THE HEALTH CONSEQUENCES OF SMOKING

CARDIO VASCULAR DISEASE

A Report of the Surgeon General

1983

INTRODUCTION, OVERVIEW, AND CONCLUSIONS

Introduction

Organization and Development of the 1983 Report

The content of the Report is the work of numerous scientists and experts within the Department of Health and Human Services as well as from outside the organization. Individual manuscripts were written by experts nationally and internationally recognized for their scientific contributions to the understanding of cardiovascular diseases. These manuscripts were reviewed individually by other experts, within and outside the U.S. Public Health Service, and the entire Report was reviewed by a broad-based panel of distinguished cardiovascular scientists. The 1983 Report includes a Foreword by the Assistant Secretary for Health of the Department of Health and Human Services and a Preface by the Surgeon General of the U.S. Public Health Service. The body of the report consists of eight sections and two appendices, as follows:

1. Section 1. Introduction, Overview, and Conclusions
2. Section 2. Arteriosclerosis
3. Section 3. Coronary Heart Disease
4. Section 4. Cerebrovascular Disease
5. Section 5. Atherosclerotic Peripheral Vascular Disease and Aortic Aneurysm
6. Section 6. Pharmacological and Toxicological Implications of Smoke Constituents on Cardiovascular Disease
7. Section 7. Changes in Cigarette Smoking Behavior in Clinical and Community Trials
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Historical Perspective

Early reports linking smoking with a greater risk of developing cardiovascular disease occurred around the turn of the century. An early series of studies, initiated in 1904 by Erb, found a much higher percentage of smokers than of nonsmokers with intermittent claudication; only 10 percent of his patients with claudication were nonusers of tobacco. As early as 1934, Howard made the observation that the increasing

prevalence of coronary heart disease noted since the first World War might be a result of the greatly increased use of cigarettes.

By the turn of the century, numerous studies had demonstrated clinically and experimentally that cigarette smoking or cigarette smoke constituents, most notably nicotine, caused an elevation in blood pressure and heart rate during smoking.

The first major prospective study results were made public in 1954 in the United States by Hammond and Horn and found a strong association between cigarette use among men and coronary heart disease (CHD). Overall, smokers were found to carry a 70 percent greater risk of dying from CHD than nonsmokers; heavy smokers had CHD mortality rates almost two and one-half times greater than nonsmokers. Hammond and Horn also noted a consistent dose-response relationship with the number of cigarettes consumed per day.

In the intervening 30 years, numerous additional epidemiological mortality studies were undertaken to examine this issue. These included studies in the United Kingdom, Canada, Sweden, Japan, and Switzerland in addition to the United States. In total, they represent more than 20 million person-years of observation. Findings from these studies have been remarkably uniform: smokers have much higher death rates from coronary heart disease than do nonsmokers, despite the fact that these studies were conducted in varying populations, were geographically diverse, and involved differing methodologies.

The first major U.S. Public Health Service review of the relationship between smoking and heart disease was conducted by the Surgeon General's Advisory Committee on Smoking and Health in 1964. Although the Committee noted that male smokers had higher death rates from coronary heart disease, it was unable to conclude that the association had causal significance. However, it was noted in the report that "the causative role of these factors [risk factors including cigarette smoking] in coronary disease, though not proven, is suspected strongly enough to be a major reason for taking countermeasures against them. It is also more prudent to assume that the established association between cigarette smoking and coronary disease has causative meaning than to suspend judgement until no uncertainty remains."

Since the release of the original Report of the Surgeon General in 1964, additional studies dealing with cigarette smoking and CHD have been summarized in the series of annual reports of the Surgeon General on *The Health Consequences of Smoking.* By 1979, the magnitude of the epidemiological, pathological, clinical, and experimental evidence had grown to the point that the Surgeon General's Report concluded: "Smoking is causally related to coronary heart disease in the common sense of the idea and for the purposes of preventive medicine."

Overview

In 1980, diseases of the circulatory system were responsible for approximately one-half of the total U.S. mortality. *CHD was the single most important cause of death,* accounting for approximately 30 percent of all U.S. deaths.

Cigarette smoking is one of the three major independent CHD risk factors. The magnitude of the risk associated with cigarette smoking is similar to that associated with the other two major CHD risk factors, hypertension and hypercholesterolemia; however, because cigarette smoking is present in a larger percentage of the U.S. population than either hypertension or hypercholesterolemia, cigarette smoking ranks as the largest preventable cause of CHD in the United States. Cigarette smoking also acts synergistically with the other major risk factors to greatly increase the risk for CHD.

Arteriosclerosis is the predominant underlying cause of cardiovascular disease, and atherosclerosis is the form of arteriosclerosis that most frequently causes clinically significant disease, including CHD, atherothrombotic brain infarction, atherosclerotic aortic disease, and atherosclerotic peripheral vascular disease. Cigarette smoking contributes both to the development of atherosclerotic lesions and to the clinical manifestations of atherosclerotic vascular disease, including sudden death. Although the precise pathophysiologic basis of these clinical manifestations is not understood, it may be related to several deleterious cardiovascular effects of cigarette smoking, including production of an imbalance between myocardial oxygen supply and demand, a decrease in the threshold for ventricular fibrillation, and an increase in platelet aggregation. Nicotine and carbon monoxide are the tobacco smoke constituents most closely associated with these adverse effects; other cigarette smoke constituents such as hydrogen cyanide, oxides of nitrogen, and carbon disulfide are being studied for possible pathogenic cardiovascular effects.

Cigarette smoking is the most important risk factor for atherosclerotic peripheral vascular disease, which usually involves the lower extremities. Smoking cessation is probably the single most important intervention in the management of this disorder. The effect of cigarette smoking to aggravate and accelerate the development of atherosclerosis is more striking in the aorta than in any other vessels. Cigarette smoking is associated with an increased risk for cerebrovascular disease, especially in younger age groups, but this effect is less marked than for atherosclerotic disease at other sites. Women cigarette smokers experience an increased risk for subarachnoid hemorrhage; the use of both cigarettes and oral contraceptives greatly increases this risk.

Smoking cessation is associated with decreased mortality and morbidity from atherosclerotic vascular disease. Prospective epidemiologic studies have shown that former cigarette smokers reduce their CHD death risk from that of current smokers to that of nonsmokers over approximately a 15-year period after stopping smoking. The beneficial effects of quitting are not explained by differences in baseline characteristics between quitters and continuing smokers. CHD intervention trials have successfully demonstrated the feasibility of reducing cigarette consumption; these trials also documented a significant reduction in CHD mortality.

Conclusions of the 1983 Report

The purpose of this Report is to review in depth the many sources of scientific evidence relating cigarette smoking to individual cardiovascular disease entities. Listed below are the major findings of this review.

Arteriosclerosis

1. Preponderance of evidence both from prospective studies with autopsy followup and from autopsy studies with retrospective smoking data indicates that cigarette smoking has a significant positive association with atherosclerosis. This evidence suggests that cigarette smoking has the effect of aggravating and accelerating the development of atherosclerotic lesions in the artery wall and that its effect is not limited to those events related to the occlusive episode. The effects are most striking for aortic atherosclerosis; a significant positive relationship also exists between cigarette smoking and atherosclerotic lesions in the coronary arteries, at least for most high risk populations. Cigarette smoking could also be associated with other factors that precipitate thrombosis, hemorrhage, or vasoconstriction leading to occlusion and ischemia.
2. Some evidence exists that cigarette smoke alters total serum cholesterol concentrations and lipoprotein composition in ways that would be expected to increase the development of atherosclerosis. Recent studies of the effects of smoking on the hemostatic system indicate effects on platelet function.
3. Although the specific mechanisms by which tobacco smoke affects arteriosclerosis have not been clearly delineated, the effects of cigarette smoking on the atherosclerotic lesions that underlie cardiovascular disease seem well established.

Coronary Heart Disease

1. Cigarette smoking is a major cause of coronary heart disease in the United States for both men and women. Because of the number of persons in the population who smoke and the increased risk that cigarette smoking represents, it should be considered the most important of the known modifiable risk factors for CHD.
2. Overall, cigarette smokers experience a 70 percent greater CHD death rate than do nonsmokers. Heavy smokers, those who consume two or more packs per day, have CHD death rates between two and three times greater than nonsmokers.
3. The risk of developing CHD increases with increasing exposure to cigarette smoke, as measured by the number of cigarettes smoked daily, the total number of years one has smoked, and the degree of inhalation, and with an early age of initiation.
4. Cigarette smokers have a twofold greater incidence of CHD than do nonsmokers, and heavy smokers have an almost fourfold greater incidence.
5. Cigarette smoking is a major independent risk factor for CHD, and it acts synergistically with other risk factors (most notably, elevated serum cholesterol and hypertension) to greatly increase the risk of CHD.
6. Women have lower rates for CHD than do men. In particular, CHD rates for women are lower prior to the menopause. A part of this difference is due to the lower prevalence of smoking in women, and for those women who do smoke, to the tendency to smoke fewer cigarettes per day and to inhale less deeply. Among those women who have smoking patterns comparable to male smoking patterns, the increments in CHD death rates are similar for the two sexes.

7. Women who use oral contraceptives and who smoke increase their risk of a myocardial infarction by an approximately tenfold factor, compared with women who neither use oral contraceptives nor smoke.
8. Cigarette smoking has been found to significantly elevate the risk of sudden death. Overall, smokers experience a two to four times greater risk of sudden death than nonsmokers. The risk appears to increase with inc reasing dosage as measured by the number of cigarettes smoked per day and diminishes with cessation of smoking.
9. The CHD mortality ratio for smokers compared with nonsmokers is greater for the younger age groups than for the older age groups. Although the smoker-to-nonsmoker mortality ratio narrows with increasing age, smokers continue to experience greater CHD death rates at all ages.
10. Cigarette smoking has been estimated to be responsible for up to 30 percent of all CHD deaths in the United States each year. During the period 1965 to 1980 there were over 3 million premature deaths from heart disease among Americans attributed to cigarette smoking. Unless smoking habits of the American population change, perhaps 10 percent of all persons now alive may die prematurely of heart disease attributable to their smoking behavior. The total number of such premature deaths may exceed 24 million.
11. Cessation of smoking results in a substantial reduction in CHD death rates compared with those of persons who continue to smoke. Mortality from CHD declines rapidly after cessation. Approximately 10 years following cessation the CHD death rate for those ex-smokers who consumed less than a pack of cigarettes daily is virtually identical to that of lifelong nonsmokers. For ex-smokers who had smoked more than one pack per day, the residual risk of CHD mortality is proportional to the total lifetime exposure to cigarette smoke.
12. Epidemiologic evidence concerning reduced tar and nicotine or filter cigarettes and their effect on CHD rates is conflicting. No scientific evidence is available concerning the impact on CHD death rates of cigarettes with very low levels of tar and nicotine.
13. Smokers who have used only pipes or cigars do not appear to experience substantially greater CHD risks than nonsmokers.

Cerebrovascular Disease

1. Data from numerous prospective mortality studies have shown an association between cigarette smoking and cerebrovascular disease. This risk is most evident in the younger age groups, and the effect diminishes with increasing age, with little or no effect noted after age 65. No consistent dose-response effect has been demonstrated.
2. Women cigarette smokers experience an increased risk for subarachnoid hemorrhage. However, the use of both cigarettes and oral contraceptives greatly increases the risk for subarachnoid hemorrhage among women.

Atherosclerotic Peripheral Vascular Disease and Aortic Aneurysm

1. Cigarette smoking is the most powerful risk factor predisposing to atherosclerotic peripheral arterial disease.

2. Smoking cessation plays an important role in the medical and surgical management of atherosclerotic peripheral vascular disease.
3. Death from rupture of an atherosclerotic abdominal aneurysm is more common in cigarette smokers than in nonsmokers.

Pharmacological and Toxicological Implications of Smoke Constituents on Cardiovascular Disease

1. Over 4,000 different compounds have been identified in tobacco smoke.
2. Nicotine exerts an effect on ganglionic cells, producing transient excitation. The pharmacological effects are small, but are reinforced several times daily in habitual smokers. The exact mechanisms whereby nicotine might influence cardiovascular events are unknown, but a lowering of the ventricular fibrillation threshold is dose related to nicotine levels.
3. Carbon monoxide may act to precipitate cardiac symptomatology or ischemic episodes in individuals already compromised by coronary disease. In addition, carbon monoxide binds to hemoproteins, potentially inhibiting their functions.
4. Several studies have shown that smokers may alter their smoking behavior when they switch to low-yield cigarettes. This compensatory behavior may lead to the increased uptake of gas phase constituents including carbon monoxide, hydrogen cyanide, and nitrous oxides.
5. It is unlikely that a "safe cigarette" can be developed that will reduce cardiovascular risk.

Changes in Cigarette Smoking Behavior in Clinical and Community Trials

1. Smokers involved in intervention programs demonstrate higher smoking cessation rates than those in control groups.
2. In general, the success of smoking intervention programs is related to the amount of intervention provided.

The Effect of Cigarette Smoking Cessation on Coronary Heart Disease

1. In the four intervention trials involving mortality followup of individual men for 5 to 10 years, the intervention groups had a combined total of 10 percent fewer CHD deaths than did the comparable control groups. Differences for other causes of death or for total deaths were not significant.
2. In these trials, the amount of cigarette smoking has been reduced 10 to 50 percent more in the intervention group than in the control group, demonstrating that intervention can alter smoking behavior.
3. In the two trials involving morbidity followup, the intervention groups had 4 and 45 percent lower total CHD incidence than did the respective control groups.
4. The relative reductions in CHD mortality in each of the four intervention studies involving individual followup are reasonably consistent with the reduction in CHD risk factors, and for a combination of all four studies, the reduction is statistically significant.
5. Numerous studies have shown that those who quit cigarette smoking experience a substantial decrease in CHD mortality and an improvement in life expectancy.

6. A number of prospective epidemiological studies indicate that former cigarette smokers substantially reduce their CHD and total death rates from that of current smokers.

Trends in Cardiovascular Diseases

The evidence supports the conclusion that changes in smoking habits have contributed to substantial improvement in mortality rates from the cardiovascular diseases in the United States.

Trends in U.S. Cigarette Use, 1965 - 1980

1. The proportion of current regular smokers declined steadily between 1965 and 1980. The decline was steeper among males (from 52.1 to 37.9 percent) than among females (from 34.2 to 29.8 percent).
2. The proportion of never smokers increased steadily from 1965 to 1980 among males (27.6 to 31.6 percent), except those 45 years old and older. Among females, only 20- to 34-year-olds showed an increase in proportion of never smokers.
3. The mean number of cigarettes smoked per day by current smokers increased slightly from 1970 to 1980 (from 20 to 21.7 cigarettes).
4. Males smoked a higher mean number of cigarettes throughout the 1970 - 1980 period, but the number of males and females increased about the same amount.
5. Heaviest daily consumption was in the middle-aged group (35 - 65 years). The greatest mean increase was observed among women aged 35 to 44.
6. The proportion of current smokers who smoked less than 20 cigarettes per day decreased between 1970 and 1980 (39.8 to 33.8 percent); the proportion smoking one pack exactly (20 cigarettes) remained constant (34.9 to 34.8 percent); the proportion smoking from 21 to 39 cigarettes increased slightly (13.7 to 14.5 percent); and the proportion smoking two or more packs per day increased from 11.4 to 16.8 percent.
7. The proportion of current smokers who attempted to quit three or more times decreased slightly from 1966 to 1980 (41.2 to 38.7 percent).
8. The proportion of former smokers having made three or more attempts to quit increased sharply (36 to 53.2 percent) from 1966 to 1975.
9. The proportion of current smokers who had attempted to quit during the past year increased from 1966 to 1980 (26.0 to 36.7 percent).
10. Among current smokers, younger persons and females were more likely than older persons and males to have attempted to quit during the previous 12 months.
11. The proportion of former smokers who had attempted to quit during the previous 12 months decreased from 1966 to 1975 (13.8 to 9.8 percent).
12. Among former smokers, younger persons and females were more likely than older persons and males to have quit during the previous 12 months.