PASSIVE SMOKING AND THE RISK OF CORONARY HEART DISEASE AMONG MARRIED NON-SMOKING WOMEN

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ABSTRACT

Several epidemiological studies have reported that passive smoking (PS) is a public health hazard and perhaps increases the risk of heart disease. This study evaluated the relationship between female coronary heart disease and PS among married women who described themselves as non-smokers in an area of south-east Iran.

Using household exposure to tobacco smoke as an estimate of PS, a hospital-based case-control study of CHD was conducted in Kerman, Iran. We interviewed 200 married female CHD cases, aged 42-84 years (mean [standard error, SE] 60.0 [0.5]) and 400 hospital-based controls aged 42-85 (mean [SE] 60.3 [0.4]). The controls were non-CHD patients, selected from the same hospital as the cases, and matched for marital status and age (±5 years). All of the cases and controls had never smoked. Information on PS was collected for each person.

The prevalence of PS at home was 39% for cases and 32.3% for controls. The corresponding prevalence rates for PS at work were 1.5% and 0.8%. Household PS increases the risk of female CHD, but this increase was not statistically significant. Compared to non-smokers, exposure to husband's smoking increases the risk by about 40% (odds ratio (OR) 1.40, 95% confidence interval (CI): 0.96-2.05), although no trends were observed with the number of years of exposure. The OR increases with an increase in PS ($p<0.05$). The odds ratio for PS at work did not suggest an increased risk.

The results suggest that passive exposure to cigarette smoke may have a deleterious effect on the health of non-smokers and that married non-smoking women may be at an increased risk of developing CHD through passive exposure to their husband's cigarette smoke.

Keywords: Passive smoking, environmental tobacco smoke, coronary heart disease, myocardial infarction, angina pectoris, epidemiology, Iran.

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INTRODUCTION

It is well established that passive smoking (PS) increases

the risk of lung cancer among never-smokers.[1-4] The possible association between PS and the risk of coronary heart disease (CHD) has stimulated several epidemiological studies.[5-17] Their results in general have demonstrated a weak positive association with no or little apparent dose-response relationship. In 1992, Steenland reviewed the evidence that exposure to PS causes heart disease and concluded that the individual lifetime excess risk of heart disease death due to PS was one to three deaths per 100 people.[18] Also, Glantz and Parmley[19] reviewed the previous studies and concluded that "heart disease is an important consequence of exposure to PS" and estimated that the excess risk of heart disease for non-smokers living with smokers was 30%. The mechanisms by which PS may increase the risk of heart disease are thought to be the same pathway caused by active smoking, including increased platelet aggregation, increased blood carbon monoxide levels, nicotine-induced hypertension and exposure to polycyclic aromatic hydrocarbons.[20] The high rate of exposure to PS among the general population in numerous countries, particularly in developing countries, and recognition of the public health hazard of active smoking are a constant stimulus for new research.

This study, using household exposure to tobacco smoke as an estimate of PS, attempted to clarify further the relationship between female CHD and PS in Kerman, Iran, where such studies have not been carried out. We conducted a case-control study based on data from local hospitals. Data were collected in the city of Kerman (south-east province of Iran) during 1995-96.

SUBJECTS AND METHODS

The cases were married female definite or possible CHD patients who were selected from 2 coronary care units (CCU) and post-coronary care units (PCCU) of training hospitals affiliated to Kerman University of Medical Sciences and Health Services, Iran. These CCU and PCCU departments are the only CCU and PCCU departments of the area and provide essentially all of the cardiological care for persons living in Kerman. Diagnosis was made by a certified cardiologist but without angiography. A "case" was defined as any subject who had a non-fatal ischemic event based on at least two of the three WHO criteria: severe prolonged chest pain, electrocardiographic evidence of a myocardial infarction and raised enzyme levels. The inclusion criteria were residents of Kerman province who were diagnosed by a cardiologist during August 1995 to March 1996. Cases who died before hospitalization, in the emergency room, or shortly after admission to the wards have been excluded. A total of 200 cases were eligible and all were interviewed face-to-face.

The controls were patients without CHD, from

Table I. Group means and proportions for selected variables between 200 coronary heart disease (CHD) cases and 400 non-CHD controls.

Variables	Cases (N=200) Mean(SE)	Controls (N=400) Mean(SE)	Difference (95% CI@)
Age (yr.)	60.0 (0.5)	60.3 (0.4)	-0.3 (-1.6, 1.02)
Duration of diabetes (yr.)	1.8 (0.3)	0.8 (0.1)	1 (0.45, 1.55)**
Duration of hypertension (yr.)	4.2 (0.4)	2.3 (0.2)	1.9 (1.07, 2.73)***
Duration of hypercholesterolemia (yr.)	1.0 (0.2)	0.4 (0.09)	0.6 (0.20, 1.00)*
Duration of OCP use (yr.)	0.2 (0.07)	0.1 (0.04)	0.1 (-0.6, 0.26)
Systolic BP (mmHg)	129.3 (1.5)	124.6 (0.8)	4.7 (1.59, 7.81)**
Diastolic BP (mmHg)	79.2 (0.9)	74.2 (0.6)	5 (2.85, 7.15)***
Fasting blood glucose (mg/dL)	145.4 (5.6)	111.2 (2.4)	34.2 (24.0, 44.4)***
Triglycerides (mg/dL)	171.0 (8.1)	157.2 (3.8)	13.8 (-1.61, 29.2)
Cholesterol (mg/dL)	223.2 (5.6)	198.2 (2.9)	25 (13.8, 36.2)***
No. of children	5.5 (0.2)	5.5 (0.1)	0.0 (-0.37, 0.37)
Husband's no. of cigarette/day	4.9 (0.06)	4.6 (0.5)	0.3 (-1.01, 1.61)
Husband's years of smoking	7.8 (0.9)	7.2 (0.7)	0.6 (-1.71, 2.91)
Hours of exposure/day	1.8 (0.3)	1.2 (0.2)	0.6 (0.03, 1.17)*
No. of cigarette exposure/day	2.5 (0.4)	1.8 (0.3)	0.7 (-0.25, 1.65)
Husband's pack-year of smoking	6.5 (1.0)	7.0 (0.8)	0.5 (-3.0, 2.0)
	No. (%)	No. (%)	
Education			
Illiteracy	173 (86.5)	330 (82.5)	4 (-2.0, 10.0)
Elementary	11 (5.5)	45 (11.2)	-5.7 (-10.2, -10.3)
Junior high	12 (6.0)	23 (5.7)	0.2 (-3.7, 4.3)
≥College	4 (2.0)	2 (0.5)	1.5 (-0.5, 3.6)
Occupation			
Housewife	189 (94.5)	394 (98.5)	-4 (-7.4, -0.6)*
Employee	11 (5.5)	6 (1.5)	
Diabetes			
Yes	67 (33.5)	63 (15.8)	17.8 (10.3, 25.2)***
No	133 (66.5)	337 (84.2)	
Hypertension			
Yes	131 (65.5)	128 (32.0)	33.5 (25.5, 41.5)***
No	69 (34.5)	272 (68.0)	
Hypercholesterolemia			
Yes	44 (22.0)	36 (9.0)	13 (6.6, 19.4)***
No	156 (78.0)	354 (91.0)	

*p<0.05, **p<0.01, ***p<0.001
@CI indicates confidence interval. The difference in the mean and proportion of the variables between cases and controls.

admissions to the same hospital during the same calendar period and matched for marital status and age (±5 years), residential area and lifetime smoking status (all were never-smokers). Information on age, residential area and the diagnosis was obtained from the patients' files and the preliminary "eligible" patients were chosen. Controls were patients who had never been diagnosed as CHD and who had been admitted for neurological disease (13.2%), ENT problems (5%), infectious disease (8.5%), eye disease (2.3%), other non-CHD disease (9.8%) or were in the hospital awaiting surgery (61.2%). Any patient resident in the area was eligible to be selected as a control if she was admitted to the same hospital for one of the conditions listed above. Two controls were selected for each case during the same period of time.

Using a pre-set structured questionnaire, the cases and controls were interviewed in the hospital by a trained

Table II. The distribution of sources of passive smoke and risk of female coronary heart disease in Kerman, Iran, 1995-96.

Sources of passive smoke	Cases (N=200) No. (%)	Controls (N=400) No. (%)	Odds ratio (95% CI)	Etiologic Fraction (%)
Husband only	63 (31.5)	100 (25)	1.40 (0.96-2.05)	11.0
Both husband and others	21 (10.5)	42 (10.5)	1.11 (0.63-1.96)	1.1
Others only	15 (7.5)	29 (7.3)	1.15 (0.59-2.22)	10.1
Total	78 (39.0)	129 (32.3)	1.34 (0.94-1.91)	11.7

Table III. Estimated number of CHD cases attributable to passive exposure to husband cigarette smoking.

Total no. of CHD cases	200
% of people who had never smoked	72.8%
No. of people who had never smoked	437
Prevalence of exposure to husband smoke among married non-smokers	27.2%
Odds ratio of CHD for exposure to husband smoke	1.40
Etiologic fraction of husband smoke	11%
No. of CHD cases in non-smokers attributable to husband smoke	48

Table IV. Characteristics of non-smoking women according to husband cigarette smoking status.

Characteristics	Husband's smoking status Smokers (N=163) Mean(SE)	Non-smokers (N=437) Mean(SE)	Difference (95% CI@)
Age (yr.)	60.1 (0.6)	60.3 (0.4)	-0.2 (-1.60, 1.20)
Duration of diabetes (yr.)	1.0 (0.2)	1.2 (0.2)	-0.2 (-0.77, 0.37)
Duration of hypertension (yr.)	3.2 (0.4)	2.8 (0.2)	0.4 (-0.51, 1.31)
Duration of hypercholesterolemia (yr.)	0.7 (0.3)	0.5 (0.1)	0.2 (-0.24, 0.64)
Duration of OCP use (yr.)	0.2 (0.08)	0.2 (0.04)	0 (-0.17, 0.17)
Systolic BP (mmHg)	129.0 (1.6)	125.2 (0.8)	3.8 (0.50, 7.10)*
Diastolic BP (mmHg)	78.0 (0.9)	75.0 (0.6)	3.0 (0.70, 5.30)**
Fasting blood glucose (mg/dL)	121.6 (4.5)	123.0 (3.0)	-1.4 (-12.60, 9.75)
Triglycerides (mg/dL)	158.5 (6.8)	163.0 (4.4)	-4.5 (-20.9, 11.9)
Cholesterol (mg/dL)	212.0 (5.8)	204.5 (3.0)	7.5 (-1.12, 16.1)
No. of children	5.6 (0.2)	5.4 (0.1)	0.2 (-0.21, 0.61)
Husband's no. of cigarette/day	17.3 (0.8)	-	-
Husband's years of smoking	27.4 (0.9)	-	-
Hours of exposure/day	5.2 (0.4)	-	-
No. of cigarette exposure/day	7.6 (0.7)	-	-
Husband's pack-years of smoking	25.3 (1.5)	-	-
	No (%)	No (%)	
Education			
Illiteracy	139 (85.3)	364 (83.3)	2.0 (-4.5, 8.5)
Elementary	13 (8.0)	43 (9.8)	-1.9 (-6.9, 3.2)
Junior high	9 (5.5)	26 (5.9)	-0.4 (-4.6, 3.7)
≥ College	2 (1.2)	4 (0.9)	0.3 (-1.6, 2.2)
Occupation			
Housewife	159 (97.5)	424 (97.0)	0.5 (-2.3, 3.4)
Employee	4 (2.5)	13 (3.0)	
Diabetes			
Yes	34 (20.9)	96 (22.0)	-1.1 (-8.5, 6.2)
No	129 (79.1)	341 (78.0)	-
Hypertension			
Yes	71 (43.6)	188 (43.0)	0.5 (-8.4, 9.5)
No	92 (56.4)	249 (57.0)	-
Hypercholesterolemia			
Yes	23 (14.1)	57 (13.0)	1.1 (-5.1, 7.3)
No	140 (85.9)	380 (87.0)	

*p<0.05, **p<0.01
@CI indicates confidence interval. The difference in the mean and proportion of the variables between cases and non-passive smokers.

interviewer. Cases and controls were interviewed within a few days of admission. The questionnaire included questions on demographic information, smoking history, and the indoor cigarette smoking habit of the regular family members, exposure to PS at work, level of education, occupation (housewife or employee) and number of children.

In this study, never-smokers were considered patients who had never smoked any kind of tobacco regularly throughout their lifetime and PS was defined as one cigarette/day or more of indoor smoking, by any member of the family sharing the same accommodation as the subject for 12 months or more. Smoking referred only to 'cigarette smoking'; the use of other tobacco products, such as pipe, cigar, cigarillo and snuff, was not considered since they are practically uncommon among Iranians. To measure the cumulative effect of PS on the CHD risk, a pack-year of exposure to passive smoking was calculated as the number of daily cigarettes × years of smoking divided by 20.

The etiologic fraction (EF) is an epidemiological measure to estimate the proportion of disease due to a specific exposure, based on the proportion of the population exposed and the odds ratio due to the exposure.[21] To estimate the proportion of CHD due to PS, based on the proportion of the population exposed and the odds ratio due to the exposure, EF was calculated and defined as:

$$EF = P(OR-1)/1 + P(OR-1)$$

Here, P is the fraction of non-smokers exposed to PS at

home (living with a smoker), OR is the odds ratio for non-smokers exposed to PS at home vs. non-smokers not exposed to PS (the truly non-exposed).

Statistical analysis

Means and standard errors (SE) are presented' for describing variables with continuous distribution. Mean and proportion of characteristics of cases and controls and passive smokers and non-smokers were compared using t-tests and chi-square tests. The OR was used to estimate the ratio of the risk of CHD among "exposed" patients to the risk among "unexposed" patients. The 95% confidence interval (CI) for OR was calculated using Cornfield's method. The Mantel-Haenszel chi-square test was used to test dose-response effects.[22] The analysis was done on a personal computer using SPSS/PC⁺ version 3,[23] Epi-Info[22] and Confidence Interval Analysis Software.[24] All testing for statistical significance was two-tailed, and performed at $p < 0.05$.

Age, gender and a prior history of CHD are important confounding factors of the relationship between risk of CHD and smoking so the estimates from the case-control study were adjusted for these factors by matching or specification techniques.

RESULTS

Differences in distribution of several risk factors among 200 cases and 400 controls are shown in Table I. Cases had longer duration of diabetes, hypercholesterolemia and hypertension, and were more likely to have diabetes, hypertension and hypercholesterolemia than controls. They had longer duration of exposure to PS each day and were more likely to be employees. The cases and controls were comparable with respect to age, triglyceride level at admission, number of children, residential area within Kerman, smoking status during their lifetime, OCP use, years of education, religion, husband's number of cigarettes per day and husband duration of smoking. The cases ranged in age at interview from 42 to 82 years, with a mean (SE) of 60.0 (0.5) years. The controls ranged in age at interview from 42 to 85 years, with a mean (SE) of 60.3 (0.4). The mean (SE) number of years of education was 1.12 (0.2) for cases and 1.12 (0.1) for controls. 83.8% of cases and controls were illiterate.

Table II presents the prevalence and distribution of sources of PS and risk of female CHD. Prevalence rates for PS at home were higher among cases (39%) than controls (32.3%). Although the PS exposure was mainly from husbands in both cases and controls, the percentage from husbands for the cases (31.5%) was higher than that for controls (25%). The difference in percentage of exposure to husband smoking between cases and controls (6.5%) was close to the level of significance (95% CI: -1.2%, 14.2%; $p=$

Table V. The risk of female coronary heart disease from exposure to tobacco smoke by duration and level of exposure period in Kerman, Iran, 1995-96.

Exposure Period (yr.)	Cases No. (%)	Controls No. (%)	Odds ratio (95% CI)
≤30	47 (23.5)	59 (14.8)	1.74 (1.13, 2.69)
>30	16 (8.0)	41 (10.3)	0.86 (0.46, 1.58)
Level of exposure (Cig./day)			
<20	33 (16.5)	41 (10.3)	1.76 (1.07, 2.91)
≥20	30 (15.0)	59 (14.8)	1.11 (0.69, 1.81)

0.09). Prevalence rate of PS at work was about 1% for both groups (1.5% in cases and 0.8% in controls). Many of the women in the study did not work outside the home, so the figures available for analysis of PS at work were too few for valid statistical analysis. When non-PS is the reference category, husband PS exposure has an OR of 1.40 (95% CI: 0.96-2.05), based on 63 cases and 100 controls whereas the OR for others household PS exposure is 1.15 (95% CI: 0.94-1.91) based on 78 cases and 129 controls. The etiologic fraction for CHD due to PS at home, assuming an odds ratio of 1.40, was 11%. The etiologic fraction shows that about 11% of CHD among non-smoking women in this study could be prevented if their husbands stopped smoking completely. The number of CHD in never-smokers attributable to PS at home is therefore estimated to have been 48 (Table III).

Table IV shows the characteristics of 600 married non-smoking women according to their husbands' cigarette smoking status. There were 163 (27.2%) whose husbands smoked. The two groups of women were similar with respect to age, duration and status of diabetes, hypertension, hypercholesterolemia, OCP use, level of fasting blood glucose, cholesterol, triglycerides at admission, number of children, residential area within Kerman, smoking status during their lifetime, years of education, religion, and occupational status, Women whose husbands smoked had slightly higher systolic (129 vs. 125.2 mmHg; $p < 0.05$) and diastolic blood pressure (78 vs. 75 mmHg; $p < 0.01$).

The mean (SE) number of years that cases reported exposure to PS was 7.8 (0.9) and 7.3 (0.7) for controls. Table V shows the OR for female CHD by duration of exposure. It was shown that the risk for female CHD was highest in those exposed for 30 years or less (OR 1.74, 95% CI: 1.13-2.69) and decreased with increasing duration of exposure. No trend in the OR was observed with the duration of exposure to PS. A similar pattern was observed when OR was calculated according to pack-years of exposure. The OR associated with ≤10 pack-years was 1.95 (95% CI: 1.08-3.50). For greater than 10 pack-years, the OR was 1.17 (95% CI: 0.76-1.81). The reported mean (SE) number of hours per day that subjects were exposed was 1.8 (0.3) for cases and 1.2 (0.2) for controls ($p < 0.05$).

No trends in the risk concerning level of husband's

smoking was seen. The OR for CHD among wives whose husbands smoked <20 and ≥20 cigarettes per day as compared to wives of non-smokers was 1.76 (95% CI: 1.07-2.91) and 1.11 (95% CI: 0.69-1.81), respectively (p for trends= 0.45).

No proportional differences were found between cases and controls who were exposed to PS at the workplace. Only 1.5% (3) of cases and 0.8% (3) of controls reported workplace exposure.

DISCUSSION

The effect of PS on CHD, particularly on female CHD, has been of increasing concern since the early 1980s. The level of various smoke contaminants, including carbon monoxide, polycyclic aromatic hydrocarbons and nicotine have been shown to be higher in PS than in mainstream smoke.[25] The mechanisms by which PS may increase the risk of heart disease are thought to be the same pathway caused by active smoking, including increased platelet aggregation and blood carbon monoxide level, nicotine-induced hypertension and exposure to polycyclic aromatic hydrocarbons,[20] which may cause as many severe health problems as active smoking.[26,27] In this study the husband was usually the single most important source of PS and household exposure to cigarette smoke, from husband or other family member, was taken as an estimate of PS. The data showed an increased but insignificant risk (possibly because of the inadequate statistical power of the study) associated with women exposure to PS, suggesting that PS increased the risk of CHD by at least some of the same mechanisms as active smoking. The statistical power to detect small significant differences in our study was limited. Previous studies on PS and heart disease have found relative risks ranging from 0.9 to 3.0.[5-17] By combining these studies, the sample size and, therefore, the power to detect an effect increases. Wells[28] and Glantz and Parmley[19] used then-available studies to compute a pooled relative risk of 1.3 (95% CI: 1.2-1.4). The increased risk found in our study is consistent with these findings. These studies that suggest an association between PS and heart disease have been done in populations where smoking among women is common. It may be possible that some women in these studies could have been wrongly classified as passive smokers while in fact they were active smokers. Smoking among women in Kerman is almost non-existent and the cases and controls were comparable with respect to smoking status during their lifetime (neither had ever smoked).

Among the most heavily exposed subjects in terms of duration or intensity of exposure, no increased risk of CHD was observed.

In this study, the estimated total of 48 cases of CHD attributable to PS represent about 25% of all cases of CHD in those who had never smoked.

The inadequate statistical power is a limitation of this study. Concerning exposure to PS at work, since few of the women worked outside the home (only 16 women), the numbers of subjects was too small for valid statistical analysis.

Information bias is "inherited" in case-control studies. At present, information on past exposure to PS is obtained by subject recall through interview. In this study, the interviewer was not blinded to the subject's case/control status. In an attempt to limit information bias, we used a structured questionnaire and standardized interview techniques, both of which are often helpful in minimizing both recall and interview bias. Another source of bias is that information on smoking was unobtainable for cases who had died. Subjects who died may have had a longer duration and amount of exposure and may have affected the non-existent dose-response effect in this study.

Nevertheless, the findings from this study provide further support to the observation that PS may increase the risk of subsequent CHD. Alternatively, cigarette smoking by a husband could reflect an otherwise less healthy lifestyle shared by the wife: this possibility was not supported by comparisons of plasma cholesterol triglycerides, fasting blood glucose and history of diabetes, hypertension or hypercholesterolemia, all of which were similar or lower in wives of smokers compared with other women.

This study supports this view that protection of the health of non-smokers, particularly in enclosed public spaces, must be given priority as an issue of environmental health protection. To our knowledge, this is the first report of an increased risk of CHD due to PS in Iran and supports the present public health argument of prohibiting smoking in enclosed spaces. Legislation is presently in effect in Iran to this end. It also confirms the previous findings of an elevated risk of CHD associated with PS at home.

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