serious threat to domestic agriculture. During the spring and summer of 1998, pesticides were used by federal and state agriculture authorities to eradicate Medfly infestations that had been detected in portions of five Florida counties. This report summarizes surveillance data, describes probable and possible cases of illness associated with the eradication effort, and

provides recommendations for future Medfly-eradication programs.

The Medfly Eradication Program began on April 4, 1998, with ground applications of malathion/bait* and diazinon, followed by aerial malathion/bait application that began on April 30. All insecticide applications were completed on September 6. The respective county health departments estimated that 132,000 persons resided in the areas treated with these pesticides.

Surveillance for Illness

Reports of potential adverse health effects attributed to the Medfly Eradication Program pesticide applications were solicited by state health and agriculture authorities and collected through telephone hotlines maintained by the Florida Poison Information Network and county health departments. The public was advised of the pesticide use and the hotline number through public meetings hosted by federal and state agriculture department officials, news articles, and radio and television reports. During April 30-September 30, 1998, 230 reports of illness were received from Florida residents and physicians and were investigated by the Florida Department of Health. Reports were classified according to a standard case classification system.† Of the 230 reports, 34 (15%) cases were classified as probable pesticide-related illness based on abnormal medical signs compatible with malathion/bait or diazinon toxicity observed by a licensed health-care professional, and 89 (39%) were classified as possible based on symptoms compatible with malathion/bait or diazinon toxicity reported to health-care providers or a state health authority. Of the remaining 107 (47%), 24 were excluded because of insufficient information, 32 were asymptomatic or had symptoms unrelated to exposure, and 51 were classified as unlikely. No reports were classified as definite cases of pesticide-related illness because this category requires confirmation by laboratory testing of clinical or environmental samples that were not available.

The 123 probable or possible cases represent a crude rate of nine cases per 10,000 residents in the exposed areas. Of the 123, 89 (72%) occurred in females; the median age was 46.5 years (range: 6 months-82 years). Eight reports (7%) involved children aged ≤5 years, and 20 (16%) involved persons aged ≥65 years. Four reports (3%) described persons whose illnesses were considered work-related (i.e., Medfly Eradication Program pesticide applicator, lawn-care worker, health department hotline worker, and hotel worker).

Among the 123 cases, signs and symptoms for 87 (71%) were respiratory (e.g., dyspnea, wheezing, coughing, and upper respiratory tract pain/irritation); 77 (63%) involved the gastrointestinal system (e.g., nausea, vomiting, diarrhea, melena, and abdominal cramping); 74 (60%) involved the neurologic system (e.g., headache, vertigo, ataxia, peripheral paresthesia, disorientation, and confusion); 28 (23%) involved the skin (e.g., erythema [with or without maculopapular rash], pruritis, and burning sensations); and 23 (19%) involved the eyes (e.g., lacrimation, conjunctivitis, blepharitis, and blurred vision)‡.

Case Reports

Case 1. A 49-year-old man experienced dyspnea, upper respiratory irritation, and headache after being exposed to aerial malathion/bait applications while working on the roof of his house. His physician diagnosed severe bronchitis and reported that the illness probably resulted from malathion/bait exposure.

Case 2. A 31-year-old man reported a blistering rash over his arms, legs, and neck following an aerial application of malathion/bait. He was exposed to malathion/bait while conducting his lawn maintenance business. He reported that the rash developed where grass trimmings coated with pesticide stuck to his skin. His physician diagnosed allergic contact dermatitis secondary to malathion/bait exposure.

Case 3. A 35-year-old man reported a pruritic rash on exposed skin surfaces. He had covered his pool in accordance with recommendations and was exposed to malathion/bait while removing the cover, which he had folded and carried under his right arm. He was not wearing a shirt, and the rash developed at those points where the pool cover had contacted his arm and torso. His physician diagnosed allergic dermatitis.

Case 4. A 32-year-old woman with a history of asthma complained of multiple symptoms in reaction to ground applications of malathion/bait and diazinon in her neighborhood. Symptoms included nausea, diarrhea, abdominal cramping, cough, upper respiratory irritation, dyspnea, wheezing, headache, and fatigue. Her physician diagnosed acute aggravation of asthma secondary to pesticide exposure from the Medfly Eradication Program.

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CDC Editorial Note: The Environmental Protection Agency (EPA) classifies malathion as an acute toxicity category III compound§, and it is considered safer than many other organophosphates because it is rapidly detoxified by the body. Nevertheless, adverse health effects have been reported by persons exposed to malathion.[1] Self-reported health effects previously associated with aerial spraying of malathion/bait include respiratory symptoms (particularly among persons with pre-existing respiratory conditions), gastrointestinal symptoms, neurologic symptoms, contact dermatitis, and conjunctivitis.[2-4] These effects may represent irritant or allergic responses to either component of the malathion/bait formulation.[5,6] Cholinesterase inhibition[3] or anxiety about aerial malathion/bait application[2,7] also may be responsible for some symptoms.

The findings in this report suggest that for most persons, aerial application of malathion/bait does not pose an acute risk to health; however, at least

123 probable or possible pesticide-related cases of illness were associated with pesticide exposure. Each case-patient had signs and/or symptoms consistent with pesticide exposure, and illness probably resulted from sensitivity to the irritant/allergic effects of malathion/bait. Although ground application of diazinon, another acute toxicity category III organophosphate, was employed in some locations, this agent was considered less likely to be responsible for the observed health effects because it was used in only three counties, was applied focally (without aerial application), and was used in minimal quantities.

The findings in this report are subject to at least three limitations. First, because this was a passive surveillance effort, persons may have become ill who did not seek medical attention or were not reported to the surveillance system. Second, rates of the health outcomes in the exposed population could not be compared with those for the general population because baseline incidence data for many of the effects attributed to the malathion/bait application are not available. Third, the role of cholinesterase inhibition was not determined because blood cholinesterase levels were not obtained.

Certain malathion formulations are registered by EPA for aerial spraying over urban areas in mosquito-control programs. The use of malathion in these programs provides an important public health benefit by controlling mosquitoes that transmit human diseases such as encephalitis, dengue fever, and malaria. Spraying malathion/bait over urban populations for Medfly eradication has generated controversy in part because these applications are directed not at preventing human illness but at eradicating an agricultural pest. Federal law does not permit spraying malathion/bait over urban areas without an emergency EPA exemption§*. To reduce the risk for illness among persons sensitive to the effects of malathion/bait applications, federal and state agricultural authorities are encouraged to pursue and enhance alternative methods for Medfly control. These methods include preventing Medfly importation into the United States, quickly detecting Medfly infestations (e.g. through increased sentinel trapping densities), releasing sterile male Medflies to interrupt the reproductive cycle, and identifying and using safer eradication agents.

During aerial malathion applications for mosquito control and Medfly eradication, the public should be advised to stay indoors and, when appropriate, persons with exposure-related health concerns should seek medical attention. The public also should be provided with an opportunity to ask questions and receive timely responses about the malathion applications (i.e., through telephone hotlines and community meetings). When malathion/bait applications are used for Medfly eradication, additional precautions are recommended, including immediately washing any skin surfaces that come into contact with malathion/bait-contaminated surfaces; providing advance public notification of spray schedules; performing aerial malathion/bait applications when residents are usually indoors (e.g., at night); directing the homeless to shelters; advising highly sensitive persons to leave the area during spraying; and convening a health advisory committee, an action that has been shown to be useful for mitigating risk.[7] Medfly Eradication Program workers should be trained in the safe handling of pesticides, and consideration should be given to measuring plasma and red blood cell cholinesterase in these workers before beginning exposure and periodically thereafter.[8] Workers should wear the personal protection equipment (PPE) listed on the pesticide label. Supplementary PPE also may be indicated.

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*Malathion (Fyfanon® ULV, Cheminova Inc., Wayne, New Jersey) combined with a corn protein bait, Nu-Lure® (Miller Chemical and Fertilizer Co., Hanover, Pennsylvania) was applied at a rate of 2.4 fluid ounces malathion and 9.6 fluid ounces bait per acre per week. The reportedly nontoxic bait comprises hydrolyzed corn gluten meal and inert ingredients including corn syrup. Backpack sprayers or truck-mounted pressure sprayers were used for ground applications; UH-1 "Huey" helicopters and DC-3 aircraft conducted the aerial applications. (Use of trade names and commercial sources is for identification only and does not imply endorsement by CDC or the U.S. Department of Health and Human Services.)

†CDC's National Institute for Occupational Safety and Health classifies a case of acute pesticide-related illness and injury as being definite, probable, possible, or suspicious as determined by the level of certainty of exposure, whether health effects were observed by a health-care provider, and whether sufficient toxicologic information supports a causal relation between the exposure and the reported health effects. When toxicologic evidence for an exposure-health effect relation is not present, the case is classified as unlikely.

‡Total is 289 cases because some persons experienced signs and symptoms in more than one system.

§EPA classifies pesticides into one of four acute toxicity categories based on established criteria (40 CFR Part 156). Pesticides with the greatest toxicity are in toxicity category I and those with the least are in category IV.

§*40 CFR Part 166.