American Thoracic Society
MEDICAL SECTION OF THE AMERICAN LUNG ASSOCIATION

Cigarette Smoking and Health

This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, March 1995.

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Purpose of the Statement

Knowledge about the adverse health effects of cigarette smoking has accumulated for more than four decades. Since the first report of the U.S. Surgeon General in 1964 causally linking cigarette smoking with the development of lung cancer, numerous scientific studies and summary reports have been published that document the contribution of cigarette smoking to morbidity and mortality from a variety of conditions. Although the prevalence of cigarette use has and continues to decline, smoking remains the leading cause of preventable death in the United States.

In 1985, the American Thoracic Society (ATS) first issued a position statement titled "Cigarette Smoking and Health," which reviewed the adverse health consequences of smoking known at that time. During the ten years since this initial statement, considerable scientific evidence has emerged confirming the findings of the initial report, expanding the number of conditions known to be caused or worsened by smoking, and describing the biological mechanisms by which smoking causes adverse health effects. Furthermore, passive inhalation of environmental tobacco smoke (ETS) by nonsmokers has been associated with many of the same health consequences as active smoking, albeit with much smaller degrees of increased risk, and has recently been classified by the Environmental Protection Agency as a Class A (known human) carcinogen.

As in the 1985 ATS statement on smoking, the conclusions reached here are based on the total weight of scientific data and consensus opinion. This evidence derives both from laboratory studies and from epidemiologic studies of human populations, with causal inferences based on the consistency, reproducibility, and strength of associations, as well as the presence of an exposure-response relationship.

Am J Respir Crit Care Med Vol 153. pp 861–865, 1996

Prevalence of Cigarette Smoking

The prevalence of cigarette smoking in the United States has decreased since 1964, when the first Surgeon General's *Report on Smoking and Health* was published. In 1965, 52% of men and 34% of women over age 18 were cigarette smokers. By 1991, these percentages had decreased to 28% for men and 24% for women. Similarly, per capita cigarette consumption (for adults over age 18) peaked in 1963 at 4,345 and by 1992 had decreased 39% to 2,640. Still, cigarette smoking among younger people remains a major public health concern. Nearly all first use of tobacco occurs before high school graduation, and each day 3,000 teenagers start to smoke. Among high school seniors, the prevalence of daily cigarette smoking, which was 29% in 1976, has remained in the 17 to 19% range since 1984. Despite an overall decline in the number of smokers in the United States and much of the developed world, the prevalence of cigarette smoking continues to increase in many developing countries.

Cost of Smoking

Cigarette smoking is very costly to both the individual and society. New long-term studies estimate that about half of all regular cigarette smokers will eventually be killed by their habit. In 1993 the estimated smoking-attributable costs for medical care were $50 billion. When lost work and productivity were added, the total cost to society was estimated to exceed $97 billion, or $373 per capita. If these costs were borne by smokers in the form of cigarette taxes, the price of each pack of cigarettes would have to rise to $4.

General Health Effects and Mortality

Cigarette smoking is a major contributor to mortality in the United States. In 1990, approximately 440,000 Americans died from diseases directly related to cigarette smoking. More impressively, at that time more than one out of every five deaths was the result of smoking. The American Cancer Society's Cancer Prevention Study II, a prospective evaluation of almost 1.2 million men and women from 1982–1986, found that the overall mortality ratio (i.e., deaths from all causes in current smokers compared to deaths in those who never smoked) was 2.22 for men smoking 1 to 20 cigarettes/d and 2.43 for men smoking ⩾ 21 cigarettes/d. Similarly, the mortality ratio was 1.60 and 2.10, respectively, for women smoking 1 to 19 and ⩾ 20 cigarettes/d. Cigarette smoking remains the most preventable cause of premature death in the United States. It has been estimated, for example, that a heavy smoker at age 25 can expect a life expectancy at least 25% shorter than a nonsmoker.

Cigarette smokers also have greater morbidity than nonsmokers. Current smokers have more acute and chronic illness as well as more restricted activity days, more bed disability days, and more school and work absenteeism than former smokers or those who never smoked.

General Pulmonary Effects

Cigarette smoking alters both the structure and function of central and peripheral airways, alveoli and capillaries, and the immune system of the lung. Further, a multistep transformation from normal pseudostratified ciliated epithelium to squamous metaplasia, carcinoma *in situ*, and eventually invasive broncho-

genic carcinoma has been reported by several prospective studies.

Numerous pulmonary function abnormalities have been documented in smokers. In general, current smokers have a lower FEV_1 and an accelerated decline in FEV_1 compared to those who formerly or never smoked. Both of these associations show a dose-response relationship and are more dramatic in men than women. A relatively low FEV_1 by middle age and a faster-than-expected annual fall in FEV_1 are the two most useful findings in identifying smokers who are likely to develop severe pulmonary impairment.

Respiratory symptoms are greatly increased among cigarette smokers. A dose-response relationship exists for chronic cough and phlegm production, wheeze, and dyspnea. Smoking-induced changes in airway epithelium including loss of cilia, mucous gland hypertrophy, an increase in the number of goblet cells, and increased permeability underlie the development of these respiratory symptoms.

Cigarette smoking is the principal risk factor for chronic obstructive pulmonary disease (COPD). The exact mechanism by which smoking causes obstructive lung disease has not been firmly established. One proposed mechanism is that cigarette smoking allows an imbalance between proteolytic and antiproteolytic activity in the lung, possibly resulting in parenchymal destruction and airflow obstruction. Population-based studies also support a role for smoking as a cause of heightened airway responsiveness, which in turn may be a risk factor for the development of COPD.

An estimated 10 to 15% of all smokers develop clinically significant airflow obstruction. $Alpha_1$-antiprotease deficiency remains the only known determinant of susceptibility for cigarette smokers. Dust and fume exposure and severe respiratory illness during childhood may also be risk factors.

Finally, data from both prospective and retrospective studies have consistently reported increased mortality from COPD, pneumonia, and influenza among cigarette smokers compared with nonsmokers.

Lung Cancer

The 1964 Surgeon General's report concluded that cigarette smoking was causally related to lung cancer. Since then, irrefutable evidence has documented that cigarette smoking is the major cause of lung cancer of each of the principal histologic types (i.e., epidermoid, small cell, large cell, and adenocarcinoma) for both men and women. In 1991, there were 161,000 new cases of lung cancer and 143,000 deaths from lung cancer.

The amount and duration of smoking determine the risk of lung cancer for individual smokers. Men or women who smoke $\geq$ 40 cigarettes/d have twice the lung cancer risk of those who smoke $\leq$ 20 cigarettes/d. Individuals who start smoking before the age of 15 yr are four times more likely to develop lung cancer than those who begin after the age of 25 yr. Epidemiologic studies have shown that brands of cigarettes that contain less tar and nicotine only marginally reduce the risk of lung cancer mortality. Similarly, little difference in mortality has been found for lifelong filter versus nonfilter smokers and for persistent smokers who switch from nonfilter to filter cigarettes.

During the last several years, lung cancer mortality has stabilized in men but continues to rise dramatically in women. Presently, lung cancer is the leading cause of cancer deaths in women, a distinction that is largely attributable to an earlier age at smoking initiation and a greater number of cigarettes smoked per day than previously reported.

The exact mechanism by which cigarette smoking causes lung cancer is not fully understood. A multistage model in which smoking sequentially transforms cells from normal to malignant is widely accepted. The components of tobacco smoke have been proven capable of initiating and promoting such a process of carcinogenesis.

Several host and environmental factors may influence the risk of lung cancer in cigarette smokers. A family history of lung cancer and exposure to radon or asbestos greatly increase the likelihood of lung cancer in smokers. The effects of genetic susceptibility and vitamin A deficiency on the risk of lung cancer in smokers are unknown, whereas the risk associated with ambient air pollution appears to be small.

Other Cancers

Epidemiologic studies have found strong associations between cigarette smoking and the development of cancer at several other sites (e.g., oral cavity, larynx, esophagus, bladder, kidney, pancreas, stomach, and cervix). In general, the smoking-related risk of developing malignancy at these sites is less than for lung cancer. Notably, smokers are at increased risk for a second smoking-related cancer once they have a tobacco-associated malignancy.

Cardiovascular Effects

Prospective studies from different countries have conclusively shown that male and female smokers are at greater risk of myocardial infarction, recurrent heart attacks, and sudden death from coronary heart disease (CHD) than nonsmokers. Smokers have a two- to fourfold increased incidence of CHD and a two- to fourfold greater risk of sudden death than nonsmokers. Mortality from CHD can be predicted by the number of cigarettes smoked per day, the depth of inhalation, the age of smoking onset, and the number of years smoked. Additionally, smoking has been shown to greatly modify the risk for CHD associated with other known risk factors such as hypercholesterolemia and diabetes.

Cigarette smoking produces acute and chronic myocardial changes that directly contribute to the development of CHD and its associated complications. Acutely, smoking may cause myocardial ischemia through an increase in oxygen demand or by reducing blood supply. This latter change may result from smoking-related coronary artery spasm and/or platelet aggregation and adhesiveness. Furthermore, smoking can lower the threshold for dysrhythmia, especially ventricular fibrillation, leading to sudden death. Chronically, cigarette smoking can result in coronary atherosclerosis, possibly by causing repetitive endothelial injury; increased platelet adherence with stimulation of smooth muscle proliferation; and increased low-density lipoprotein cholesterol and/or reduced high-density lipoprotein cholesterol.

Several major studies have shown that smoking causes stroke in both men and women. Cigarette smokers have almost a twofold greater risk of stroke than nonsmokers. This risk is dose-dependent and strongest in younger groups.

Smoking and Pregnancy

Maternal smoking during pregnancy is responsible for a number of significant health risks to the unborn child. Studies over several decades around the world have consistently demonstrated that infants born to mothers who smoke during pregnancy are on average 200 g lighter and 1 cm shorter than infants of nonsmoking mothers. Smoking-related fetal intrauterine growth retardation is a major health concern because it is associated with increased perinatal mortality from a variety of causes. Additionally, women who smoke at the time of conception or during pregnancy are more likely to have spontaneous abortions and pregnancy complications of placenta previa, placental abruption, and premature rupture of membranes than nonsmoking women.

Many of the carcinogenic and mutagenic constituents of tobacco smoke found in the blood of active smokers readily cross the placenta to the fetal circulation. However, the degree to which

most of the more than 4000 compounds present in tobacco smoke cross the placenta and the specific compounds that cause adverse health effects on the developing fetus are largely unknown. Nonetheless, the risk of several major birth defects is higher among infants exposed to maternal prenatal smoking.

Recent studies have documented residual developmental consequences of maternal smoking in pregnancy. *In utero* exposure of the infant to active maternal smoking has been associated with deficits in lung function, an increased risk of wheezing respiratory illness in early infancy, impairment of somatic (height) growth in childhood, and small deficits in intelligence and behavior as measured on standardized tests. For some of these adverse outcomes, the relative contributions of maternal smoking in pregnancy and in early infancy have not been fully elucidated.

Smoking and Occupation

Cigarette smoking can increase the risk of developing occupational lung disease and trigger exacerbations of existing work-related disease. Workers who smoke and are exposed to coal, silica, grain, or cotton dust are more likely to develop chronic bronchitis than nonsmoking workers with similar exposures or nonexposed smokers. The risk appears to be additive. Similarly, exposure to cigarette smoke and cotton dust or silica increases the likelihood of chronic airflow obstruction above what would be expected in occupationally unexposed smokers. Smoking also increases the risk of developing IgE antibodies and asthma among selected workers exposed to potential workplace allergens, such as platinum and humidifier-associated antigens.

Cigarette smoking plays an important etiologic role in occupationally associated lung cancer. Smoking workers exposed to asbestos, radon, arsenic, diesel exhaust, aromatic amines, and silica are more likely to develop cancer than nonsmoking workers. For two exposures, asbestos and radon daughters, the risk is synergistic. For example, nonsmoking asbestos insulation workers have a fivefold increased risk for lung cancer compared to nonsmoking individuals not exposed to asbestos, but smoking asbestos insulation workers have a more than fiftyfold increased risk of lung cancer.

Passive Smoking

Since the previous ATS statement on smoking and health in 1985, knowledge about the harmful effects of exposure to ETS indoors has greatly expanded. Many of the deleterious health effects known to be caused by active smoking have now been associated with passive smoking.

Environmental tobacco smoke is composed of sidestream and mainstream smoke. Sidestream smoke (SS), which is emitted from the burning end of a lit cigarette, contains the same compounds found in mainstream smoke (MS), which is inhaled into the smoker's lungs and exhaled. In fact, many of the 4000 known compounds and more than 40 known carcinogens found in MS are present in greater concentrations in SS. This fact supports the potential for adverse health outcomes also to be associated with intensive and/or protracted exposure to ETS. Furthermore, increased levels of some of the known constituents of ETS have been measured in exposed nonsmokers and vary, in part, with room size, ventilation, number of smokers, and rate of smoking.

Early in the 1980s, several controversial epidemiologic studies found that nonsmoking women living with husbands who smoked cigarettes had a significantly higher risk of developing lung cancer than nonsmoking women whose husbands were also nonsmokers. More than 30 additional studies have since provided compelling evidence that nonsmoking women living with smoking spouses have a 1.2 to 2 times the risk of developing lung cancer during their lives than nonsmoking women in smoke-free homes. The causal nature of this association can be inferred from the consistency of investigational results from many different geographic areas and/or cultures, the presence of a dose-response relationship, and the persistence of the association in studies where other potential confounding factors were controlled. In addition, passive smoking has been causally related to the development of lung cancer among occupational cohorts, independent of exposure to carcinogenic occupational toxins.

In 1992, the U.S. Environmental Protection Agency issued a report in which environmental tobacco smoke was classified as a Class A (known human) carcinogen. The report estimated that annually in the U.S., 3,000 new cases of lung cancer in nonsmokers or former smokers can be attributed to ETS exposure. The risk of lung cancer posed by shorter periods of passive smoke exposure, as might occur in occupational settings or public environments such as restaurants, or to children exposed to ETS by household smokers remains to be determined.

Most studies that have evaluated the association of involuntary smoking in CHD have reported an excess risk of 20 to 50% among nonsmokers living with smokers. Further, an exposure-response relation has been documented in several of these investigations. The American Heart Association estimates that even with risk ratios as low as 1.2, ETS exposure would account for 30,000 to 40,000 excess heart disease deaths per year in the U.S., which is a significantly larger number than suggested for lung cancer.

In adults, few studies have examined the impact of household involuntary smoking on respiratory symptom frequency. Most of the available investigations, however, do support a modest increase in symptoms for exposed nonsmoking spouses. Involuntary smoking has also been associated with modest declines in levels of forced expiratory flow measures (1 to 3%) such as FEV_1, which are unlikely to be of clinical significance.

Results from more than 50 epidemiologic studies support a substantial increase in respiratory morbidity for children exposed to household ETS. Infants exposed to maternal smoking have a greater incidence of acute lower respiratory illnesses such as pneumonia, bronchiolitis, and bronchitis in the first 2 yr of life. This increased risk may also be influenced by paternal smoking and the total number of smokers in the household. The EPA report estimates that ETS causes 150,000 to 300,000 excess cases of lower respiratory illnesses per year in the U.S. in infants 18 mo or younger. Chronic respiratory symptoms such as cough, phlegm, and wheeze are all more common in children whose parents smoke. The association of passive smoking with childhood asthma is less certain.

Involuntary smoking reduces the growth rate of lung function in children. Fewer studies of the effects of ETS on airway responsiveness have been reported, but most describe increased bronchial responsiveness in children from homes of smokers.

Why People Smoke

Research investigating the reasons that people smoke has evaluated social, psychological, and pharmacological factors. It is widely accepted that the factors that influence smoking initiation differ from those that influence maintenance of smoking behavior. Social and familial factors seem to be particularly important in smoking initiation. Smoking by family members and friends is strongly associated with smoking initiation in adolescence, whereas nicotine dependence, genetic, and psychosocial factors are relevant in maintenance of smoking behavior.

Recent studies have examined the impact of genetics on smoking status. Studies in twins suggest that genetic factors do contribute to tobacco use; heritability estimates range from 35 to 68%. There also appear to be genetic contributions to the amount of smoking (light versus heavy) and to the ability to quit smoking. It has been postulated that the mechanism for a genetic in-

fluence may be constitutional differences in sensitivity or reactiveness to nicotine's toxic effects. This hypothesis suggests that smokers are constitutionally sensitive to nicotine, and smoking initiation by these individuals leads to a rapid decrease in sensitivity, which in turn leads to tolerance of nicotine's effects. Further research is needed in this area.

Despite growing evidence for a genetic contribution, smoking behavior is multifaceted. The 20-yr, 1 pack/d smoker has inhaled smoke over one million times; this repetitive act creates many strong cues for smoking throughout daily life. Smoking-related cues have been found to precipitate relapse, particularly when experienced in social or stressful situations. Laboratory experiments have shown that individuals who are highly reactive to smoking-related cues are more likely to relapse. Smokers who are successful at quitting report lower overall levels of perceived stress. Further, smoking by family and friends is associated with difficulty quitting. It is important for smoking cessation programs to take these psychosocial factors into consideration.

The Role of Addiction

It is well established that nicotine meets the criteria of a highly addictive drug. Like other psychoactive drugs (e.g., cocaine, heroin, or alcohol), nicotine serves as a reinforcer of its use and meets criteria for abuse liability in humans. The nicotine withdrawal syndrome, which is now well characterized, includes craving to use nicotine, irritability, anxiety, difficulty concentrating, restlessness, and increased appetite. Addictions are governed by behavioral, psychological, and biological processes. The fact that smoking has a biologically addictive component does not obviate educational, social, and policy strategies to prevent its onset and promote cessation.

Smoking Cessation

Although smoking prevalence has declined over the past several years, smoking cessation rates have not changed appreciably. Initial abstinence rates tend to be high, but decline significantly over time. On average, most cessation programs yield between 20 to 40% abstinence rates at 12 mo, with programs that focus specifically on relapse prevention having slightly better outcomes. Although these outcomes are somewhat disappointing, they are significant from a public health perspective, especially in light of the enormous economic and health costs of smoking. In addition, relapse appears to be an important part of the cessation process. Smokers who have been successful in achieving long-term abstinence report having previously stopped and relapsed multiple times. Therefore, relapse should not be considered failure.

Substantial scientific and community resources have been dedicated to efforts to increase rates of smoking cessation. Many alternatives are currently available, including self-help, group programs, nicotine replacement therapy, mass media, and physician-delivered, worksite, and community programs. Regardless of the mode of delivery, most smoking cessation programs focus on providing behavioral skills thought to be necessary for achieving initial cessation and maintaining abstinence. Although individual smokers are often the best judge of what smoking cessation approach will suit them best, a stepped-care model has been advocated, wherein over subsequent attempts to quit, smokers progress from the least intensive strategies (e.g., self-help) to the most intensive (e.g., medication plus psychological intervention).

In intervention planning, it is also important to consider the level of motivation for quitting. The Stages of Change Model provides a conceptual model for understanding the process of smoking cessation. According to this model, smokers' readiness to quit can be characterized on a continuum with five stages; precontemplation (not at all ready to quit), contemplation (considering cessation at some future time), preparation (actively considering cessation soon and engaging in some quit-oriented behaviors), action (having quit smoking within 6 mo), and maintenance (having quit for at least 6 mo). Population-based data suggest that a relatively small percentage of smokers are "ready" to actively try to quit smoking at any given time. Smoking cessation efforts should be matched to smokers' readiness, or level of motivation for cessation.

Over the last decade, there has been increased attention to the key role that physicians play in motivating smokers to consider cessation. Brief advice from a physician has been found to double the spontaneous quit rate of 2 to 4%. Thus, physician-delivered smoking cessation interventions can have a tremendous public health impact. Several resources are available for providing guidance to physicians in developing and implementing office-based smoking cessation systems.

The availability of pharmacological aides (e.g., nicotine polacrilex, nicotine patch) has increased smokers' reliance on their physicians for advice and assistance regarding cessation. Nicotine replacements have been developed to provide nicotine without the adverse health consequences associated with the other constituents of cigarettes, and thereby give the smoker a means of reducing nicotine withdrawal symptoms while dealing with the behavioral and psychological components of smoking cessation. Nicotine replacement therapies have doubled smoking cessation rates over that found with placebo, particularly for smokers who are heavily dependent on nicotine. In addition, combining nicotine replacement with psychological therapy has been found to further increase smoking cessation rates. Neither the patch nor the gum are appropriate as a long-term alternative to smoking. Furthermore, there have been cases of myocardial infarction reported among smokers who continued to smoke while using the nicotine patch. Therefore, it is critical that all patients considering nicotine replacement therapy be counselled and monitored regarding proper use. Alternative forms of nicotine delivery that may further enhance smoking cessation, such as a nicotine inhaler or nasal sprays, are currently being tested.

Over the past 20 years, women have begun to smoke in great numbers, and therefore special attention needs to be paid to the impact of smoking for women. Some studies suggest that women may have lower cessation rates and higher relapse rates than men. There may be important gender differences in strategies needed for effective smoking cessation, although few studies have used gender-specific treatment protocols. This is an important area for future study.

Health Benefits of Quitting

Smoking cessation has major and immediate health benefits for men and women of all ages. People who quit smoking before age 50 have one-half the risk of dying over the next 15 yr compared to people who continue to smoke. This improvement is directly related to specific smoking-related diseases. After 1 yr of smoking cessation, the excess risk of CHD mortality is reduced by about one-half and continues to decline with time. Smoking cessation also decreases other nonmalignant disease morbidity and mortality, including that from stroke, peripheral vascular disease, COPD, pneumonia, and gastric or duodenal ulcers. The risk of lung cancer declines steadily in people who quit smoking until, after 10 yr, the risk is about 30 to 50% of that in continuing smokers. Smoking cessation also reduces the risk of other tobacco-related malignancies, including cancers of the larynx, esophagus, pancreas, and urinary bladder. The health benefits of smoking cessation are immediate and substantial, extending to men and women of all ages, both sick and healthy.

Smoking Prevention

Efforts to enhance smoking cessation are important, but smoking prevention is central to a smoke-free society. Almost 90% of smokers start smoking before age 20. Over 3,000 young Americans become new smokers every day. Although some differences have been found in smoking acquisition among ethnic groups, the patterns are strikingly similar. Factors that predict smoking initiation include dropping out of school, having parents and friends who smoke, and being from a low-income or single-parent household.

Smoking prevention efforts have focused on school-based curricula. Such programs focus on the social influences that encourage smoking, provide training and skills rehearsal for resistance of those influences, and target social norms. Numerous studies suggest that smoking prevention curricula can delay the onset of smoking for 2 to 4 yr. The most successful interventions have included mass media campaigns as part of community-wide interventions.

Policy and environmental interventions are widely considered to be cornerstones of smoking prevention efforts. Examples of such interventions include bans of cigarette vending machines in order to reduce adolescents' access to cigarettes and enforcement of age restrictions on cigarette sales. In addition, increased taxes on cigarettes have been found to be directly related to reductions in smoking initiation among children and adolescents.

Summary

Cigarette smoking remains the primary cause of preventable death and morbidity in the United States. Smoking causes lung cancer, COPD, and CHD and contributes significantly to mortality from other conditions such as stroke. Maternal smoking during pregnancy causes low birthweight and perinatal mortality, and it may have lasting impact on the child's physical and cognitive growth. Passive exposure to ETS causes lung cancer and poses particular danger to the respiratory health of young children. Smoking cessation strategies are important, but they should be supplemented by community and policy-level interventions. Workplace or community smoking bans, statewide taxes on tobacco, and antismoking media campaigns may be effective adjuncts to individual cessation strategies. These strategies may be an even more important disincentive to smoking initiation. The expanding horizon of health consequences of smoking and its costs to American society should again challenge public health agencies to develop and implement effective strategies to prevent smoking acquisition by young people. These health effects should also motivate health professionals in other countries where smoking prevalence is increasing, rather than decreasing, to initiate more effective efforts to reverse this trend and minimize the excess morbidity and death that accompany this dangerous habit.

Acknowledgment: This statement was prepared by a subcommittee of the Scientific Assembly on Environmental and Occupational Health. The members of the committee are:

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