Foreword

Smoking kills an estimated 434,000 Americans each year, most of whom began smoking during their adolescence. The key to reducing this enormous death toll and the health consequences that accompany tobacco use, according to public health officials, is preventing young people from starting to use tobacco. Very few people begin to use tobacco as adults; almost all first use has occurred by the time people graduate from high school. By the age of 18, one in three persons is using tobacco.

The earlier young people begin using tobacco, the more heavily they are likey to use it as adults, and the longer potential time they have to be users. Both the duration and the amount of tobacco use are related to eventual chronic health problems.

This publication is adapted from *Preventing Tobacco Use Among Young People: A Report of the Surgeon General* released by the U.S. Department of Health and Human Services in 1994. The excerpts presented here provide important information for educators about the vulnerable ages of 10 through 18 when most users start smoking, chewing, or dipping and become addicted to tobacco. It underscores the seriousness of tobacco use and the relationship of tobacco use to other adolescent problem behaviors.

We appreciate the cooperation of the Office of the Surgeon General in making this report available for distribution by the Department of Education. Copies of the complete document are available for sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C., 20402, S/N 017-001-00491-0.

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Chapter 1
Introduction

Previous Surgeon General's reports on tobacco use and health have largely focused on the epidemiologic, clinical, biologic, and pharmacologic aspects of adult use of tobacco products. This report on *Preventing Tobacco Use Among Young People* provides a more detailed look at adolescence, the time of life when most tobacco users begin, develop, and establish their behavior. Because regular use soon results in addiction to nicotine, this behavior may persist through adulthood, significantly increasing, through the extended years of use, the risk of long-term, severe health consequences.

Despite three decades of explicit health warnings, large numbers of young people continue to take up tobacco; currently, over three million adolescents smoke cigarettes, and over one million adolescent males currently use smokeless tobacco. Clearly, effective interventions are needed to prevent more young people from trying tobacco. To achieve significant long-term reductions in tobacco use and tobacco-related deaths in the United States, we must examine the nature and scope of adolescent tobacco use, consider the social, psychological, and marketing factors that influence young people in their decision to use tobacco products, and evaluate current efforts to prevent young people from becoming users. This report addresses the crucial problems of adolescent tobacco use.

Development of the Report

This report of the Surgeon General was prepared by the Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Public Health Service, U.S. Department of Health and Human Services, as part of the department's responsibility, under Public Law 91-222 and Public Law 99-252, to report current information on the health effects of cigarette smoking and smokeless tobacco use to the United States Congress. This report is the first to focus on the problem of tobacco use among young people. Given the continuing onset of use in adolescence and the growing evidence of health consequences associated with early use, the report was seen as both needed and timely.

The current report has been produced through the efforts of experts in the medical, pharmacologic, epidemiologic, developmental, economic, behavioral, legal, and public health aspects of smoking and smokeless tobacco use among young people. Initial manuscripts for the report were prepared by 28 scientists who were selected for their expertise in specific content areas. This material was consolidated into chapters, each of which underwent peer review. The entire document was reviewed by a number of experts in the field, as well as by institutes and agencies within the U.S. Public Health Service. The final draft of the report was reviewed by the Assistant Secretary for Health and by the Secretary, Department of Health and Human Services.

Several concerns guided the development of this report. The first, which is addressed in Chapter 2, is whether tobacco use is associated with health consequences during the period of adolescence (broadly defined as ages 10 through 18, although research cited in this report varies somewhat in the ages considered adolescent). The long-term health consequences—that is, those that emerge in adulthood—have been the subject of extensive review and are widely acknowledged in the scientific and public literature. The chapter thus focuses on the serious health consequences, as well as the increased risk factors for subsequent health consequences, that are evident early in life among young smokers and smokeless tobacco users. Chapter 3 examines the epidemiologic patterns of tobacco use among the young. National data on trends in adolescent use are analyzed to determine the extent of the current problem, as well as to note changes in patterns of initiation and use. The factors that influence adolescents in their decision to use tobacco are examined in Chapter 4, which considers psychosocial risk factors, and Chapter 5, which examines the influence of tobacco advertising and promotion. The final concern, the focus of Chapter 6, was to assess what has been done—from the individual level to the legislative level—to prevent tobacco use among young people.

Major Conclusions

1. Nearly all first use of tobacco occurs before high school graduation; this finding suggests that if adolescents can be kept tobacco-free, most will never start using tobacco.
2. Most adolescent smokers are addicted to nicotine and report that they want to quit but are unable to do so; they experience relapse rates and withdrawal symptoms similar to those reported by adults.
3. Tobacco is often the first drug used by those young people who use alcohol, marijuana, and other drugs.

4. Adolescents with lower levels of school achievement, with fewer skills to resist pervasive influences to use tobacco, with friends who use tobacco, and with lower self-images are more likely than their peers to use tobacco.
5. Cigarette advertising appears to increase young people's risk of smoking by affecting their perceptions of the pervasiveness, image, and fur tion of smoking.
6. Communitywide efforts that include tobacco tax i creases, enforcement of minors' access laws, yout oriented mass media campaigns, and school-bas tobacco-use prevention programs are successful reducing adolescent use of tobacco.

Summary

Introduction

The health effects of cigarette smoking have been the subject of intensive investigation since the 1950s. Cigarette smoking is still considered the chief preventable cause of premature disease and death in the United States. As was documented extensively in previous Surgeon General's reports, cigarette smoking has been causally linked to lung cancer and other fatal malignancies, atherosclerosis and coronary heart disease, chronic obstructive pulmonary disease, and other conditions that constitute a wide array of serious health consequences (USDHHS 1989). More recent studies have concluded that passive (or involuntary) smoking can cause disease, including lung cancer, in healthy nonsmokers. In 1986, an advisory committee appointed by the Surgeon General released a special report on the health consequences of smokeless tobacco, concluding that smokeless tobacco use can cause cancer and can lead to nicotine addiction (USDHHS 1986). In the 1988 report, nicotine was designated a highly addictive substance, comparable in its physiological and psychological properties to other addictive substances of abuse (USDHHS 1988).

Considerable evidence indicates that the health problems associated with smoking are a function of the duration (years) and the intensity (amount) of use. The younger one begins to smoke, the more likely one is to be a current smoker as an adult. Earlier onset of cigarette smoking and smokeless tobacco use provides more life-years to use tobacco and thereby increases the potential duration of use and the risk of a range of more serious health consequences. Earlier onset is also associated with heavier use; those who begin to use tobacco as younger adolescents are among the heaviest users in adolescence and adulthood. Heavier users are more likely to experience tobacco-related health problems and are the least likely to quit smoking cigarettes or using smokeless tobacco. Preventing tobacco use among young people is therefore likely to affect both duration and intensity of total use of tobacco, potentially reducing long-term health consequences significantly.

Health Consequences of Tobacco Use Among Young People

Active smoking by young people is associated with significant health problems during childhood and adolescence and with increased risk factors for health problems in adulthood. Cigarette smoking during adolescence appears to reduce the rate of lung growth and the level of maximum lung function that can be achieved. Young smokers are likely to be less physically fit than young nonsmokers; fitness levels are inversely related to the duration and the intensity of smoking. Adolescent smokers report that they are significantly more likely than their nonsmoking peers to experience shortness of breath, coughing spells, phlegm production, wheezing, and overall diminished physical health. Cigarette smoking during childhood and adolescence poses a clear risk for respiratory symptoms and problems during adolescence; these health problems are risk factors for other chronic conditions in adulthood, including chronic obstructive pulmonary disease.

Cardiovascular disease is the leading cause o death among adults in the United States. Atherosclerosis, however, may begin in childhood and become clinically significant by young adulthood. Cigarette smoking has been shown to be a primary risk factor for coronary heart disease, arteriosclerotic peripheral vascular disease, and stroke. Smoking by children and adolescents is associated with an increased risk o early atherosclerotic lesions and increased risk factor for cardiovascular diseases. These risk factors include increased levels of low-density lipoprotein cholesterol increased very-low-density lipoprotein cholesterol increased triglycerides, and reduced levels o

high-density lipoprotein cholesterol. If sustained into adulthood, these patterns significantly increase the risk for early development of cardiovascular disease.

Smokeless tobacco use is associated with health consequences that range from halitosis to severe health problems such as various forms of oral cancer. Use of smokeless tobacco by young people is associated with early indicators of adult health consequences, including periodontal degeneration, soft tissue lesions, and general systemic alterations. Previous reports have documented that smokeless tobacco use is as addictive for young people as it is for adults. Another concern is that smokeless tobacco users are more likely than nonusers to become cigarette smokers.

Among addictive behaviors such as the use of alcohol and other drugs, cigarette smoking is most likely to become established during adolescence. Young people who begin to smoke at an earlier age are more likely than later starters to develop long-term nicotine addiction. Most young people who smoke regularly are already addicted to nicotine, and they experience this addiction in a manner and severity similar to what adult smokers experience. Most adolescent smokers report that they would like to quit smoking and that they have made numerous, usually unsuccessful attempts to quit. Many adolescents say that they intend to quit in the future and yet prove unable to do so. Those who try to quit smoking report withdrawal symptoms similar to those reported by adults. Adolescents are difficult to recruit for formal cessation programs, and when enrolled, are difficult to retain in the programs. Success rates in adolescent cessation programs tend to be quite low, both in absolute terms and relative to control conditions.

Tobacco use is associated with a range of problem behaviors during adolescence. Smokeless tobacco or cigarettes are generally the first drug used by young people in a sequence that can include tobacco, alcohol, marijuana, and hard drugs. This pattern does not imply that tobacco use causes other drug use, but rather that other drug use rarely occurs before the use of tobacco. Still, there are a number of biological, behavioral, and social mechanisms by which the use of one drug may facilitate the use of other drugs, and adolescent tobacco users are substantially more likely to use alcohol and illegal drugs than are nonusers. Cigarette smokers are also more likely to get into fights, carry weapons, attempt suicide, and engage in high-risk sexual behaviors. These problem behaviors can be considered a syndrome, since involvement in one behavior increases the risk for involvement in others. Delaying or preventing the use of tobacco may have implications for delaying or preventing these other behaviors as well.

The Epidemiology of Tobacco Use Among Young People

Overall, about one-third of high-school-aged adolescents in the United States smoke or use smokeless tobacco. Smoking prevalence among U.S. adolescents declined sharply in the 1970s, but this decline slowed significantly in the 1980s, particularly among white males. Although female adolescents during the 1980s were more likely than male adolescents to smoke, female and male adolescents are now equally likely to smoke. Male adolescents are substantially more likely than females to use smokeless tobacco products; about 20 percent of high school males report current use, whereas only about 1 percent of females do. White adolescents are more likely to smoke and to use smokeless tobacco than are black and Hispanic adolescents.

Sociodemographic, environmental, behavioral, and personal factors can encourage the onset of tobacco use among adolescents. Young people from families with lower socioeconomic status, including those adolescents living in single-parent homes, are at increased risk of initiating smoking. Among environmental factors, peer influence seems to be particularly potent in the early stages of tobacco use; the first tries of cigarettes and smokeless tobacco occur most often with peers, and the peer group may subsequently provide expectations, reinforcement, and cues for experimentation. Parental tobacco use does not appear to be as compelling a risk factor as peer use; on the other hand, parents may exert a positive influence by disapproving of smoking, being involved in children's free time, discussing health matters with children, and encouraging children's academic achievement and school involvement.

How adolescents perceive their social environment may be a stronger influence on behavior than the actual environment. For example, adolescents consistently overestimate the number of young people and adults who smoke. Those with the highest overestimates are more likely to become smokers than are those with more accurate perceptions. Similarly, those who perceive that cigarettes are easily accessible and generally available are more likely to begin smoking than are those who perceive more difficulty in obtaining cigarettes.

Behavioral factors figure heavily during adolescence, a period of multiple transitions to physical maturation, to a coherent sense of self, and to emotional independence. Adolescents are thus particularly vulnerable to a range of hazardous behaviors and activities, including tobacco use, that may seem to assist in these transitions. Young people who report that smoking serves positive functions or is potentially useful are at increased risk for smoking. These functions are associated with

bonding with peers, being independent and mature, and having a positive social image. Since reports from adolescents who begin to smoke indicate that they have lower self-esteem and lower self-images than their nonsmoking peers, smoking can become a self-enhancement mechanism. Similarly, not having the confidence to be able to resist peer offers of tobacco seems to be an important risk factor for initiation. Intentions to use tobacco and actual experimentation also strongly predict subsequent regular use.

The positive functions that many young people attribute to smoking are the same functions advanced in most cigarette advertising. Young people are a strategically important market for the tobacco industry. Since most smokers try their first cigarette before age 18, young people are the chief source of new consumers for the tobacco industry, which each year must replace the many consumers who quit smoking and the many who die from smoking-related diseases. Despite restrictions on tobacco marketing, children and adolescents continue to be exposed to cigarette advertising and promotional activities, and young people report considerable familiarity with many cigarette advertisements. In the past, this exposure was accomplished by radio and television programs sponsored by the cigarette industry. Barred since 1971 from using broadcast media, the tobacco industry increasingly relies on promotional activities, including sponsorship of sports events and public entertainment, outdoor billboards, point-of-purchase displays, and the distribution of specialty items that appeal to the young. Cigarette advertisements in the print media persist; these messages have become increasingly less informational, replacing words with images to portray the attractiveness and function of smoking. Cigarette advertising frequently uses human models or human-like cartoon characters to display images of youthful activities, independence, healthfulness, and adventure-seeking. In presenting attractive images of smokers, cigarette advertisements appear to stimulate some adolescents who have relatively low self-images to adopt smoking as a way to improve their own self-image. Cigarette advertising also appears to affect adolescents' perceptions of the pervasiveness of smoking, images of smokers, and the function of smoking. Since these perceptions are psychosocial risk factors for the initiation of smoking, cigarette advertising appears to increase young people's risk of smoking.

Efforts to Prevent the Onset of Tobacco Use

Most of the U.S. public strongly favors policies that might prevent tobacco use among young people. These policies include mandated tobacco education in schools, a complete ban on smoking by anyone on school grounds, further restrictions on tobacco advertising and promotional activities, stronger prohibitions on the sale of tobacco products to minors, and increases in earmarked taxes on tobacco products. Interventions to prevent initiation among young people—even actions that involve restrictions on adult smoking or increased taxes—have received strong support among smoking and nonsmoking adults.

Numerous research studies over the past 15 years suggest that organized interventions can help prevent the onset of smoking and smokeless tobacco use. School-based smoking-prevention programs, based on a model of identifying social influences on smoking and providing skills to resist those influences, have demonstrated consistent and significant reductions in adolescent smoking prevalence; these program effects have lasted one to three years. Programs to prevent smokeless tobacco use have used a similar model to achieve modest reductions in initiation of use. The effectiveness of these school-based programs appears to be enhanced and sustained, at least until high school graduation, by adding coordinated communitywide programs that involve parents, youth-oriented mass media and counteradvertising, community organizations, or other elements of adolescents' social environments.

A crucial element of prevention is access: adolescents should not be able to purchase tobacco products in their communities. Active enforcement of age-at-sale policies by public officials and community members appears necessary to prevent minors' access to tobacco. Communities that have adopted tighter restrictions have achieved reductions in purchases by minors. At the state and national levels, price increases have significantly reduced cigarette smoking; the young have been at least as responsive as adults to these price changes. Maintaining higher real prices of cigarettes provides a barrier to adolescent tobacco use but depends on further tax increases to offset the effects of inflation. The results of this review thus suggest that a coordinated, multicomponent campaign involving policy changes, taxation, mass media, and behavioral education can effectively reduce the onset of tobacco use among adolescents.

Summary

Smoking and smokeless tobacco use are almost always initiated and established in adolescence. Besides its long-term effects on adults, tobacco use produces specific health problems for adolescents. Since nicotine addiction also occurs during adolescence, adolescent tobacco users are likely to become adult tobacco users. Smoking and smokeless tobacco use are associated with other problem behaviors and occur early in the sequence of these behaviors. The outcomes of adolescent smoking

and smokeless tobacco use continue to be of great public health importance, since one out of three U.S. adolescents uses tobacco by age 18. The social environment of adolescents, including the functions, meanings, and images of smoking that are conveyed through cigarette advertising, sets the stage for adolescents to begin using tobacco. As tobacco products are available and as peers begin to try them, these factors become personalized and relevant, and tobacco use may begin. This process most affects adolescents who, compared with their peers, have lower self-esteem and self-images, are less involved with school and academic achievement, have fewer skills to resist the offers of peers, and come from homes with lower socioeconomic status. Tobacco-use prevention programs that target the larger social environment of adolescents are both efficacious and warranted.

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Chapter 2: The Health Consequences of Tobacco Use by Young People

Introduction

The health consequences of tobacco use among adults have been reviewed extensively in previous Surgeon General's reports (Public Health Service [PHS] 1964; U.S. Department of Health and Human Services [USDHHS] 1986b, 1989). Among young people, the short-term health consequences of smoking include respiratory and nonrespiratory effects, addiction to a toxic substance (nicotine), and the associated risk of other drug use. Long-term health consequences of adolescent smoking may be seen in the association between early onset of tobacco use and future (adult) smoking, with concomitant health consequences. Passive (also called "involuntary") smoking during adolescence is also associated with harmful respiratory and nonrespiratory effects. Lastly, the use of smokeless tobacco poses serious health consequences to young people.

Health Consequences of Smoking Among Young People

Introduction

The health effects of cigarette smoking have been the subject of intensive investigation since the 1950s. Extensive evidence, documented in numerous reports of the Surgeon General, has causally linked cigarette smoking to a wide array of health outcomes that extend from annoying symptoms to fatal malignancies (USDHHS 1989). Until recently, this research was largely directed at the effects of smoking on adults. As is discussed in Chapter 3 (see "Age or Grade When Smoking Begins"), the onset and development of cigarette use occur primarily during adolescence (USDHHS 1989); the health consequences of smoking among young people thus have great public health significance. In recent years, investigations of the health effects in school-age youth have reported sufficient data to support conclusions about adverse effects of smoking during childhood and adolescence.

Most of the evidence reviewed here is gathered from epidemiologic studies of young people ranging from 10 through 20 years old. Selected studies that relate to older age groups, yet are relevant to young people, are also included. Emphasis is placed on the respiratory effects of smoking, for which the evidence is abundant. Data on smoking and cardiovascular risk factors and atherogenesis are also addressed, as are the adult health implications of starting to smoke during childhood.

Overview of the Toxicology of Tobacco Smoke

Cigarette smoke is a complex mixture of organic and inorganic compounds generated by the combustion of tobacco and additives. Current knowledge about the physicochemical nature of tobacco smoke is well described in earlier Surgeon General's reports (PHS 1964; USDHHS 1981, 1989). Thousands of individual compounds have been isolated in cigarette smoke, including pharmacologically active agents (e.g., nicotine), toxic agents (e.g., carbon monoxide, hydrogen cyanide, and acrolein), and mutagens and carcinogens (e.g., polycyclic aromatic hydrocarbons).

Cigarette smoke is further classified as mainstream smoke (MS), the smoke drawn through the mouthpiece of the cigarette, and sidestream smoke (SS), the smoke given off by smoldering tobacco between puffs and the smoke diffusing through the cigarette paper and escaping from the burning cone during puffing. Because of the differing combustion conditions under which MS and SS are generated, their chemical compositions differ; in particular, undiluted SS tends to have higher concentrations of many toxic and tumorigenic agents (USDHHS 1986a, 1989). The quantitative yields of tar (the material deposited in a filter as MS is being drawn), nicotine, and carbon monoxide from cigarettes can be assessed by using a smoking machine standardized to a particular pattern of puffing (USDHHS 1989).

Passive smoking refers to nonsmokers' inhalation of tobacco smoke. The term "environmental tobacco smoke" (ETS) is now widely used to refer to the mixture of predominantly SS and exhaled MS that is inhaled by the passive smoker. Passive smoking was the subject of the 1986 Surgeon General's report (USDHHS 1986a); that report reviews in detail the components of ETS, as did a contemporaneously prepared report of the National Research Council (1986). In 1991, the National Institute for Occupational Safety and Health recommended that ETS be regarded as a potential occupational carcinogen and

that exposures to ETS be reduced to the lowest feasible concentration (USDHHS 1991b). A recent monograph by Guerin, Jenkins, and Tomkins (1992) updates and extends these earlier reviews. The U.S. Environmental Protection Agency (USEPA) also recently reviewed the evidence on involuntary smoking and respiratory health (USEPA 1992). These and other health consequences of passive smoking are discussed later in this chapter.

Many of the components of SS and MS have been identified in ETS. On the other hand, ETS is an inherently dynamic mixture that changes in physical and chemical characteristics as it ages and reacts with other pollutants in indoor air and with surfaces (USDHHS 1986a; Guerin, Jenkins, Tomkins 1992). The 1986 Surgeon General's report concluded, however, that ETS was sufficiently close to MS and SS to permit generalization of the evidence on the health consequences of active smoking to passive smoking (USDHHS 1986a).

The human body is most susceptible to these health consequences along cigarette smoke's path of ingress through the respiratory tract. The respiratory tract includes the upper airway (nose, oropharynx, and larynx) and the lung (airways and the parenchyma). The airways are lined by an epithelium that varies in form and function at different levels of the respiratory tract. The parenchyma includes the alveoli pulmonis (the delicate gas-exchanging surface of the lung) and the interstitium (the location of the blood and lymphatic vessels and of the lung's supporting connective tissue).

The effects of active[1] cigarette smoking on these structures of the lung and on many physiological functions of the lung have been extensively studied (USDHHS 1984, 1990; Bates 1989). Changes in lung physiology attributable to smoking include the weakening of an individual's defenses against infectious organisms and inhaled particles and gases, changes in the numbers and types of cells present within the lung, and the activation of potentially damaging proteolytic enzymes and the inactivation of the proteins that inhibit them. Many of these effects of smoking have been demonstrated in young adult smokers who have served as volunteer research subjects (USDHHS 1984).

The effects of smoking on lung structure and function have been demonstrated repeatedly in young adult smokers (USDHHS 1984; Bates 1989). Studies using spirometry, tests of small airway function, and lung volume measurements have shown a higher frequency of abnormalities in smokers than nonsmokers (USDHHS 1984; Bates 1989). Effects of smoking on lung structure, particularly the small airways, have been found in smokers in their mid-twenties. Niewoehner, Kleinerman, and Rice (1974) examined peripheral airways of 20 nonsmokers and 19 smokers who had died from nonrespiratory causes at an average age of 25. A characteristic lesion, termed "respiratory bronchiolitis," was found in all 19 of the smokers but in only 5 of the nonsmokers. The affected small airways of the smokers demonstrated an inflammatory process consisting of aggregates of pigment-containing macrophages with edema, fibrosis, and epithelial hyperplasia in adjacent bronchioles and alveoli.

These observations on the effects of smoking in young people are consistent with current concepts of pathogenesis and natural history in adult smokers (USDHHS 1984, 1990). Severe chronic airflow obstruction, sufficient to result in a clinical diagnosis of chronic obstructive pulmonary disease (COPD), follows sustained smoking and lung injury with progressive loss of respiratory function through adulthood. In smokers who develop COPD, decline of lung function at a rate well beyond that associated with aging alone eventually leads to impairment. Changes in lung function can be demonstrated in young adult smokers; these losses are consistent with the histopathologic evidence that the small airways of young smokers are damaged (USDHHS 1984).

Epidemiologic Evidence of Respiratory Effects

Respiratory Symptoms

The cardinal symptoms of respiratory tract injury and disease are cough, sputum production, wheezing and dyspnea (or shortness of breath). In epidemiologic studies of respiratory diseases, symptoms are usually discovered through responses to a standardized questionnaire (Samet 1978). In adults, the occurrence of cough and phlegm is causally associated with cigarette smoking; the frequency of the symptoms rises with the number of cigarettes smoked per day (USDHHS 1984). In some studies, wheezing is also more frequent in adult smokers than in adults who have never smoked (Schenker, Samet, Speizer 1982). The frequency of dyspnea rises as the extent of smoking-related impairment of lung function increases (Samet 1978).

Questionnaire-based epidemiologic studies of children and adolescents document that smoking is also a cause of respiratory symptoms in preteen and teenage regular smokers (those who smoke at least weekly). Studies conducted from the 1960s through the 1980s involving thousands of children provide consistent evidence that smoking is associated with the occurrence of cough and phlegm (Table 1; see Table 31 in Chapter 3 for additional data). In several studies, smoking also increased the frequency of wheezing and dyspnea. These associations have been found in studies conducted in the

[1]Unless otherwise indicated, "smoking" will hence refer to active smoking.

United States, the United Kingdom, New Zealand, and Scandinavia and at levels of smoking as low as one cigarette per week.

In one of the first studies on smoking and respiratory symptoms in children, Holland and Elliott (1968) administered a questionnaire concerning respiratory symptoms and cigarette smoking to all children in schools in four areas of southeast England. Smoking education was then provided to half of the schools, and the questionnaire was readministered one year later. Although the intervention had no effect on the prevalence of smoking, the study documented that smoking in childhood was associated with cough and phlegm and that these symptoms were reduced in those who had stopped smoking.

Many later studies continued to show that smoking increased the frequency of respiratory symptoms in children and adolescents. In the United States, research with high school students (Addington et al. 1970; Seely, Zuskin, Bouhuys 1971; Rush 1974) and college students (Peters and Ferris 1967) provided early evidence of adverse effects of smoking on young smokers. Large studies of schoolchildren (including preteens) in the United Kingdom showed that symptom rates were increased by smoking. Bewley, Halil, and Snaith (1973) reported that the frequency of cough was increased in boys and girls no older than 11.5 years who reported smoking at least one cigarette per week. Other studies in the United Kingdom and the United States found further evidence of the effects of smoking on symptom frequency in children of similar ages (Bewley and Bland 1976; Charlton 1984; see Table 31 in Chapter 3).

The health effects of smoking among adolescents may be confounded by a history of passive smoking if the parents of an adolescent smoker also smoke. However, in a study of 5,835 secondary schoolchildren in Derbyshire (United Kingdom), students who smoked at least one cigarette per week persisted in having an increased risk for cough and dyspnea even after parental smoking was taken into account (Bland et al. 1978).

Control for other potential confounding or mediating factors varies among the investigations. Residence location, a surrogate for exposure to ambient air pollution, was considered in several of the studies (Bewley, Halil, Snaith 1973; Bewley and Bland 1976), and a study of 20-year-olds (Colley, Douglas, Reid 1973) controlled for socioeconomic status.

Lung Function

Numerous cross-sectional studies of adults have shown that cigarette smokers have a lower level of lung function, as assessed by tests of lung mechanics and gas exchange, than persons who have never smoked (USDHHS 1984; Bates 1989). Longitudinal studies show that smoking speeds the age-related decline of lung function. The most abundant evidence describes changes in lung function as assessed by spirometry, or the measure of the volume of air entering and leaving the lungs. One measure of scientific and clinical interest obtained through spirometry is the forced expiratory volume in one second (FEV_1), the volume of air blown out during the first second of the forced vital capacity maneuver. FEV_1 increases with lung growth and development during childhood, and rises even more steeply with the growth spurt of adolescence (Tager et al. 1988; Sherrill et al. 1992). In persons who have never smoked, FEV_1 begins to decline from a maximum at some time during the third or fourth decades of life (Beck, Doyle, Schachter 1982; Tager et al. 1988). In smokers, the age-related decline commences at a younger age and proceeds at a steeper average rate (Beck, Doyle, Schachter 1982; USDHHS 1984; Tager et al. 1988). When people stop smoking, their average decline gradually returns to the rate observed in those who never smoked (USDHHS 1990).

Cross-sectional and longitudinal data show that smoking also adversely affects lung function in children and adolescents (Table 2). The evidence comes principally from spirometry studies of high school students, although one of the first studies to show reduced lung function in young people involved college seniors (Peters and Ferris 1967). In these studies, impaired lung function has been primarily indicated through reduced flow rates after 50 percent or more of the vital capacity has been exhaled. This effort-independent, latter portion of the flow-volume loop is sensitive to abnormalities of the lung's small airways and the lung parenchyma (Bates 1989). Several studies have also found that smokers have a reduced peak expiratory flow rate (PEFR) (Table 2). This effort-dependent portion of the flow-volume loop is more sensitive to abnormal function of the lung's larger airways than of its small airways (Bates 1989).

Among the first researchers to study smoking among younger people were Peters and Ferris (1967), who obtained spirometric and peak-flow data from 124 Harvard College seniors. Smokers had lower (although not significantly) FEV_1 than persons who had never smoked. Spirometric flow rates and PEFR were significantly lower in the smokers. In an early study involving high school students, Seely, Zuskin, and Bouhuys (1971) found evidence of abnormal function of the small airways in both boys and girls who smoked. Subsequent cross-sectional studies of teenagers have tended to confirm that smokers have reduced lung function, as assessed by spirometry or PEFR measurement.

More recent, longitudinal data show that smoking reduces the rate of lung growth, as would be anticipated

Table 1. Published studies of the effects of smoking on respiratory symptoms among young people, various countries, 1965–1983

Reference*	Location/year	Study population
Peters and Ferris 1967	Massachusetts, 1965	124 Harvard College seniors
Holland and Elliott 1968	England, 1965–1966	9,786 13- and 14-year-olds in 1965; 9,433 in 1966
Addington et al. 1970	Oklahoma§	557 high school students, (grades 9–12) aged 13–19 years
Seeley, Zuskin, Bouhuys 1971	Connecticut*	195 male and 170 female high school students aged 15–19 years
Bewley, Halil, Snaith 1973	England, 1971	8,682 schoolchildren aged 10 and 11 years
Colley, Douglas, Reid 1973	United Kingdom, 1966	3,899 persons aged 20 years sampled from 1946 birth cohort study
Rush 1974	New York, 1968	12,595 high school students aged 13–18 years

*Listed chronologically by publication date.
§Year not provided.

Symptoms	Prevalence (%) by smoking status	
	Never smoker	Smoker†
Phlegm ≥ 3 months/yr	2.4	26.5‡
Breathlessness	2.4	20.5‡
Wheezing (apart from colds)	7.3	31.3‡
Colds go to chest	4.9	31.3‡

General findings: Increased cough and phlegm in smokers of ≥ 1 cig/week versus never smokers. Dose–response evident. Prevalence of cough and phlegm dropped among smokers who quit smoking between 1965 and 1966.

	Never smoker	Smoker†
Daily cough ≥ 3 months	4	10
Daily phlegm ≥ 3 months	3	9
Dyspnea when hurrying	16	30
Chest cold for 1 week	22	30
Wheezing or asthma	12	13

	Number of cigarettes smoked per day				
	0	< 1	1–10	11–20	> 20
Cough	12.0	15.8	18.1	27.8	64.7
Phlegm	8.3	5.8	19.4	31.9	58.8
Shortness of breath	9.3	13.5	13.5	36.1	58.8

	Never smoker	Smoker△
Morning cough		
Boys	5.4	18.2
Girls	5.9	19.8
Cough 3 months		
Boys	3.8	15.4
Girls	3.5	12.1

	Never smoker	Ex-smoker	Present smoker
Cough (day or night in winter)			
Boys	5.2	7.1	13.9
Girls	6.5	10.5	16.0

	Nonsmoker	Ex-smoker	Smoker		
			Number of cigarettes smoked per day		
Cough ≥ 3 months/yr¶			≤ 1–9	10–14	≥ 15
Boys	2.9	4.5	9.2	16.2	29.0
Girls	4.4	6.0	12.0	23.1	35.9

†At least one cigarette daily for the past year.

‡$p < 0.01$.

△Smoking at least one cigarette weekly. Percentages combine data reported separately in authors' Table 4 for urban and rural children.

¶For white children only.

Table 1. Continued

Reference	Location/year	Study population
Stanhope and Prior 1975	New Zealand, 1972	Maori and European high school students aged 13–15 years
Bewley and Bland 1976	England, 1971	5,355 schoolchildren aged 10–12 years
Bland et al. 1978	England, 1974	5,835 schoolchildren; first-year level in secondary school
Weiss et al. 1980	Massachusetts, 1975	650 children aged 5–9 years, population sample
Kujala 1981	Finland, 1976	1,075 male military recruits, mean age = 20 years
Charlton 1984	England, 1982	15,709 students aged 8–19 years
Adams et al. 1984	England, 1975–1979	405 secondary schoolchildren
Rimpela and Rimpela 1985	Finland, 1983	4,279 16- and 17-year-olds in a national sample
Oechsli, Seltzer, van den Berg 1987	California, 1977–1979	1,445 children in a cohort study

**Smoking at least one cigarette weekly. Percentages combine data reported separately in authors' Table V for urban and rural children.
††RR = Relative risk for children smoking ≥ one cigarette weekly versus children who had never smoked, adjusted for parental smoking.
‡‡Smoking at least one cigarette weekly.

Symptoms	Prevalence (%) by smoking status		

General finding: Cough grade, phlegm grade, and loose cough sign significantly associated with smoking.

	Never smoker	Smoker**	RR††
Morning cough			
Boys	8.3	16.3	5.9
Girls	8.5	28.6	6.8
Cough 3 months			
Boys	7.2	13.4	2.4
Girls	6.0	10.7	2.6

	Never smoker	Smoker‡‡	RR§§
Morning cough			
Boys	3.1	19.2	5.9
Girls	1.8	13.5	6.8
Cough day or night			
Boys	20.4	46.5	2.4
Girls	18.5	47.3	2.6
Breathlessness			
Boys	11.8	34.9	2.9
Girls	16.5	39.2	2.3

General findings: Persistent wheezing reported for 13.8% of ever smokers and 9.7% of never smokers; difference not significant.

	Nonsmoker^^	Ex-smoker¶¶	Smoker***
Cough all day	1	2	8
Phlegm all day	1	1	7
Wheezing	5	13	22

	Number of cigarettes smoked per day		
Frequent cough	0	1–6	> 6
Boys			
Age 11–13	23	32	42
Age ≥ 14	9	16	29
Girls			
Age 11–13	19	34	49
Age ≥ 14	9	18	32

General findings: Increased risk of cough, dyspnea, and phlegm.

	Never smoker	Low-tar smoker†††	Medium-tar smoker‡‡‡
Morning phlegm	2.7	7.6	11.4
Morning cough	6.3	20.7	20.5
Phlegm day or night	5.2	13.8	13.2
Cough day or night	19.1	43.9	40.6

General findings: Starting smoking associated with bronchitis and pneumonia.

§§RR = Relative risk for children smoking at least one cigarette weekly versus children who had never smoked.
^^Nonsmoker = Never smoking and smoking not more than one cigarette daily for ≤ one year.
¶¶Ex-smoker = Smoking one month or more before date of the interview.
***Smoker = Smoking ≥ 1g of tobacco daily; one cigarette was estimated to contain 1g of tobacco.
†††Smoking daily, cigarettes < 10mg of tar.
‡‡‡Smoking daily, cigarettes 10–18mg of tar.

Table 2. Published studies of the effects of smoking on lung function among young people, various countries, 1965–1981

Reference*	Location/year	Study population	Findings†	Comment
Peters and Ferris 1967	Massachusetts, 1965	124 Harvard College seniors	Significant reduction in spirometric flow rates when comparing NS with persons smoking a pack a day for four years during college; dose response with amount smoked.	Age distribution not given, nonsignificant reduction for FEV_1.
Addington et al. 1970	Oklahoma‡	140 male and 417 female high school students aged 13–19 years (grades 9–12)	No significant difference in VC and FEV_1 when comparing NS with smokers of ≥ 1 cig/day for last year.	Age distribution not given; no adjustment for height in analysis of spirometric data.
Seely, Zuskin, Bouhuys 1971	Connecticut‡	195 male and 170 female high school students aged 15–19 years	From MEFV curves, V_{50} and V_{75} significantly reduced in boys smoking > 15 cigs/day and girls smoking > 10 cigs/day, when compared with NS.	Age distribution not given, nonsignificant reduction for FEV_1.
Lim 1973	Nebraska‡	50 male and 50 female high school students aged 15–18 years	No significant difference in FEV_1 and FVC when comparing NS with smokers of ≥ 10 cigs/day for 1 year; 10 of 50 smokers abnormal by partial MEFV curves.	None
Comstock and Rust 1973	Nationwide, 1970–1971	3,409 U.S. Navy recruits, median age = 19 years	PEFR lower in smokers (99.5% predicted) than in nonsmokers (100.7% predicted).	No definition of smoker, nonsmoker; tests of statistical significance not provided.

*Listed chronologically by publication date.

†NS = never smoker; FEV_1 = forced expiratory volume in one second; VC = vital capacity; MEFV = maximal expiratory flow volume; V_{50} = flow rate at 50% of vital capacity; V_{75} = flow rate after exhalation of 75% of vital capacity; FVC = forced vital capacity; PEFR = peak expiratory flow rate; FEF_{25-75} = forced expiratory flow from 25% to 75% of FVC.

‡Year not provided.

Table 2. Continued

Reference	Location/year	Study population	Findings†	Comment
Backhouse 1975	United Kingdom‡	195 boys at a detention center, mean age = 18 years	PEFR on arrival dropped significantly with daily smoking amount; significant improvement during 8-week stay while unable to smoke	None
Walter, Nancy, Collier 1979	India‡	102 male medical students aged 19–21 years	Significantly lower PEFR and spirometric flows when comparing NS with smokers of > 10,000 cigarettes per lifetime.	Values for smokers of ≤ 10,000 cigarettes were between those of nonsmokers and heavy smokers.
Woolcock et al. 1979	Australia, 1971–1980	10,898 school children, mean ages = 8.9 years for primary school and 12.6 years for high school groups	No overall effect of smoking on spirometric values in 1974 data; decreased lung growth in smoking boys who had had bronchitis before age 2 years.	See text for review of longitudinal findings.
Weiss et al. 1980	Massachusetts, 1975	650 children aged 5–9 years, population sample	Smoking not associated with FEF_{25-75}	Only 58 children reported ever smoking; see text for longitudinal findings.
Kujala 1981	Finland, 1976	1,075 male military recruits, mean age = 20 years	Significantly reduced FEV_1 and spirometric flows when comparing NS with smokers at interview.	None
Spinaci et al. 1985	Italy, 1980–1981	1,266 male and 1,119 female 6th graders, mean age = 11 years	Smoking negatively associated with FEF_{25-75} and V_{max}	Definition for smoking not given; lung function data not provided.

from the findings from cross-sectional studies. Beck, Doyle, and Schachter (1982) examined white residents of Lebanon, Connecticut, in 1972 and 1978. Among male and female subjects aged 15 through 24 in 1972, smoking had reduced the increment of FEV_1 during the six-year follow-up interval.

In a 10-year study in Sydney, Australia, Woolcock et al. (1984) periodically measured lung function in an initial cohort of 11,497 schoolchildren. Two groups of children were included: a younger cohort that was 8.9 years of age on average at enrollment and an older cohort aged 12.6 years on average at enrollment. The investigators followed up the cohort annually, measuring respiratory function and assessing symptoms, illnesses, and smoking. A small number of children were studied more intensively with the single-breath nitrogen test. The effect of smoking was examined only in the older cohort. Cross-sectional assessment of these data showed that at 50 percent of vital capacity, smokers tended to have lower maximal expiratory flow than nonsmokers. For example, adolescents who smoked at least 10 cigarettes per week had about a 5 percent lower expiratory flow rate than nonsmokers. The investigators concluded that abnormalities attributable to smoking were found in adolescents as young as age 14 and as soon as one year after beginning to smoke at least 10 cigarettes per week. They also concluded that smoking was more harmful for children and adolescents who had a history of respiratory illness, particularly asthma.

A cohort study of children in East Boston, Massachusetts, has been informative on the effects of passive and active smoking on lung function (Tager et al. 1979, 1983, 1985, 1988). In 1974, the study enrolled a cohort of children aged five through nine who were sampled from schools in East Boston. The families of these children were then invited to participate in the initial survey and in periodic follow-up examinations that included a respiratory questionnaire and spirometry.

Several relevant longitudinal analyses of the East Boston data have been reported (Tager et al. 1985, 1987, 1988). Using data from the first seven follow-up examinations, Tager et al. (1985) described the effect of smoking on the growth rates of FEV_1 and on forced expiratory flow (FEF) from 25 to 75 percent of forced vital capacity (FEF_{25-75}) in a group of 669 subjects aged 5 through 19 years at enrollment. Using a Markov-type autoregressive model, researchers found significant effects of smoking on both measures of lung function. The model predicted that a child's smoking, beginning at age 15 and continuing through age 20, would reduce FEV_1 to 92 percent of the expected value and FEF_{25-75} to 90 percent of the expected value. A subsequent analysis using a nonparametric curve-smoothing method on these same data showed that male smokers had a smaller increase of FEV_1 at the end of the growth phase (a suggestion of a lower maximum lung function) than males who had not smoked; those who continued to smoke into early adulthood also showed no evidence of the plateau observed in never smokers before lung function began to decline. Similar findings were reported for females.

Relevant information is also available from a community population study in Tucson, Arizona (Lebowitz and Holberg 1988). The Tucson cohort was derived from a population sample of 325 non-Hispanic white residents, originally sampled in 1972 when they were an average age of 8.8 years. Like the East Boston study, the Tucson study was directed primarily at passive smoking but also gathered information on active smoking by measuring FEV_1 and FEF_{25-75}. The Tucson study found effects of comparable magnitude with those observed in the East Boston study. Although these effects did not reach statistical significance in the Tucson data, they were in the same direction as those from East Boston, and the sample population was only half the size.

Sherrill et al. (1992) examined the longitudinal effects of active and passive smoking on lung function in a cohort of New Zealand children observed from ages 9 through 15. Active smoking did not have statistically significant effects on FEV_1, vital capacity, or FEV_1/vital capacity (percent), but the numbers of regular smokers were small. By age 15, 43 percent reported occasional smoking (during the last year but not every day), but only 10 percent were daily smokers (smoking any number of cigarettes on a daily basis).

Jaakkola et al. (1991) carried out an eight-year longitudinal study of lung function in a cohort of young adults aged 15 through 40 at enrollment. Of 1,044 enrolled, 391 were subsequently followed. Smoking was found to have a significant effect on change in FEV_1 during the study period, but the results were not reported by age interval.

Respiratory Morbidity

In adults, smoking is associated with increased morbidity, as indexed by such measures as use of outpatient medical services and absenteeism from work, and with increased respiratory morbidity, as indexed by frequency or severity of respiratory infections (USDHHS 1990). Because smoking has been shown to alter immune and inflammatory responses (U.S. Department of Health, Education, and Welfare [USDHEW] 1979b), these effects on an individual's defenses could

plausibly lead to increased frequency and severity of respiratory infections in smokers.

Studies involving a wide age range of young people indicate that smoking increases respiratory morbidity (Table 3). A number of these studies compared medical care by smokers and nonsmokers in settings where all medical care was obtained at a single clinic. In one of the earliest studies, Haynes, Krstulovic, and Bell (1966) examined the numbers of diagnoses for respiratory tract illnesses among male students (aged 14–19 years) at a preparatory school. Nearly half of the students were smokers. All respiratory illnesses were more common in the smokers; the increase was greatest for the illnesses considered "severe." The findings of studies involving student nurses (Parnell, Anderson, Kinnis 1966) and military cadets (Finklea et al. 1971) were similar.

A series of studies have included military recruits as subjects (Table 3); their ages ranged from 18 through 22. In the study of Pollard et al. (1975), the rates of respiratory diagnoses were not significantly different between smokers and nonsmokers. In the more recent study of military recruits by Blake, Abell, and Stanley (1988), self-report of smoking was associated with increased risk for diagnosis of an upper respiratory tract infection during a 13-week basic training period. Kark and Lebiush (1981) and Kark, Lebiush, and Rannon (1982) examined attack rates for influenza and influenza-like illnesses in Israeli military recruits and found that smoking was associated with an increased attack rate in both male and female recruits.

Recently, in a study that examined adolescents and young adults who had sickle cell anemia, Young et al. (1992) found a strong relationship between cigarette smoking and acute chest syndrome. In sickle cell anemia patients, acute chest syndrome is characterized by fever, cough, chest pain, leukocytosis, and pulmonary infiltrates in the chest radiograph. All smokers in this study had a history of acute chest syndrome, whereas 65 percent of the nonsmokers did. Smoking also appeared to increase the frequency of sequelae of sickle cell lung disease.

A study in the United Kingdom (Charlton and Blair 1989) associated smoking with increased absenteeism from school among 2,885 children aged 12 and 13 years. Children who on an initial questionnaire reported regular smoking were more likely than nonsmokers to be absent when a follow-up questionnaire was administered four months later. The authors interpreted these findings as showing a higher rate of minor ailments in children who smoked; however, the design could not exclude other plausible explanations (such as truancy) for the difference. In a survey of adolescents invited for an overall evaluation in three general practices in the United Kingdom, smokers reported a higher prevalence of health problems than nonsmokers (25 percent vs. 16 percent, $p = .06$) (Townsend et al. 1991).

Epidemiologic Evidence of Nonrespiratory Effects

Cardiovascular Disease

In adults, cigarette smoking is a cause of coronary heart disease, arteriosclerotic peripheral vascular disease, and stroke (USDHHS 1989). Although these diseases rarely occur in children and adolescents, autopsy studies of young male victims of combat during the Korean and Vietnam conflicts and community-based autopsy studies of adolescents and young adults have shown that atherosclerosis begins in childhood and may become clinically significant in young adulthood (McNamara et al. 1971; Enos, Holmes, Beyer 1986; Strong 1986).

Several autopsy-study series link cigarette smoking to the occurrence and extent of atherosclerosis in young adults. Strong and Richards (1976) described the association of cigarette smoking with atherosclerosis in 1,320 men from the New Orleans area. In the youngest group (aged 25 to 34 years), the development of atherosclerosis in the coronary arteries and the abdominal aorta was consistently greater with higher levels of smoking.

More recently, an eight-community study by the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group (1990) found associations of smoking with atherosclerosis in 390 males aged 15 through 34 years who died of violent causes (e.g., accidents, homicides, suicides). In this study, lipids were measured in postmortem serum, and smoking was assessed by the level of serum thiocyanate. After controlling for lipid levels, age, and race, a multiple regression analysis revealed a significant association between smoking and atherosclerosis (i.e., having raised lesions greater than or equal to 5 percent of the intimal surface area) in the abdominal aorta. A multiple logistic analysis controlling for the same factors found that smoking was a significant predictor of atherosclerosis in both the abdominal aorta and the right coronary artery.

The Bogalusa Heart Study is an epidemiologic study of cardiovascular disease risk factors encountered from birth through age 26. Among deceased subjects whose average age was 18 years, cigarette smoking was not associated with aortic fatty streaks or involvement of the coronary arteries with atherosclerosis (Newman et al. 1986; Freedman et al. 1988). However, in subjects who

Table 3. Published studies of the effects of smoking on respiratory morbidity among young people, various countries, 1963–1987

Reference*	Location/year	Study population
Haynes, Krstulovic, Bell 1966	New Jersey†	191 male prep school students aged 14–19 years
Parnell, Anderson, Kinnis 1966	Canada, 1963–1964	175 senior student nurses
Finklea et al. 1971	South Carolina, 1968–1969	1,900 college students
Pollard et al. 1975	Florida, 1971–1972	1,100 U.S. Navy recruits, most aged 18–22 years
Kark and Lebiush 1981	Israel, 1979	Female military recruits, mean age = 18.5 years
Kark, Lebiush, Rannon 1982	Israel, 1978	Male military recruits, mean age = 18.5 years
Blake, Abell, Stanley 1988	Georgia, 1982	1,230 Army recruits, most aged < 22 years
Charlton and Blair 1989	England, 1987	2,885 schoolchildren aged 12 and 13 years
Schwartz and Zeger 1990	California†	100 student nurses

*Listed chronologically by publication date.
†Year not provided.

Health effect	Prevalence (%) by smoking status		
	Nonsmoker	Occasional smoker[†]	Regular smoker[§]
Annual illness rates[Δ] / 10 students			
All respiratory	11.0	16.0	22.0
Severe respiratory	1.4	3.6	5.4

	Nonsmoker	Smoker
Illness incidence[¶] (per 1,000 days)		
All respiratory	6.6	10.6
Upper respiratory	5.2	7.5
Lower respiratory	1.4	3.2

	Number of cigarettes smoked per day		
	0	≤ 1 pack	> 1 pack
Incidence rate[**] (per 100 school years)			
Upper respiratory			
Outpatient	52.5	59.9	67.0
Hospital	7.6	12.0	10.2
Lower respiratory			
Outpatient	2.5	3.0	6.8
Hospital	0.4	0.7	0.9

	Number of cigarettes smoked per day			
	0	≤ 10	10–19	≥ 20
Rate of outpatient visits[††] for respiratory episodes (per 1,000 recruits)				
Febrile	249	256	257	222
Afebrile	436	469	562	560

	Occasional/regular smoker[§§]	Never/past smoker
Attack of influenza-like morbidity[‡‡]	60%	40%

	Number of cigarettes smoked per day			
	0	≤ 10	11–20	> 20
Influenza morbidity[ΔΔ] during an outbreak				
Affected	47.2	62.9	67.7	71.8
Severe cases	30.1	42.9	51.6	53.5

General Findings: Relative risk = 1.46 for upper respiratory infection for smokers versus nonsmokers. Illnesses ascertained by visits to clinics.

General Findings: Smoking associated with increased absence from school: odds ratio = 1.29 for sometimes smokers and 3.09 for regular smokers (compared with never smokers).[††]

General Findings: Smoking significantly associated with incidence of cough and phlegm. Current amount smoked significantly predicted duration of an episode of phlegm or chest discomfort.

[†]Smoked at least 1 cigarette or pipe per week.

[§]Smoked at least 1 cigarette or pipe per day.

[Δ]Illness rates based on infirmary visits during a school year.

[¶]Illness incidence based on records of the health service.

[**]Incidence rates based on self-administered questionnaire.

[††]Respiratory-related (similar symptoms) visits to dispensary, with one week grouped.

[‡‡]Based on self-administered questionnaire.

[§§]These categories were not defined.

[ΔΔ]Illness occurrence based on medical records and serology.

died after age 20, smoking appears to have been related to atherosclerosis (Berenson et al. 1992).

Smoking among young people has been associated with serum lipid profiles in a pattern predictive of increased risk for cardiovascular diseases. In a published meta-analysis of studies on children who smoke, Craig et al. (1990) found that among 8- through 19-year-olds, smoking increased levels of low-density lipoprotein cholesterol by 4 percent, triglycerides by 12 percent, and very-low-density lipoprotein cholesterol by 12 percent. Levels of high-density lipoprotein (HDL) cholesterol were reduced by 9 percent. These changes were comparable to—and of larger magnitude than—those observed in smoking adults.

Physical Fitness

Even among young people trained as endurance runners, smoking appears to compromise physical fitness in levels of both performance and endurance. Cigarette smoking reduces the oxygen-carrying capacity of the blood and increases both heart rate and basal metabolic rate—changes that counter the benefits of physical activity in a direct relation to the duration of smoking and the number of cigarettes regularly smoked (Royal College of Physicians of London 1992). In a study of 19-year-old army conscripts (N = 6,500), those who smoked ran a significantly shorter distance in 12 minutes and took significantly longer to sprint 80 meters than their nonsmoking counterparts (Marti et al. 1988). In the same study, the smokers among 4,100 joggers in a 16-kilometer race were consistently slower than the nonsmokers.

Young adult smokers also have chronic, mild adverse cardiovascular physiologic changes, including diminished exercise performance on standard treadmill testing and blunted heart rate response to exercise (Sidney et al. 1993). The left ventricular mass is increased in young adult smokers, and their resting heart rates are two to three beats per minute more rapid than nonsmokers' (Gidding et al. 1992).

Health Outcomes in Pregnancy

Cigarette smoking during pregnancy has been linked with a variety of adverse outcomes (USDHHS 1989, 1990). Early reports of the Surgeon General (USDHEW 1971, 1973, 1979a) concluded that smoking by a mother during pregnancy retards fetal growth and may cause fetal death late in pregnancy as well as infant mortality. The 1977–1978 report (USDHEW 1979a) further concluded that smoking during pregnancy has dose–response relationships with abruptio placenta, placenta previa, bleeding during pregnancy, premature and prolonged rupture of placental membranes, and preterm delivery. The comprehensive reviews of the 1979 and 1980 reports (USDHEW 1979a; USDHHS 1980) concluded that the risk of spontaneous abortion increases with the amount of smoking and that the risk of sudden infant death syndrome (SIDS) is increased by maternal smoking. A more recent study confirms the increased risk of SIDS with maternal smoking (Schoendorf and Kiely 1992). Impaired fertility was linked to smoking in the 1980 report (USDHHS 1980). These adverse health effects of smoking on reproduction have not been specifically investigated in young women in the 10- through 20-year age range.

Epidemiologic Evidence of the Health Effects of Passive Smoking

The health effects of passive smoking were comprehensively addressed in the 1986 report of the Surgeon General (USDHHS 1986a) and in a report of the National Research Council (1986). These reviews and subsequent reports (Samet, Cain, Leaderer 1991; USEPA 1992) have demonstrated that exposure to parental smoking during childhood significantly increases the occurrence of lower respiratory illnesses during the first years of life, increases the frequency of chronic respiratory symptoms, and reduces the rate of lung growth during childhood and adolescence. Evidence is accumulating to suggest that smoking by parents increases the severity of childhood asthma (USDHHS 1991b; Samet, Cain, Leaderer 1991), as indicated by the need for medication and hospital treatment. SIDS, the most common cause of death in the first year of life, has been linked to parental smoking in several epidemiologic studies. Children of parents who smoke have a twofold increased risk of dying of SIDS; this relationship appears to be dose-related (Schoendorf and Kiely 1992; Malloy et al. 1988).

The evidence on passive smoking and respiratory health was recently reviewed by the USEPA (1992). This review confirmed that ETS is causally linked to lung cancer. Janerich et al. (1990) noted that approximately 17 percent of lung cancers among nonsmokers can be attributed to high levels of ETS during childhood and adolescence. The USEPA report also concluded that exposure to ETS causes lower respiratory illness in infants and young children; this finding is stronger than that of the 1986 Surgeon General's report, which did not characterize this association as causal. The agency's report also inferred from its data that childhood exposure to ETS reduced lung function, increased respiratory symptoms, caused middle ear effusion, and exacerbated asthma. For example, the report estimated that ETS exposure exacerbates symptoms of asthma in about 20 percent of the two million to five million asthmatic children in the United States. The report also hypothesized that ETS may be associated with the onset of asthma.

Many chronic changes in cardiovascular physiology have been observed in children exposed to ETS. These changes include lower HDL cholesterol, increased carboxyhemoglobin concentration, and increased red-cell 2.3-diphosphoglycerate, as well as physiologic response suggesting mild, chronic hypoxemia (Moskowitz et al. 1990). ETS is also known to increase platelet aggregation (Glantz and Parmley 1991).

The effect of peer smoking—as a source of ETS—on nonsmoking children has not been studied but may also be a health risk.

Adult Health Implications of Smoking Among Young People

Respiratory Diseases

As was discussed previously, sustained smoking during adulthood is associated with the development of COPD and the progressive loss of lung function (USDHHS 1984, 1990). Evidence suggests that smoking during childhood may increase the risk for developing COPD in adulthood as well as at an earlier age. The adult who smoked during childhood may have experienced early inflammatory changes—childhood smoking is known to reduce lung growth—and thereby not attained the level of function achieved during the normal growth and development of the lungs. Any age-related decline in lung function during adulthood would thus start from a lower level—and might begin at a younger age—than declines observed in adults who have never smoked. In fact, the proportionate impeding effect of childhood smoking on lung growth greatly exceeds the loss of lung function associated with smoking during adulthood (Tager et al. 1985, 1988).

If one or both parents of an adolescent smoke, the effects of parental smoking on early childhood respiratory illnesses and on the growth of lung function may increase the risk of COPD. Illnesses in the lower respiratory region during childhood are a suspected risk factor for COPD (Samet, Tager, Speizer 1983), and passive smoking reduces the rate at which lung function grows (USDHHS 1986a).

Cardiovascular Disease

In adults, cigarette smoking has been causally associated with coronary heart disease, arteriosclerotic peripheral vascular disease, and stroke (USDHHS 1983, 1989). Smoking contributes to increased risk for coronary heart disease probably through at least five interrelated processes, including the development of atherosclerosis (USDHHS 1990). It is likely that the earlier the age at which one starts to smoke, the earlier the onset of coronary heart disease. The recent evidence from the PDAY Research Group shows more atherosclerosis in young smokers than in young nonsmokers. The unfavorable effects of smoking on lipid levels in children may contribute to the development of atherosclerosis in young adulthood.

Cancer

The multistage concept of carcinogenesis implies that the risk of smoking-related cancers is strongly dependent on the duration and intensity of smoking (Armitage and Doll 1954; Doll 1971; Taioli and Wynder 1991). The relevant epidemiologic data and mathematical analyses are most abundant for lung cancer. Both epidemiologic and experimental evidence suggest that the risk for lung cancer varies more strongly with the duration of cigarette smoking than with the number of cigarettes smoked (Peto 1977; Doll and Peto 1978). Analysis of data from a cohort study of British doctors showed that lung cancer incidence increased with the fourth or fifth power of duration of smoking but with the second power of number of cigarettes smoked daily (Doll and Peto 1978). Although these data can be adequately described by alternative mathematical models that give lesser weight to duration (Moolgavkar, Dewanji, Luebeck 1989), the dependence of lung cancer risk on duration of smoking implies that starting smoking at an earlier age increases the potential number of life-years of smoking and therefore increases lung cancer risk. If one assumes, for example, that lung cancer risk rises exponentially as a function of the duration of smoking, then the risk at age 50 for a person who began smoking regularly at age 13 is 350 percent greater than that for a 50-year-old who started smoking at age 23.

Similar analyses have not been done for other smoking-related sites of cancer. Nevertheless, for most smoking-related cancers, the risk rises with the duration of smoking (USDHHS 1982, 1989, 1990; International Agency for Research on Cancer 1985). One could