Does Cigarette Smoking Have an Independent Effect on Coronary Heart Disease Incidence in the Elderly?

ABSTRACT

In order to evaluate the effects of cigarette smoking on coronary heart disease (CHD) in elderly persons in the Honolulu Heart Program, 1,394 men between ages 65 and 74 were followed during an average 12-year period for new cases of nonfatal myocardial infarction and fatal CHD. Incidence rates increased progressively in individuals classified at baseline as never, former, and current smokers, respectively. The absolute excess risk associated with cigarette smoking was nearly twice as high in elderly compared with middle-aged men. (*Am J Public Health*. 1991;81:897–899)

Richard Benfante, PhD, MPH, Dwayne Reed, MD, PhD, and John Frank, MD, MSc

Introduction

While cigarette smoking is well established as a major risk factor for coronary heart disease (CHD) in middle-aged populations,[1–3] the question of whether it is a risk factor for CHD in the elderly has recently arisen because some studies have reported a diminished effect of cigarette smoking on CHD in older persons.[4–9] This reported lack of effect of cigarette smoking on CHD in the elderly is puzzling because the deleterious effects of smoking on CHD risk have been implicated at various stages of atherosclerosis, a pathologic process that generally progresses with age.[9–12] We had an opportunity to examine this question in an elderly group of the Honolulu Heart Program (HHP) cohort. The present analysis was undertaken to determine whether cigarette smoking behavior in the elderly predicts subsequent disease to the same degree as in middle-aged men.

Methods

In the HHP cohort, 1,394 men between ages 65 and 74 and free of clinical cardiovascular disease were followed for 12 years after baseline examination for new cases of definite CHD (non-fatal myocardial infarction and fatal CHD). Details of the differential diagnosis of CHD, surveillance procedures, and case ascertainment in the HHP have been previously described.[13–16]

In order to examine the effect of smoking on CHD risk, age-adjusted incidence rates were calculated by smoking status (current, former, and never) at baseline and expressed in terms of person-years of follow-up. The independent role of cigarette smoking on CHD risk was evaluated using a Cox proportional hazards model that included other major risk factors for CHD.[17,18] Relative risks for cigarette smoking were based on the risk ratio between current smokers and never smokers.

In order to see whether the effect of cigarette smoking on CHD risk was different in elderly compared to middle-aged men, 3,429 of the youngest men in the cohort (ages 51–59 at the examination) were studied as well.

Results

Average values for selected smoking characteristics in elderly and middle-aged men are presented in Table 1. Current elderly smokers on average smoked 20 cigarettes per day compared to 24 cigarettes per day for middle-aged men. Current elderly smokers smoked an average of 10 years longer than middle-aged smokers. For both elderly and middle-aged men, former smokers had higher daily consumption rates than current smokers.

In both age groups there was a consistent and progressive increase in CHD incidence rates by increasing level of smoking experience (Figure 1). Never smokers showed the lowest rates of CHD, followed by former smokers, with current smokers exhibiting the highest. When the relative risks for elderly and middle-aged men were compared in a Cox multivariate lifetable regression that included other major risk factors for CHD (systolic blood pressure, serum cholesterol, diabetes history, body mass index, and alcohol intake) the relative risk for the elderly was 1.62 (95% CI = 1.01, 2.61) compared to 1.80 (95% CI = 1.25, 2.61) for the younger men (Table 2). The difference in relative risks (relative risk ratio) between elderly and middle-aged men was not statistically significant. When an age-smoking interaction term was included in the model, it was also not statistically significant. From the perspective of excess risk

From the Honolulu Heart Program (Benfante, Frank) and the National Heart, Lung and Blood Institute (Reed). Address reprint requests to Richard Benfante, PhD, MPH, Honolulu Heart Program, Kuakini Medical Center, 347 N. Kuakini, Honolulu, HI 96817.

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TABLE 1—Average Values for Smoking Characteristics in Elderly and Middle-Aged Smokers at Baseline Examination

Smoking Characteristics	Smoking Status by Age Group at Examination			
	Ages 51–59		Ages 65–74	
	Past N = 1167	Current N = 1303	Past N = 527	Current N = 406
Cigarettes/day	27.5	23.8	26.3	19.7
Years Smoked	24.3	36.1	31.2	46.5
Years Quit	11.6	—	14.9	—

TABLE 2—Relative Risks* for Coronary Heart Disease in Elderly and Middle-Aged Men Based on Current Smoking Status at Baseline Examination

Age Group	N	b	S.E.	RR†	95% CI
65–74	1394	0.485	0.243	1.62	1.01, 2.61
51–59	3429	0.589	0.188	1.80	1.25, 2.61

*Based on Cox multivariate regression (includes age, cigarette smoking, systolic blood pressure, diabetes history, serum cholesterol, body mass index and alcohol intake).
†Determined by taking the exponent of the b-coefficient for smoking (Current—Never) in multivariate setting.

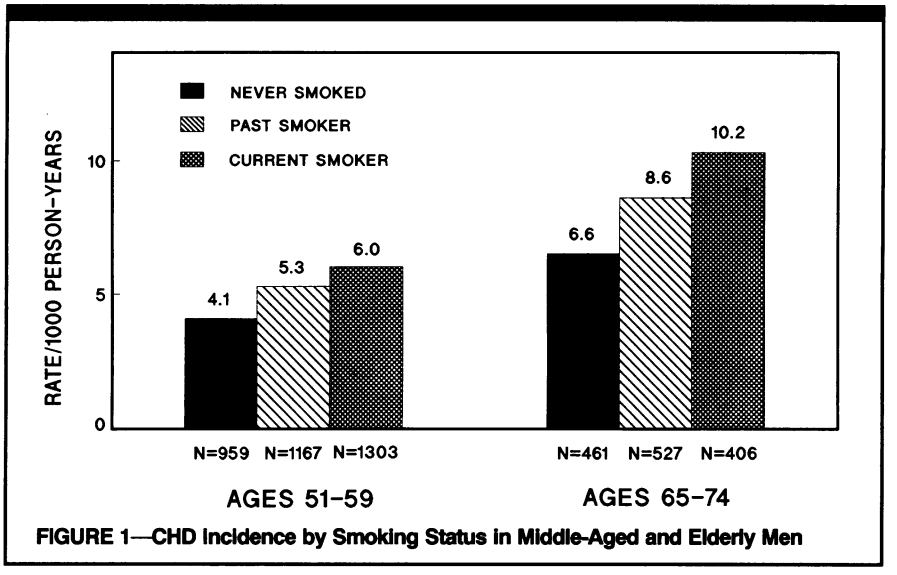

FIGURE 1—CHD Incidence by Smoking Status in Middle-Aged and Elderly Men

for CHD, the difference between current and never smokers for the elderly was 3.6 events per 1,000 person-years, compared to 1.9 events for middle-aged smokers.

Discussion

The results of the present study show that cigarette smoking continues to be an independent predictor of CHD incidence in this cohort of Japanese-American men over age 65, and that the effect is relatively undiminished compared with middle-aged men. CHD incidence for past smokers was intermediate between current and never smokers in both age groups. This is consistent with other evidence that the beneficial effects of quitting are effective later in life as well as during middle years.[1,19–21] It is also important to note that while the relative risk for cigarette smoking on CHD was similar in elderly and middle-aged men, there was nearly a two-fold increase in the excess absolute risk in the elderly men. Thus, since CHD rates increase exponentially with age, the public health impact of a risk factor such as smoking may be greater in older persons even though the relative risk is unchanged.

While a few other studies of older persons have reported an association of cigarette smoking with cardiovascular disease,[19–22] some prospective cohort studies report a diminished effect in the elderly.[4–9] Most notable is the Framingham study which has recently reported that the relationship between cigarette smoking and CHD disappears in both men and women over age 65.[7,8] Several factors may account for differences between the results of Framingham and Honolulu. One is the different analytical methods of relating risk factors to CHD. We used a single baseline measure with an average 12 years of follow-up, compared to the use of the most recent measure from a series of biennial examinations in Framingham. If there was a change in risk factor level, the most recent measure would be used; for example, if a smoker quit, he would be coded as a nonsmoker and then followed for subsequent disease.

Another factor is the potential intervention effect of numerous repeat examinations in Framingham versus just a few examinations in Honolulu. Subjects and their physicians could be notified of high risk levels numerous times in Framingham, but only a few times in Honolulu. Thus, behavioral change or medical intervention would be more likely to occur in Framingham.

There is also the selective attrition effects related to the different time periods of the two studies. The first 20 years of the Framingham follow-up were 1948–68, a period when CHD rates were the highest known in the US. The Honolulu study started in 1965 during a period of declining CHD mortality rates.[23,24] It is possible that these secular effects resulted in differences in the proportion of susceptible subjects reaching the elderly group in the two studies. In addition, the sample of men between ages 65–74 in Honolulu was about twice that for the same age group for men in Framingham, providing a distinct advantage in statistical power.

In conclusion, the present study supports the hypothesis that the biological or clinical effects of such a strong risk factor as cigarette smoking in middle age are maintained over time with increased exposure, and that the benefits of quitting accrue regardless of age. It is therefore recommended that the cessation of cigarette smoking be promoted as part of an overall strategy for prevention of CHD at all ages. □

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References

1. US Department of Health, Education, and Welfare: Smoking and Health: A Report of the Surgeon General. DHEW Pub. No. (PHS) 79-50066. Washington, DC: Government Printing Office, 1979.
2. US Department of Health and Human Services: The Health Consequences of Smoking: Cardiovascular Disease: A Report of the Surgeon General. Pub. No. DHHS (PHS) 84-50204. Washington, DC: Government Printing Office, 1983.
3. Wilhelmsen L: Coronary heart disease: Epidemiology of smoking and intervention studies of smoking. Am Heart J 1988; 115:242–249.
4. Miettinen OS, Neff RK, Hershel J: Cigarette smoking and nonfatal myocardial infarction: Rate ratio in relation to age, sex and predisposing conditions. Am J Epidemiol 1976; 103:30–36.
5. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR: Predicting coronary heart disease in middle-aged and older persons. The Framingham Study. JAMA 1977; 238:497–499.
6. Stokes J, Kannel WB, Wolf PA, Cupples LA, D'Agostino RB: The relative importance of selected risk factors for various manifestations of cardiovascular disease among men and women from 35 to 64 years old: 30 years of follow-up in the Framingham study. Circulation 1987; 75(Suppl V):V65–V73.
7. Harris T, Cook EF, Kannel WB, Goldman L: Proportional hazards analysis of risk factors for coronary heart disease in individuals aged 65 or older. The Framingham Heart Study. J Am Geriatr Soc 1988; 36:1023–1028.
8. Castelli WP, Wilson WF, Levy D, Anderson K: Cardiovascular risk factors in the elderly. Am J Cardiol 1989; 63:12H–19H.
9. Hennekens C, Buring J, Mayrent S: Smoking and aging in coronary heart disease. *In:* Bosse R, Rose C (eds): Smoking and Aging. Lexington, MA: Lexington Books, 1984.
10. McGill HC: The cardiovascular pathology of smoking. Am Heart J 1988; 115:250–257.
11. FitzGerald GA, Oates JA, Nowak J: Cigarette smoking and hemostatic function. Am Heart J 1988; 115:267–271.
12. Mjos OD: Lipid effects of smoking. Am Heart J 1988; 115:272–275.
13. Worth RM, Kagan A: Ascertainment of men of Japanese ancestry in Hawaii through World War II Selective Service Registration. J Chronic Dis 1970; 23:389–397.
14. Belsky JL, Kagan A, Syme SL: Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California. Hiroshima, Japan: Research Plan Atomic Bomb Casualty Commission, 1971; Technical Report 12–71.
15. Nichaman MZ, Hamilton HB, Kagan A, Grier T, Sacks ST, Syme SL: Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: Distribution of biochemical risk factors. Am J Epidemiol 1975; 102:491–501.
16. Yano K, Reed DM, McGee DL: Ten-year incidence of coronary heart disease in the Honolulu Heart Program. Am J Epidemiol 1984; 119:653–666.
17. Cox DR: Regression models and life tables. J R Stat Soc B 1972; 34:187–220.
18. Kalbleisch JD, Prentice RL: The Statistical Analysis of Failure Time Data. New York: John Wiley and Sons, 1980; Ch 4.
19. Jajich CL, Ostfeld AM, Freeman DH Jr: Smoking and coronary heart disease mortality in the elderly. JAMA 1984;252:2831–2834.
20. Hermanson B, Omenn GS, Kronmal RA, Gersh BJ: Beneficial six-year outcome of smoking cessation in older men and women with coronary heart disease: Results from the CASS registry. N Engl J Med 1988; 319:1365–1369.
21. Weintraub WS, Lloyd KW, Seelaus PA, Agarwal JB, Helfant RH: Importance of total life consumption of cigarettes as a risk factor for coronary heart disease. Am J Cardiol 1985; 55:669–672.
22. Siegel D, Kuller L, Lazarus NB, Black D, Feigal D, Hughes G, Schoenberger JA, Hulley SB: Predictors of cardiovascular events and mortality in the systolic hypertension in the elderly program pilot project. Am J Epidemiol 1987; 126:385–399.
23. Stallones RA: The rise and fall of ischemic heart disease. Sci Am 1980; 243:53–59.
24. Thom TJ, Epstein FH, Feldman JJ, Leaverton PE: Trends in total mortality and mortality from heart disease in 26 countries from 1950 to 1978. Int J Epidemiol 1985; 14:510–520.

Staff Involvement and Special Follow-up Time Increase Physicians' Counseling About Smoking Cessation: A Controlled Trial

Carol Duncan, RD, MPH, Morton J. Stein, MD, and Steven R. Cummings, MD

ABSTRACT

We compared the counseling behaviors of two groups of health maintenance organization physicians: one group received training about smoking cessation counseling; the other group received the same training plus staff support and appointment time specially designated for follow-up of smokers. We interviewed patients after their office visits to measure smoking counseling. The group receiving staff support and designated follow-up time counseled more and made more follow-up appointments about smoking. (*Am J Public Health.* 1991;81:899–901)

Introduction

Only a minority of smokers recall being counseled to quit by physicians[1,2] and internists who do counsel smokers seldom use effective strategies such as setting quit dates and making follow-up appointments.[3,4] We have found that even after intensive training in counseling, physicians discussed smoking with only half of their smoking patients and infrequently made follow-up appointments about smoking cessation.[5,6] To determine whether greater involvement of administrative and office staff, and provision of specified follow-up time, would increase physician counseling, we conducted a controlled trial.

From the Division of General Internal Medicine, Department of Medicine, University of California, San Francisco (Duncan, Cummings); Kaiser Permanente Medical Center, San Francisco (Stein); and the Department of Epidemiology and International Health, UCSF (Cummings). Address reprint requests to Carol Duncan, RD, MPH, Substance Abuse Research Group, San Francisco Veterans Administration Medical Center, Bldg. 203, Room 3B-4, 4150 Clement Street, San Francisco, CA 94121-SFVAMC 116W. This paper, submitted to the Journal January 16, 1990, was revised and accepted for publication October 24, 1990.