Chapter 1

Overview – The Health Consequences of Smoking

Source:

1975 Report, Overview – The Health Consequences of Smoking, pages 1 - 8.

OVERVIEW – HEALTH CONSEQUENCES OF SMOKING

The statement, *"Warning: The Surgeon General Has Determined That Cigarette Smoking Is Dangerous to Your Health,"* has been required by law on cigarette packaging since 1970 as a part of the Public Health Cigarette Smoking Act of 1969. This Act was a response by the U.S. Congress to the scientific information on the health consequences of cigarette smoking summarized in reports then available (the Surgeon General's Report of 1964 and the subsequent 1967, 1968, and 1969 PHS Health Consequences of Smoking). This Act was passed because a series of important questions concerning cigarette smoking and health had been answered.

The following discussion summarizes the basic questions, the methodology used to determine the answers, and the answers themselves.

The initial question to be answered concerning the health consequences of smoking was *"Are there any harmful health effects of smoking cigarettes?"* The answer to this question was provided in two ways. First, it was demonstrated that some diseases occurred more frequently in smokers than in nonsmokers. Second, a causal relationship was established between smoking and these diseases.

Concern about the possible health effects of smoking started when scientists began looking for an explanation to account for the rapidly increasing death rate from lung cancer. The early retrospective studies showed a link between lung cancer and smoking. The first prospective studies, however, found that only one-eighth of the excess overall mortality found among smokers could be accounted for by lung cancer; the rest was largely due to coronary heart disease, chronic respiratory disease, and other forms of cancer. They also found that the effect on overall mortality was largely confined to cigarette smokers rather than the users of other forms of tobacco.

However, demonstrating an association by statistical probability is not enough to establish the causal nature of a relationship. Determining that the association between smoking and excess death rates is cause and effect was a judgment made after a number of criteria had been met, no one of which by itself is sufficient to make this judgment. These criteria as listed in the Surgeon General's

Advisory Committee Report (1964) were the consistency, strength, specificity, temporal relationship, and coherence of the association.

In addition, convincing theories about the mechanisms whereby smoking contributes to the various diseases responsible for the excess mortality among cigarette smokers were developed from the evidence on the biochemical, cytologic, pathologic, and pathophysiologic effects of cigarette smoking, thereby providing the necessary support for the decision that the relationship was causal.

The most important specific health consequence of cigarette smoking in terms of the number of people affected is the development of premature coronary heart disease (CHD). Both prospective and retrospective studies clearly established that cigarette smokers have a greater risk of death due to CHD and have a higher prevalence of CHD than nonsmokers. Long-term followup of healthy populations has confirmed that a cigarette smoker is more likely to have a myocardial infarction and to die from CHD than a nonsmoker. Cigarette smoking has been shown to be one of the major independent CHD risk factors and to act in combination with other major alterable CHD risk factors (high blood pressure and elevated serum cholesterol). Autopsy studies have shown that persons who smoked cigarettes have more severe coronary atherosclerosis than persons who did not smoke. Physiologic studies and animal experiments have indicated several mechanisms whereby these effects can take place.

A second major health consequence of smoking is the development of cancer in smokers. Cigarette smoking was firmly established as the major risk factor in lung cancer. The risk of developing lung cancer was found to be 10 times greater for cigarette smokers than for nonsmokers. The risk of developing lung cancer increases with the number of cigarettes smoked per day and is greater in cigarette smokers who report inhaling, who started smoking at an early age, or who have smoked for a greater number of years. Smokers of filter cigarettes have been shown to have a lower risk of developing lung cancer than smokers of nonfilter cigarettes, but the risk remains well above that for nonsmokers. The risk of developing cancer of the larynx, pharynx, oral cavity, esophagus, pancreas, and urinary bladder was also found to be significantly higher in cigarette smokers than in nonsmokers. Pipe and cigar smokers were found to have elevated risks for the development of cancer of the oral cavity, pharynx, larynx, and esophagus when compared to nonsmokers. Fewer pipe and cigar smokers than cigarette smokers report that they inhale. As a result lungs of pipe and cigar smokers receive much less

exposure to smoke than the lungs of cigarette smokers. This is probably the primary reason for the lower incidence of cancer of the lung for pipe and cigar smokers compared to cigarette smokers.

Women have had far lower rates of lung cancer than men. This has been attributed to the fact that fewer women than men smoke and the fact that women smokers generally select filter and low tar and nicotine cigarettes. However, the percentage of women smokers in the United States has increased steadily in the last 30 years, and since 1955 the death rates from lung cancer in women have increased proportionately more rapidly than the rates for men, reflecting this increased proportion of women smokers.

The tar from cigarette smoke has been found to induce malignant changes in the skin and respiratory tract of experimental animals, and a number of specific chemical compounds contained in cigarette smoke were established as potent carcinogens or co-carcinogens. Malignant changes including carcinoma *in situ* were found in the larynx and in the sputum exfoliative cytology of experimental animals exposed to cigarette smoke.

Nonmalignant respiratory disease is a third area of smoking-induced morbidity and mortality. Cigarette smokers have been shown to have more frequent minor respiratory infections, miss more days from work due to respiratory illness, and report symptoms of cough and sputum production more frequently than nonsmokers. Retrospective and prospective studies with long-term followup have found that cigarette smoking is the primary factor in the development of chronic bronchitis and emphysema in the United States. Cigarette smokers have also been found to be more likely to have abnormalities of pulmonary function and have higher death rates from respiratory diseases than nonsmokers. Data from autopsy studies have shown that cigarette smokers were more likely to have the macroscopic changes of emphysema, and that these changes are closely related to the number of cigarettes smoked per day. Mucous cell hyperplasia has been found more often in cigarette smokers. Cigarette smoke also inhibits the ciliary motion responsible for cleansing the respiratory tract.

An additional area of health concern has been the effect of cigarette smoking during pregnancy. Mothers who smoke cigarettes during the last two trimesters of their pregnancy have been found to have babies with a lower average birth weight than nonsmoking mothers. In addition cigarette smoking mothers had a higher risk of having a stillborn child, and their infants had higher late fetal and

neonatal death rates. There are some data to show that these risks due to cigarette smoking are even greater in women who have a high risk pregnancy for other reasons. These effects may occur because carbon monoxide passes freely across the placenta and is readily bound by fetal hemoglobin, thereby decreasing the oxygen carrying capacity of fetal blood.

Having established that cigarette smoking is a significant causal factor in a number of serious disease processes, two additional questions became important. They are *"Can the health consequences to the individual be averted by stopping smoking or by changing the cigarette,"* and *"What are the overall public health consequences of cessation and of the changes made in cigarettes?"*

The first question is the simpler of the two to answer. In the individual, cessation of cigarette smoking results in a rapid decline of the carbon monoxide level in the blood over the first 12 hours. Symptoms of cough, sputum production, and shortness of breath usually improve over the next few weeks. A woman who stops smoking by the fourth month of her pregnancy has no increased risk of stillbirth or perinatal death in her infant related to smoking. The deterioration in pulmonary function tests that occurs in some smokers becomes less rapid than that of continuing smokers. The death rates from ischemic heart disease, chronic bronchitis, and emphysema also become less than those of the continuing smoker. The risk of developing cancer of the lung, larynx, and oral cavity declines relative to the continuing smoker in the first few years after cessation and 10 to 15 years after stopping smoking approximates that of nonsmokers. A smoker who switches to filter cigarettes and has smoked them for 10 years or longer has a lower risk of developing lung cancer than a smoker who continues to smoke nonfilter cigarettes. The risk to a filter cigarette smoker, however, still remains well above that of a nonsmoker.

The public health benefits of cessation are more difficult to determine than the effects of cessation on the individual. Just as cause-specific death rates have reflected the effect of cigarette smoking on certain diseases, they should also reflect any substantial benefits to be gained by cessation or reduction in cigarette smoking. Several factors combined to produce a reduction in per capita dosage of tobacco exposure in the United States for the years 1966-1970. First, per capita consumption of cigarettes declined from 4,287 cigarettes per person in 1966 to 3,985 in 1970. Second, during this period there was a slow but significant decrease in the average tar and nicotine content of cigarettes as well as a decrease in the amount of

tobacco contained in the average cigarette. The decline in per capita consumption during those years occurred in the face of a substantial increase in the proportion of young women becoming smokers as compared to women of previous generations and so reflected predominantly a decrease in cigarette consumption by men.

Since 1970, although the per capita consumption of cigarettes has increased, the average levels of tar and nicotine have continued to decline, making it more difficult to predict what has happened to per capita dosage.

Examination of cause-specific death rates for the period of this declining per capita consumption reveals that there was a downturn in the male death rate from ischemic heart disease beginning in 1966 which reversed the upward trend that had occurred over the previous two decades. This decline in the death rate from ischemic heart disease has not occurred in women.

The male death rate from chronic bronchitis has also been declining since 1967, and the male death rate for emphysema has declined since 1968 when it was first recorded as a separate category. Female death rates for these two diseases have not shown these trends.

Despite the impressive coincidences of the decline in death rates among males occurring at the same time that there was a decline in per capita cigarette consumption, it is impossible to be certain of the exact cause of the decline in the death rates. These diseases are influenced by a variety of factors in addition to cigarette smoking such as blood pressure and air pollution. Some of these factors have also been subject to major control efforts which may have contributed to the decline in the death rates. In addition, there have been therapeutic advances in the treatment of these problems which may also have helped lower the death rates.

A decline in male death rates from lung cancer should also follow the decline in per capita consumption. This rate would not be influenced as much by changes in other etiologic factors or changes in therapy because cigarette smoking causes from 85 to 90 percent of all lung cancer and there have been no major improvments in survival due to changes in therapy. With lung cancer, however, two additional considerations must be kept in mind. A decline in death rates from lung cancer would be expected to lag several years behind a decline in per capita consumption. In addition, the decline in consumption and switch to low tar and nicotine cigarettes occurred

predominantly in the younger age groups where death rates from lung cancer are low. For these reasons, it is necessary to look at lung cancer death rates by age group rather than total lung cancer death rates. The lung cancer rates by age groups for 1971 suggest a decline in the lung cancer rates for the younger males (under 45), but the confidence limits on these trends at present remain wide enough that it is impossible to say whether this is a real decline or merely a leveling off. The national health statistics broken down by 5-year age groups are currently available only through 1971. The data by age group from a few more years will be necessary to determine whether the changes in smoking behavior which have taken place have reversed the trend of the preceding 40 years of continually increasing lung cancer rates in men. In 1971, the last year for which detailed mortality statistics are available, the accumulated exposure to cigarettes reached its peak among men born between 1915 and 1919, a group then in their early 50's. Cumulative exposure has continued to decline with each successive 5-year birth cohort born since then. The trends of the last few years offer some hope that the peak of the "lung cancer epidemic," as some have termed this phenomenon, may have been reached with this group and that future years will show a slow but consistent decline.

Chapter 2

Cardiovascular Diseases

Part I

Part II

Sources:

Part I — 1971 Report, Chapter 2, pages 15 - 174.
Part II — 1975 Report, Chapter 1, pages 9 - 38.

Chapter 2

Cardiovascular Diseases

Part I

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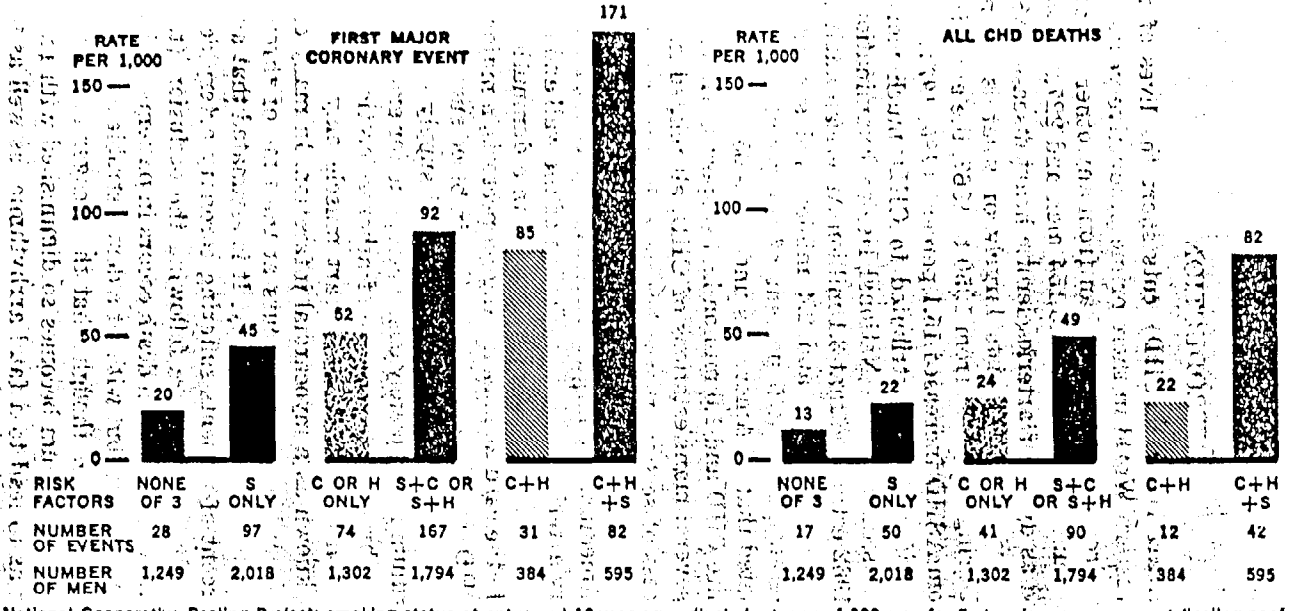
INTRODUCTION

Coronary Heart Disease (CHD) cuts short the lives of many men in the Western World in their prime productive years. More Americans die from heart disease than from any other disease. In 1967, in this country, a total of 345,154 men and 227,999 women were classified as dying of arteriosclerotic heart disease (ASHD) (*196*), a category which consists largely of what is commonly called CHD. During the years from 1950 to 1967, the age-adjusted death rate from ASHD increased 15.1 percent (*196, 197*).

Besides the many deaths attributed to CHD, much morbidity results from this disease. The National Health Examination Survey of 1960–1962 estimated that 3.1 million American adults, ages 18 to 79, had definite CHD and 2.4 million had suspect CHD, together representing about 5 percent of the population. It was further estimated that of Americans under age 65, almost 1.8 million had definite CHD and 1.6 million had suspect CHD (*195*).

There are several manifestations of CHD, all related in part to the basic process of severe atherosclerosis, a disease of arteries in which fatty materials (lipids) accumulate in the form of plaques in the walls of medium and large arteries. This process, as it occurs in the coronary arteries, leads to stiffening of the wall and narrowing of the lumen which, when severe, result in a diminution in the blood supply to the cardiac muscle. Angina pectoris, a major manifestation of CHD, results from diminution in blood supply relative to the needs of the myocardium. If the blood supply to a portion of the myocardium is completely obstructed, due for example to the formation of a thrombus at the site of atherosclerotic narrowing, necrosis or death of a portion of heart muscle may occur. This occurrence is known as a myocardial infarction. In many cases, a disturbance of cardiac rhythm occurs at the time of thrombosis, and the patient may die immediately. It is estimated that approximately 25 percent of patients suffering coronary artery occlusion die within the first three hours following the occlusion (table 1) (*88*). Not infrequently, sudden death occurs in patients with severe coronary atherosclerosis but without a demonstrable arterial occlusion. In these cases, it is thought that the meager blood flow to a portion of the myocardium becomes so diminished with respect to cardiac needs as to lead to a fatal arrhythmia, as well as to, perhaps, a myocardial infarction.

CIGARETTE SMOKING(S) AT ENTRY—WITH CONTROL OF SERUM CHOLESTEROL (C) AND DIASTOLIC BLOOD PRESSURE (H)—AND TEN YEAR INCIDENCE AND MORTALITY RATES. 7,594 WHITE MALES AGE 30–59 AT ENTRY, POOLING PROJECT



RISK FACTORS	NONE OF 3	S ONLY	C OR H ONLY	S+C OR S+H	C+H	C+H +S	NONE OF 3	S ONLY	C OR H ONLY	S+C OR S+H	C+H	C+H +S
NUMBER OF EVENTS	28	97	74	167	31	82	17	50	41	90	12	42
NUMBER OF MEN	1,249	2,018	1,302	1,794	384	595	1,249	2,018	1,302	1,794	384	595

National Cooperative Pooling Project; smoking status at entry and 10-year age-adjusted rates per 1,000 men for first major coronary event (incling nonfatal MI, fatal MI, and sudden death due to CHD) and any coronary death. U.S. white males age 30–59 at entry. All rates age-adjusted by 10-year age groups to the U.S. white male population 1960. Graphs present rates for noncigarette vs. cigarette smokers at entry with simultaneous control of blood pressure and serum cholesterol level. For this latter analysis, the following cutting points were used:

(a) Cigarette smoking S —any use at entry
(b) Serum cholesterol C — 250 mg./dl.
(c) Diastolic blood pressure H — 90 mm. Hg.

SOURCE: Inter-Society Commission for Heart Disease Resources. National Cooperative Pooling Project Data (88).

FIGURE 1—National Cooperative Pooling Project; smoking status at entry and 10-year age-adjusted rates per 1,000 men for first major coronary event (includes nonfatal MI, fatal MI, and sudden death due to CHD) and any coronary death. U.S. white males age 30–59 at entry. All rates age-adjusted by 10 year age groups to the U.S. white male population 1960. Graphs present rates for noncigarette vs. cigarette smokers at entry with simultaneous control of blood pressure and serum cholesterol level. For this latter analysis, the following cutting points were used:

(a) Cigarette smoking—S–any use at entry
(b) Serum cholesterol—C–≧250 mg./dl.
(c) Diastolic blood pressure—H–≧90 mm. Hg.

SOURCE: Inter-Society Commission for Heart Disease Resources. National Cooperative Pooling Project Data (*88*).

TABLE 1.—*Sudden death and acute mortality with first major coronary episodes*

Author, year, country, reference	Number and type of population	Data collection	Event	Number of events	Proportion per 1,000 events (as calculated on the basis of age-adjusted rates)	Comment
Pooling Project, American Heart Association, 1970, U.S.A. (*88*).	7,594 males males 30–59 years of age at entry. Ten-year experience.	Medical examination and follow-up.	All first major coronary episodes, nonfatal and fatal.	501	1,000.0	Data from the Pooling Project, Council on Epidemiology, American Heart Association, a national cooperative project for pooling data from the Albany civil servant, Chicago Peoples Gas Co., Chicago Western Electric Co., Framingham Community, Los Angeles civil servant, Minneapolis-St. Paul business men, and other prospective epidemiologic studies of adult cardiovascular disease in the United States.
			Sudden death (death within 3 hours of onset of acute illness)	123	245.5	
			All acute deaths with first episodes.	165	329.3	

SOURCE: Inter-Society Commission for Heart Disease Resources (*88*). Representative references include: (*54, 94, 148, 177*) and others listed as 6a–6k in Inter-Society Commission for Heart Disease Resources report.

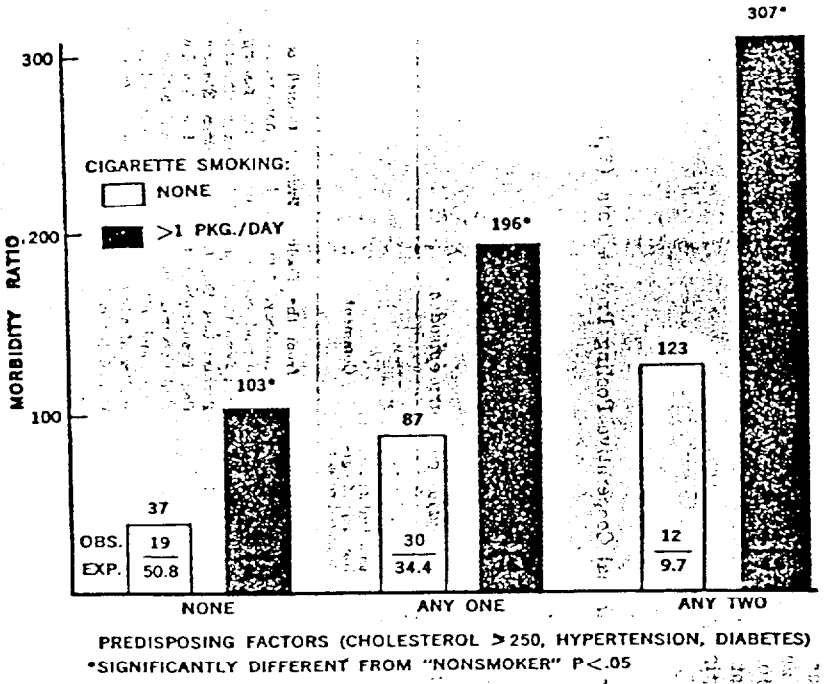


FIGURE 2—Risk of coronary heart disease (12 years) according to cigarette smoking habit and presence of "predisposing factors" (men 30–59 at entry). Framingham Heart Study.

SOURCE: Kannel, W. B., et al. (*94*).

Numerous epidemiological studies have indicated that cigarette smokers have increased mortality ratios for CHD; that is, cigarette smokers show significantly increased death rates compared with nonsmokers (table 2). The risk incurred by cigarette smoking increases with increasing dosage and, as measured by mortality ratios, is more marked for men in the younger age groups, under age 60, although the absolute increment in death rates experienced by smokers over that of nonsmokers continues to increase with increasing age. Table 2 lists the mortality ratios found in the major studies. Certain of these studies, including those at Framingham, Massachusetts, the Health Insurance Plan of New York City (HIP), and at Tecumseh, Michigan, have analyzed morbidity as well as mortality from CHD and have indicated that the risk of developing fatal and nonfatal CHD is greater among cigarette smokers than among nonsmokers (tables 3 and 4). Conflicting evidence has been published concerning the relationship of cigarette smoking and the incidence of angina pectoris. While some

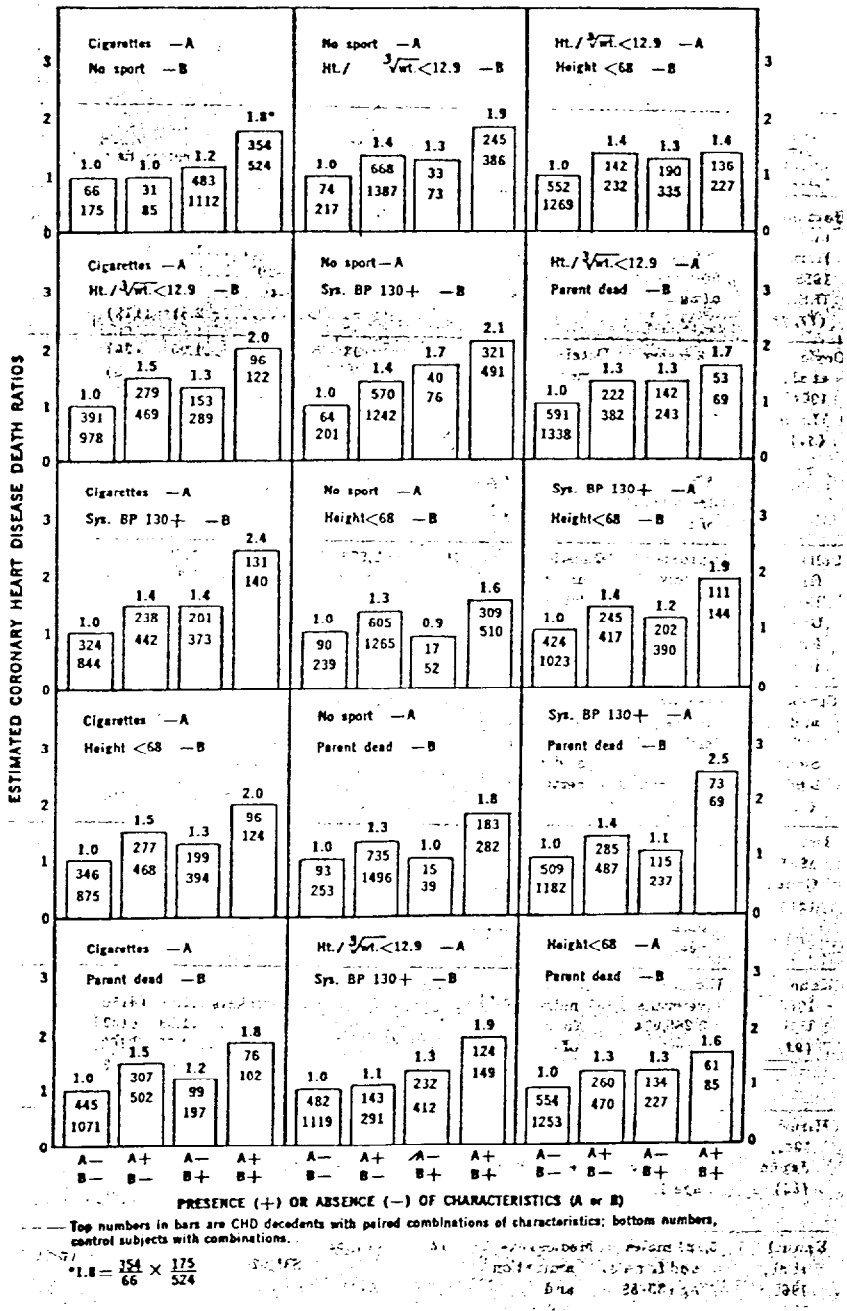


FIGURE 3—Estimated coronary heart disease death ratios in a 17–51 year follow-up, and frequencies of paired combinations of six high-risk characteristics in college, for all ages at death.

SOURCE: Paffenbarger, R. S., et al. (*146*).

TABLE 2.—*Coronary heart disease mortality*

(Actual number of deaths

[SM = Smokers

Author, year, country, reference	Number and type of population	Data collection	Follow-up (years)	Number of deaths	Cigarettes/day		
Hammond and Horn, 1958, U.S.A. (*77, 78*).	187,783 white males in 9 states 50–69 years of age.	Questionnaire and follow-up of death certificate.	3½	5,297	NS 1.00	(709)	[1](p<0.001)
					All smokers .1.70	(3361)	
					<10 .....1.29	(192)	
					10–20 ....1.89	(864)	
					20–40 ....2.20	(604)	
					>40 .....2.41	(118)	
Doyle et al., 1964, U.S.A. (*54*).	2,282 males, Framingham, 30–62 years of age.	Detailed medical examination and follow-up.	10	93	NS 1.00	(20)	
					All smokers .2.40	(73)	
					<20 .....2.00	(17)	
					20 .....1.70	(20)	
					>20 .....3.50	(36)	
	1,913 males, Albany, 39–55 years of age.		8				
Doll and Hill, 1964, Great Britain (*50*).	Approximately 41,000 male British physicians.	Questionnaire and follow-up of death certificate.	10	1,376	NS 1.00		
					All smokers .1.35		
					1–14 ....1.29		
					15–24 ....1.27		
					>25 .....1.43		
Strobel and Gsell 1965 Switzerland (*180*).	3,749 male Swiss physicians.	Questionnaire and follow-up of death certificate.	9	162	NS 1.00		
					1–20 .....1.48		
					>20 .....1.76		
Best, 1966 Canada (*24*).	Approximately 78,000 male Canadian veterans.	Questionnaire and follow-up of death certificate.	6	2,000	NS 1.00		
					All smokers .1.60	(1380)	
					<10 .....1.55	(337)	
					10–20 .....1.58	(766)	
					>20 .....1.78	(277)	
Kahn 1966 U.S.A. (*93*).	U.S. male veterans 2,265,674 person years.	Questionnaire and follow-up of death certificate.	8½	10,890	NS 1.00	(2997)	
					All smokers .1.74	(4150)	
					1–9 .....1.39	(439)	
					10–20 .....1.78	(2102)	
					21–39 .....1.84	(1292)	
					>39 .....2.00	(266)	
Hirayama, 1967, Japan (*84*).	265,118 Japanese adults over age 40.	Trained interviewers and follow-up of death certificate.	1	91	NS 1.00	(17)	
					1–24 .....1.13	(69)	
					>25 .....1.00	(5)	
Kannel et al., 1968, U.S.A. (*94*).	5,127 males and females age 30–59.	Medical examination and follow-up.	12	52	NS 1.00	(27)	(p<0.05)
					SM>20 ....2.20	(25)	

[1] Unless otherwise specified, disparities between the total number of deaths and the sum of the individual smoking categories are due to the exclusion of either occasional, miscellaneous, mixed, or ex-smokers.

ratios related to smoking—prospective studies

shown in parentheses)[1]

NS = Nonsmokers]

Cigars, pipes	Age variation								Comments
Cigars		*50–54*		*55–59*		*60–64*		*65–69*	
NS..1.00	NS	1.00	(90)	1.00	(142)	1.00	(204)	1.00 (273)	
SM..1.28 (420)	All smokers	1.93	(765)	1.85	(962)	1.66	(921)	1.41 (718)	
Pipes	<10	1.38	(35)	1.38	(50)	1.17	(49)	1.27 (58)	
NS..1.00	10–20	2.00	(213)	2.04	(258)	1.91	(235)	1.58 (158)	
SM..1.03 (312)	>20	2.51	(203)	2.47	(199)	1.92	(129)	1.56 (78)	
									Data apply only to males aged 40–49 and free of CHD at entry. NS include pipe, cigar and ex-smokers.
		35–44		*45–64*		*65–84*			
	NS	1.00		1.00		1.00			
	1–14	3.73		1.40		1.71			
	15–24	4.45		1.73		1.27			
	>25	1.36		1.92		1.58			
NS..1.00									
SM..1.45									
Cigars		*30–49*		*50–69*		*70 and over*			
NS..1.00	NS	1.00		1.00		1.00			
SM..0.98 (16)	<10	0.97	(18)	1.56	(220)	1.71	(99)		
Pipes	10–20	1.45	(115)	1.67	(557)	1.29	(94)		
NS..1.00	>20	1.85	(65)	1.76	(184)	1.73	(28)		
SM..0.96 (95)									
Cigars									
NS..1.00									
SM..1.04 (623)									
Pipes									
NS..1.00									
SM..1.08 (886)									
									Preliminary report.

[1] "p" values specified only for those provided by authors.

TABLE 2.—*Coronary heart disease mortality ratios*

(Actual number of deaths

{SM = Smokers

Author, year, country, reference	Number and type of population	Data collection	Follow-up (years)	Number of deaths	Cigarettes/day	Males	Females	
Hammond and Garfinkel, 1969, U.S.A. (*76*).	358,534 males 445,875 females age 40–79 at entry.	Questionnaire and follow-up of death certificate.	6	14,819		*Males*	*Females*	
					NS	1.00	1.00	
					1–9	1.27	0.84	
					10–19	1.60	1.22	
					20–30	1.78	1.52	
					>40	1.77	0.61	
Paffenbarger and Wing 1969 U.S.A. (*146*)	50,000 male former students.	Baseline interview and examination and follow-up by death certificate.	17–51	1,146 matched with 2,292 controls	NS SM	1.00 1.50	(385)	($p<0.01$)
Paffenbarger et al., 1970, U.S.A. (*144*).	3,263 male longshoremen 35–64 years of age.	Initial multiphasic screening and follow-up of death certificate.	16	291	NS and <20 SM >20	1.00 2.08	(137) (154)	($p<0.01$)
Taylor et al., 1970, U.S.A. (*185*).	2,571 male railroad employees 40–59 years of age at entry.	Interviews and regular follow-up examination.	5	46	NS <20 >20	1.00 1.97 3.60	(4) (20) (22)	
Weir and Dunn, 1970, U.S.A. (*205*).	68,158 California male workers 35–64 years of age at entry.	Questionnaire and follow-up of death certificate.	5–8	1,718	NS All smokers ±10 ±20 >30	1.00 1.60 1.39 1.67 1.74		
Pooling Project, American Heart Association, 1970, U.S.A. (*88*).	7,427 white males 30–59 years of age at entry.	Medical examination and follow-up.	10	289	NS <10 20 >20	1.00 1.65 1.70 3.00	(27) (84) (86) (68)	

[1] Unless otherwise specified, disparities between the total number of deaths and the sum of the individual smoking categories are due to the exclusion of either occasional, miscellaneous, mixed, or ex-smokers.

related to smoking—prospective studies (cont.)

shown in parentheses)[1]

NS = Nonsmokers]

Cigars, pipes	Age variation				Comments
		Males			†Based on
	40–49	*50–59*	*60–69*	*70–79*	5–9 deaths.
NS	1.00	1.00	1.00	1.00	
1–9	1.60	1.59	1.48	1.14	
10–19	2.59	2.13	1.82	1.41	
20–30	3.76	2.40	1.91	1.49	
>40	5.51	2.79	1.79	1.47	
		Females			
NS	1.00	1.00	1.00	1.00	
1–9	1.81	1.15	1.04	0.76	
10–19	2.08	2.37	1.79	0.98	
20–30	3.62	2.68	2.08	1.27	
>40	†3.31	3.73	†2.02	—	

	30–44	*45–54*	*55–69*	
NS	1.00	1.00	1.00	
	($p<0.01$)			
SM	1.80 (88)	1.60 (163)	1.20 (134)	

Data apply only to those free of CHD at entry.

	35–44	*45–54*	*55–64*	*65–89*	NS includes
NS	1.00	1.00	1.00	1.00	pipes and
±10	4.22	2.05	1.41	1.17	cigars.
±20	6.14	3.17	1.64	1.26	SM includes
±30	8.57	3.83	1.66	1.36	ex-smokers.
>40	7.93	3.15	1.42	1.42	
All	6.24	2.95	1.56	1.24	

1.00 (27)
1.20 (24)