Statement on Smoking and Cardiovascular Disease for Health Care Professionals

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Cigarette smoking substantially increases the risk of cardiovascular disease, including coronary heart disease, stroke, sudden death, peripheral artery disease, and aortic aneurysm. The overwhelming and consistent evidence supporting a causal role of smoking in cardiovascular disease derives from large numbers of observational analytic studies, both case-control and prospective cohort, in the United States, Europe, and Japan that include more than 20 million person-years of follow-up. The public health toll of this habit is enormous. In the United States, smoking causes more than 230,000 deaths from cardiovascular disease each year, compared with more than 140,000 cancer deaths caused by smoking. It is the leading avoidable cause of all deaths as well as cancer deaths in the United States, accounting for more than 400,000 of the more than 2 million deaths each year. Decreases in United States smoking rates have been encouraging, as is the mounting evidence of the substantial and almost immediate benefits of smoking cessation on the risk of cardiovascular disease. Nevertheless, smoking is responsible for more than one in every five deaths in the United States, accounting for 29% of total deaths from CHD and 17% of all fatalities from stroke. (Reference 2 and National Center for Health Statistics, personal communication, September 1992)

Research is increasingly focusing on the health risks of passive smoking, or environmental tobacco smoke. Environmental tobacco smoke causes lung cancer as well as respiratory disease in children. The possible effects of environmental tobacco smoke on cardiovascular disease are the subject of an American Heart Association special report.

Quantification of Risk and Populations at Risk

Coronary heart disease is the largest contributor to cardiovascular disease morbidity and mortality. Current cigarette smokers have a 70% increased risk of fatal CHD. The overall incidence of nonfatal CHD as well as sudden death is twofold to fourfold higher in cigarette smokers. Among persons with a prior myocardial infarction, current smokers are at increased risk of reinfarction and perioperative mortality following coronary artery bypass surgery. Smoking may also interfere with the treatment of patients with cardiovascular disease by diminishing the benefits of some drugs, such as atenolol or nifedipine, and by altering the metabolism of other drugs, such as propranolol or theophylline.

There is a strong and consistent dose–response relation of smoking with coronary disease. In a follow-up study of British doctors, the death rate for ischemic heart disease among men younger than 60 years of age was 166 per 100,000 for nonsmokers, 278 per 100,000 for those who smoked 1–14 cigarettes per day, and 427 per 100,000 for those smoking 25 or more cigarettes daily. In a very large prospective cohort study of women, the relative risk of fatal CHD was 1.9 for those smoking 1–14 cigarettes per day, 4.3 for those smoking 14–24 cigarettes daily, and 5.4 for those smoking 25 or more cigarettes per day. The risks of smoking are demonstrable in younger, middle-aged, and older individuals. Although the relative risk for coronary heart disease among smokers is far greater in those under 50 than in those over 50, the overall risks increase so markedly with age that the absolute excess risk of coronary heart disease attributable to cigarette smoking is greater in older than in younger people.

Cerebrovascular disease is the second leading manifestation of cardiovascular disease and the third leading cause of all deaths in the United States. The possibility of a causal association of cigarettes with cerebrovascular disease was first raised in the 1964 US Surgeon General's report on smoking, although evidence supporting this judgment was less extensive than that for coronary heart disease. Many but not all studies have reported increased risks of stroke among smokers. Because the larger studies tend to show positive associations and the smaller studies no association, it may be that the smaller studies were less informative than if they had verified the null hypothesis (that there was no association). A meta-analysis has been performed to provide more statistically meaningful estimates. The overview comprised data from 32 studies, including a total of 11,776 stroke cases, and provides strong evidence that cigarette smoking increases the risk of stroke. There is also evidence of a dose–response relation.

As with coronary heart disease, the relative risk for stroke is greater in younger populations. For those
younger than 55, cigarette smoking is associated with a threefold increase in total stroke, while the risk in smokers 55 and older is less marked but still appreciable.20 Several studies have reported greater relative risks for subarachnoid hemorrhage than thrombotic or total stroke. A large prospective cohort study of female nurses found that heavy smokers (≥25 cigarettes daily) had a threefold to fourfold increased risk of total stroke and a nearly 10-fold greater risk of subarachnoid hemorrhage.17 A study of men and women also found greater relative risks of subarachnoid hemorrhage than nonhemorrhagic stroke.21 In a meta-analysis, smoking was associated with a 50% increased risk of total stroke, including an approximate doubling of the risk of cerebral infarction and a threefold increase in risk of subarachnoid hemorrhage.20

Cigarette smoking is the strongest risk factor known for atherosclerotic peripheral vascular disease. The number of cigarettes smoked is highly correlated with the extent of atherosclerotic disease in the large and small arteries of the lower extremities.22 Complications of vascular disease in the lower extremities include tissue ischemia, intermittent claudication, and gangrene. The most severe cases of peripheral vascular disease may result in loss of the affected limb, and smokers are at increased risk of limb amputation consequent to peripheral artery disease.23 Smokers are twice as likely to develop leg pain suggestive of peripheral artery disease during exercise testing and have an increased risk of intermittent claudication.24 In the Framingham Study the risk of intermittent claudication was four times greater in heavy smokers than in nonsmokers.25 In reconstructive arterial surgery of the lower extremities, smokers have higher rates of graft occlusion than nonsmokers.1 The promotion of atherosclerosis by cigarette smoking is most pronounced in the aorta, causing a significantly increased risk of aortic aneurysm. In a recent study of British male civil servants, death from aortic aneurysm was six and a half times greater among smokers than nonsmokers.20

Smoking Cessation and Decreases in Risk of Cardiovascular Disease

The risk of cardiovascular disease begins to decline almost immediately after smoking cessation, even among elderly men and women.13 These findings are a strong reason to stop smoking. Cardiovascular disease risk is far more closely related to the number of cigarettes currently smoked than duration of the habit. One year after smoking cessation, coronary heart disease risk is already reduced by more than 50%, and within several years the coronary heart disease risk of former smokers approaches those of lifelong abstainers.21 Smoking cessation yields significant reductions in CHD risk among those with and without prior CHD. The benefits of smoking cessation on stroke risk are also largely immediate. There were substantial decreases in risk of stroke among those who stopped smoking for 2 years in a large cohort of women17 and after 5 years in the Framingham Heart Study.14 Smoking cessation is the most important intervention in the management of peripheral artery disease, yielding significant reductions in pain in rest in patients with intermittent claudication as well as decreased risk of subsequent myocardial infarction, cardiac death, and total mortality.28 Former smokers also have lower risk of aortic aneurysm than continuing smokers.1

Changing Demographics of Populations at Risk

Gender. Smoking rates have declined significantly during the past several decades, from 42% of the adult population in 1965 to 25% in 1990.29 Cardiovascular mortality rates have also declined steadily during this time by about 2% per year, in men and women, blacks and whites. Although the overall decline in smoking rates in the United States is encouraging, patterns in women, especially adolescents, are alarming and require particular attention in current public health efforts.

Absolute rates of smoking in women remain lower than those in men, but they are declining far more slowly. From 1965 to 1990, smoking rates in women decreased by 32% (from 34% to 23%). During the same period, the rate for men dropped by 46% (from 52% to 28%).29(p203) This far slower decline in smoking rates among women is explained in part by changes in adolescents' smoking behavior. As with adults, smoking rates among teenagers have historically been higher for males than females. In every year since 1980, however, the prevalence of daily cigarette smoking among high school seniors has been higher for females than males.29(p207) As a result, among adults 18–24, the gap in smoking rates between women and men is narrowing (22.5% and 26.6% respectively).29(p203) If this trend continues, by the end of the decade smoking rates for men and women will be about equal and may eventually be higher for women. Thus, current smoking rates in younger women in the United States will contribute substantially to the future burden of both cardiovascular disease and cancer.30

Race. In 1965 smoking rates were 59% for black males and 32% for black females, compared with 51% for white males and 34% for white females.29(p203) Smoking rates of all four groups have decreased markedly since then, with 1990 rates being 32% for black males and 20% for black females, compared with 28% for white males and 24% for white females.29(p203) Presently, among high school seniors, the percentage of whites who at least occasionally smoke cigarettes is 32.5%, significantly higher than the 12.0% of blacks.29(p207) Also among high school seniors, rates of frequent cigarette use, defined as smoking on more than 25 of the 30 preceding days, are 15.9% in whites and 2.3% in blacks. The comparable rates among Hispanic youths are 30.8% and 7.4%.31

Education. Data on smoking and level of education indicate that public health efforts have led to dramatic decreases in smoking rates among those with more education but have had little appreciable impact among those with the lowest educational background. Among college graduates the smoking rate dropped from 34% in 1965 to 14% in 1990, while the rate among those without a high-school degree remained virtually unchanged (36% and 32% respectively).32

Changing demographies of smoking. In the 1989 report “Reducing the Health Consequences of Smoking,” the US Surgeon General outlined the following public health challenge:

Compared with nonsmokers, smokers are disproportionately found in groups that are harder to reach,
and this disparity may grow over time. Greater effort and resources will need to be devoted to achieve equivalent reductions in smoking among those whose behavior has survived strong, countervailing social pressures.

Interrelations With Other Risk Factors

Along with cigarette smoking, hypertension and elevated blood cholesterol level are major independent risk factors for cardiovascular disease. In both men and women, smoking acts synergistically with both of these risk factors to markedly increase the risk of cardiovascular disease; for example, smokers with elevated cholesterol levels have risks greater than the sum of each risk factor independently. Furthermore, smokers are more likely to have lower ratios of high density lipoprotein cholesterol to low density lipoprotein cholesterol. There is also synergy between smoking and hypertension, and smoking accelerates the development of malignant hypertension. People with all three risk factors—smoking, elevated blood cholesterol level, and hypertension—have a far greater risk of cardiovascular disease than if the combined effects were additive.

The use of oral contraceptives is associated with adverse effects on lipid profile and coagulation factors, including fibrinolysis and platelet aggregation. Cigarette smoking acts synergistically with use of oral contraceptives to increase risk of coronary disease. Among premenopausal women, oral contraceptive users have a twofold increased risk of fatal coronary disease while premenopausal women who smoke cigarettes have a 13-fold increase. Women who smoke and use oral contraceptives have a 40-fold increased risk of fatal coronary disease. Oral contraceptive use is also associated with twofold to sixfold increases in risks of ischemic and hemorrhagic stroke as well as venous thromboembolism. Despite the increased relative risks of cardiovascular disease for current oral contraceptive users, absolute excess risks are low, particularly in younger women. Thus, oral contraceptive use in the United States probably accounts for no more than 400 excess vascular deaths each year, in contrast to the tens of thousands of premature deaths attributable to cigarette smoking. Women who smoke should be encouraged to quit. Those who continue to smoke, however, should avoid using oral contraceptives. Past use of oral contraceptives does not appear to confer any increased risk of cardiovascular disease.

Pathophysiology of Smoking

Knowledge about the pathophysiology of smoking is a crucial component of the body of evidence available to public health professionals, and this information may be valuable in communicating to patients the serious health hazards of smoking.

Endothelial injury is a central feature of vascular disease induced by cigarette smoking. Extensive endothelial changes are evident in the umbilical arteries of infants born to mothers who smoke. Lesions of the endothelial cells, subendothelial damage, and platelet adhesion in vessels of laboratory animals exposed to cigarette smoke have been described. Functional abnormalities of the endothelium, such as impairment of prostacyclin biosynthetic capability, also result from cigarette smoking.

The smoking-induced abnormalities in the endothelium are associated with enhanced platelet activation in humans in vivo. In chronic smokers, there is increased urinary excretion of the metabolites of the platelet activation product thromboxane A₂, and the turnover of platelets is accelerated. Narrowing of the small intramyocardial arteries with intimal thickening, in smokers is also consistent with endothelial injury and the resultant adherence of platelets, which release growth factors that induce smooth muscle cell migration, proliferation, and dedifferentiation in the vascular intima. Cigarette smoke is clearly toxic to the vasculature. However, conclusive evidence linking one chemical substance, or a combination, in cigarette smoke to vascular toxicity is not yet available, even though both carbon monoxide and nicotine are known to have deleterious effects on endothelial structure and function. Evidence is still evolving for possible additional effects of smoking on the pathophysiology of the blood–endothelium interface by actions on the function of platelets and on fibrinogen levels.

Injury of the endothelium, causing impaired resistance to platelet adherence and aggregation, favors the growth and propagation of the thrombus that is initiated by rupture of an atheromatous coronary plaque. Such an exaggeration of thrombus development in the coronary arteries of smokers is consistent with the epidemiological finding that smokers have a substantially increased risk of unstable angina, myocardial infarction, and sudden death, the acute cardiac events that ensue from plaque rupture. The rapid reversal of the risk of coronary heart disease morbidity and mortality following smoking cessation is consistent with an important but reversible enhancement of the thrombotic response to vascular injury in active smokers.

Determinants of Smoking Behavior and Change

With the availability of a large and consistent body of evidence on the harmful effects of smoking, as well as on the substantial benefits of quitting, public health research has sought a better understanding of smoking behavior and effective means to achieve cessation. Cigarette smoking is a complex behavior pattern, affected by multiple and interacting physiological, personal (cognitive factors, personality, and demographic factors), and environmental (social, cultural, and economic) influences. Analogously, cessation of smoking is a multifactorial, dynamic process of change that begins with a desire to stop and ends with abstention maintained over a long period of time.

The 1988 Surgeon General’s report, “The Health Consequences of Smoking: Nicotine Addiction,” concluded that cigarettes and other forms of tobacco are addicting; nicotine is the drug in tobacco that causes addiction; and the pharmacological and behavioral processes that determine tobacco addiction are similar to those that determine addiction to heroin and other addictive drugs. Thus, although physiological processes are an important aspect of addiction, nicotine addiction is defined by the interplay between social, behavioral, and physiological factors. Many people dependent on nicotine, like those dependent on alcohol or other drugs, can quit without special treatment pro-
grams, whereas others who are more physiologically addicted may require assistance.

**Smoking Cessation Efforts**

Nearly half of all living adults who ever smoked have stopped, and more than 90% of those quitting between 1964 and 1982 did so without the aid of a formal, organized program.27,57 As a result, the past decade has seen, in addition to the refinement of formal cessation programs, a rapid increase in the availability of self-help materials and the accessibility of smoking interventions (e.g., groups and educational classes) in community sectors such as physicians’ offices, worksites, and schools as well as in the media.58 Voluntary organizations such as the American Heart Association, the American Cancer Society, and the American Lung Association make cessation groups and educational classes available to smokers. These large-group systems, which are easily accessible to smokers, permit wider dissemination of cessation messages than clinic-based approaches and can achieve a sustained impact. This accessibility is extremely important for the large majority of smokers who prefer to stop without the aid of a special program or device. There has also been a growing recognition that smoking is a socially mediated practice susceptible to change in the social environment.59-63 Although most health agencies continue to sponsor group and educational programs to help individual smokers stop, these organizations are also increasingly advocating policies such as smoke-free work environments, addressing environmental factors that support or discourage the habit.60-64 Smokers who receive social support for their efforts to stop are more successful. Those who encounter fewer smokers in their home and work environments are also more successful, while nonsmokers in such surroundings are less likely to start.1 Belief in one’s ability to change a behavior, stress, and self-management and coping skills are also significantly related to whether one smokes. These variables are also related to success in both self-initiated efforts to stop smoking65,66 and those undertaken as part of specialized intervention programs.67

In recent years there has been a trend toward combining educational, behavioral, and pharmacological elements of different cessation methods into a multicomponent program, either group- or individual-based, to respond to the multifactorial nature of smoking.56,68 Multicomponent cessation programs generally produce the best results, although evidence suggests that even with such methods the majority of smokers return to the habit within 1 year.66,67 Other programs include hypnotherapy and acupuncture. However, data on the results of these interventions are scarce. Most cessation treatments yield 1-year quit rates of 10-40% of all original participants.56,67

Because rates of maintenance of abstinence fall over time,27,68 more recent smoking cessation research has focused on prevention of relapse.69 Relapse prevention strategies have included teaching smokers how to recognize cues and use behavioral strategies for dealing with the urge to smoke;80 increasing support for not smoking (e.g., support from telephone contacts, spouses, and coworkers);71 and facilitating changes in self-perception, attitudes, and cognition.69 The latter category includes interventions to help smokers feel more confident about their ability to stop, for example by facilitating their efforts to focus on past successes.

The likelihood of addicted smokers quitting can be improved if they can decrease the distress of withdrawal, either by tapering their nicotine intake before stopping “cold turkey” or by using nicotine replacement therapy such as nicotine gum or a transdermal nicotine patch. When prescribing either nicotine delivery system health care professionals should first assess nicotine dependency. Nicotine replacement only addresses physiological dependency on nicotine; smokers still must overcome the elements of psychological and behavioral dependency on smoking. It is therefore crucial that nicotine replacement be used in the context of a broader behavioral smoking cessation program.

The use of nicotine gum (2 mg nicotine polacrilex [Nicorette, Marion-Merrill Dow]), was approved in 1984 by the Food and Drug Administration as a prescription for tobacco dependency to be used in combination with behavioral treatment. When so used, nicotine gum has been effective in aiding cessation,72 and is more effective in increasing short-term rather than long-term abstinence.73,74 The overall urge to smoke is not reliably decreased by the use of nicotine gum, but specific withdrawal effects, such as anxiety and difficulty concentrating, are often decreased. The transdermal nicotine patch has been shown to be an effective means of nicotine replacement75 and appears to decrease all withdrawal symptoms, including cravings. A nasal nicotine solution and other pharmacological approaches such as Clonidine, an antihypertensive drug, are currently being investigated.

**Special Role of Health Care Professionals**

Health care delivery settings, including physicians’ and dentists’ offices, provide a special opportunity for prevention of smoking or intervention with smokers. Seventy-five percent of the adult population sees a physician at least once a year, and the average is five visits per year. Thus, the physician or other health care professional, acting as educator, facilitator, or counselor, can be a powerful agent for smoking cessation.88 Furthermore, people may think more seriously about their health and the possible deleterious consequences of smoking when they are in a physician’s office or a hospital than at any other time. This opportunity for health promotion with smokers, exsmokers, or would-be smokers merits vigorous pursuit. Family practitioners and health care professionals who treat children can also inform parents who smoke about the harmful effects of passive smoking on the health of their children and offer to help them with smoking cessation.

Smokers value the advice of their physician and believe that their doctor’s intervention can influence their attempts at cessation. Randomized trials indicate that physicians and dentists who intervene with their smoking patients, which takes only a few minutes, have a significant impact on patients’ cigarette smoking behavior.76-79 Furthermore, as the physician-delivered smoking intervention becomes more intensive, the effects are greater, yielding 8-15% cessation depending on the method used.88,89 At the very least, the health care professional needs to ask if a patient smokes, advise smokers to stop and children and teenagers to avoid smoking, help smokers who want to stop, and
schedule a follow-up contact. The health care professional who wants to give smokers help beyond simply providing advice to stop can develop a plan for change and identify strategies to quit. To do this, a patient-centered counseling approach that emphasizes the importance of the patient’s input is optimal. The health professional can ask a series of questions to help the smoker identify motivational factors, strengths, possible problem areas, and solutions. Using this approach, health care professionals can help smokers gain confidence in their ability to quit. This approach, which focuses on strengths as well as past experiences with cessation, has a significant effect on smoking cessation rates. It is most important that the health care provider and patient recognize that smoking cessation is often a long-term process, not a one-time event. Therefore, smokers who relapse need to be encouraged to learn from the experience.

**Conclusion**

Current knowledge provides health care professionals with overwhelming evidence of the cardiovascular disease hazards of cigarette smoking. Cigarette smokers have increased risks of various manifestations of cardiovascular disease at all ages, and with increasing age the absolute excess risks become substantial. Overall, smoking accounts for one fifth of all deaths due to cardiovascular disease. In counseling patients who smoke, health professionals can provide abundant information on the large and almost immediate decrease in cardiovascular risks following smoking cessation. Since the risk of cardiovascular disease is reduced significantly in even elderly smokers after cessation, the clear public health message is that it is never too late to quit. Although smoking rates are decreasing, demographic changes over the past several decades, such as increasing rates in younger women, present particular challenges to health care providers. Smoking rates in the United States are inversely correlated with level of education. Smoking cessation campaigns must, therefore, increasingly be designed and targeted to those people of lower educational levels, whose smoking rates have not decreased significantly in the past 25 years. Extensive research findings on smoking behavior and the efficacy of various cessation strategies are now available to health care professionals. Health care providers can significantly influence smoking behavior in their patients and should vigorously exercise this influence to further the decline in smoking rates in the United States.

**References**

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