Passive smoking at work as a risk factor for coronary heart disease in Chinese women who have never smoked

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Abstract

Objective—To study whether passive smoking at work is a risk factor for coronary heart disease.

Design—Case-control study.

Setting—Xi'an, China.

Subjects—59 patients with coronary heart disease and 126 controls, all Chinese women with full time jobs, who had never smoked cigarettes.

Results—The crude odds ratio for passive smoking from husband was 2.12 (95% confidence interval 1.06 to 4.25) and at work was 2.45 (1.23 to 4.88). The final logistic regression model, with passive smoking from husband and at work as the base, included age, history of hypertension, type A personality, and total cholesterol and high density lipoprotein cholesterol concentrations; the adjusted odds ratios for passive smoking from husband and at work were 1.24 (0.56 to 2.72) and 1.85 (0.86 to 4.00) respectively. For passive smoking at work, statistically significant linear trends of increasing risks (for both crude and adjusted odds ratios) with increasing exposures (amount exposed daily, number of smokers, number of hours exposed daily, and cumulative exposure) were observed. When these exposure variables were analysed as continuous variables, the crude and adjusted odds ratios were also significant.

Conclusion—Passive smoking at work is a risk factor for coronary heart disease. Urgent public health measures are needed to reduce smoking and to protect non-smokers from passive smoking in China.

Introduction

Passive smoking has been confirmed as a cause of lung cancer in adults and respiratory ill health in children,¹ but the role of passive smoking in causing coronary heart disease is still unsettled. Evidence that passive smoking is associated with coronary heart disease is increasing. Glantz and Parmley reviewed the results of 10 epidemiological studies, together with physiological and biochemical data, and concluded that passive smoking causes heart disease.² The same conclusion was reached in a review by Taylor *et al.*³ There have since been two case-control studies, both associating passive smoking with increased risk of coronary heart disease.^{4,5}

As for the source of passive smoking, most previous studies reported associations with exposure from spouse exclusively. Of the 12 studies (eight prospective and four case-control studies) mentioned above, only two case-control studies presented data on passive smoking at work. In Britain Lee *et al* found no increase in risk for a combined index of passive smoking which included exposures at home, at work, during travel, and during leisure. This study had a small sample size, only 30 cases in men and 36 in women.⁶ In Australia Dobson *et al* found increased odds ratios for passive smoking at home for men and women, but the odds ratios for passive smoking at work did not suggest increased risk.⁴ This study had a larger sample, 174 cases in men but only 27 in women.

Steenland recently reviewed nine epidemiological

studies, pointing out that the lack of data on exposures to environmental tobacco smoke outside the home was one of the major weaknesses in the epidemiological evidence.⁷ Glantz and Parmley also considered that the studies they reviewed underestimated the risk because exposures at home are generally smaller than exposures at work.²

In China, our earlier study on non-smoking women in Xi'an included 34 cases and 68 controls.⁸ It found an adjusted odds ratio for passive smoking from husband of 1.50 (95% confidence interval 1.28 to 1.77). However, this study did not include details of exposure for passive smoking at work. The present study is a second case-control study which aims to study whether passive smoking at work is a risk factor for coronary heart disease in women who have never smoked. It takes into account passive smoking from the husband and other risk factors.

Subjects and methods

The cases were patients with coronary heart disease (non-fatal, incident cases) from the three large teaching hospitals of two medical universities in Xi'an between December 1989 and November 1992. The final diagnosis was myocardial infarction according to WHO criteria⁹ or coronary stenosis confirmed by coronary arteriography. Coronary arteriography was performed using Judkins's technique¹⁰ and analysed by two experienced cardiologists independently. The criterion of diagnosis of coronary heart disease was a finding of more than 50% occlusion in at least one of the major arteries. The agreement between the two radiologists was over 90%. Patients with occlusion of 50% or less were excluded.

The controls were from three sources: patients admitted because of suspected or diagnosed coronary heart disease but confirmed to be normal after coronary arteriography (no coronary stenosis at all); other medical outpatients attending cardiology departments (patients with psychosomatic symptoms, menopausal syndrome, dysrhythmia, or non-cardiac chest pain); and a random sample of healthy subjects from a community screening programme for coronary heart disease. The latter two groups were confirmed to be free of coronary heart disease by WHO criteria⁹ and normal exercise electrocardiography.

A standardised questionnaire was designed to collect information on demographic characteristics (such as ethnic origin, age, residential history, educational level, occupation, and marital status); history of hypertension, hyperlipidaemia and diabetes mellitus; family history of hypertension, stroke, and coronary heart disease; history of smoking and passive smoking from husband and at work; drinking history; exercise; and psychosocial factors (such as Type A personality, experience of mental trauma, and stressful life events). The interviews were carried out by three trained interviewers, before coronary arteriography for patients scheduled for coronary arteriography or during recovery for those with myocardial infarction. The non-response rate was 8%.

Physical examinations followed standard methods

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and included height, weight, body mass index, systolic and diastolic blood pressure, chest radiography, and electrocardiography. Laboratory investigations included serum concentrations of total cholesterol, triglycerides, low density lipoprotein cholesterol, high density lipoprotein cholesterol, and apolipoprotein A-1 and B and tests of liver and renal function.

Only ethnic Chinese women who had never smoked (lifelong non-smokers) and who had a full time job were included (full time housewives, peasants, and those who were retired for five years or more were excluded).

Passive smoking from husband was defined as living with a smoking husband for over five years. Single women would have been considered as not exposed, but all subjects were found to be married. Passive smoking at work was defined as working with smoking coworkers in the same office or factory unit for over five years. Periods away from the work environment were considered as no exposure in the workplace. All subjects were found to be either not exposed or exposed for over five years.

TABLE I—Characteristics of cases and controls

Characteristic	No (%) of cases (n=59)	No (%) of controls (n=126)	χ^2	P value
Age (years):				
36-45	1 (2)	6 (5)		
46-55	19 (32)	61 (48)		
≥ 56	39 (66)	59 (47)	6.26	0.044
Marital status:				
Married	53 (90)	119 (94)		
Divorced	1 (2)	2 (2)		
Widowed	5 (9)	5 (4)	1.60	0.45
Occupation:				
Intellectual	16 (27)	34 (27)		
Cadre	10 (17)	31 (25)		
Factory worker	33 (56)	61 (48)	1.51	0.47
Education (years):				
0-3	1 (2)	1 (1)		
4-6	15 (25)	26 (21)		
7-9	16 (27)	34 (27)		
10-12	8 (14)	33 (26)		
≥ 13	19 (32)	32 (25)	4.29	0.37

TABLE II—Exposure to passive smoking in 91 controls and cases

	No (%) exposed to passive smoking from husband	No (%) exposed to passive smoking at work
Controls:		
No coronary arteriography or coronary heart disease (n=65)	29 (45)	21 (32)
Misdiagnosed as having coronary heart disease (n=26)	12 (46)	8 (31)
Cases (n=59)	38 (64)	33 (56)

TABLE III—Passive smoking from husband and at work and crude odds ratios of coronary heart disease

	No of cases	No of controls	Crude odds ratio (95% confidence interval)	χ^2 (corrected)	P value
Passive smoking from husband:					
No	21	68	1.00		
Yes	38	58	2.12 (1.06 to 4.25)	4.72	0.030
Passive smoking at work:					
No	26	83	1.00		
Yes	33	43	2.45 (1.23 to 4.88)	7.02	0.008

Quality controls for interviewing included tape-recording of the interviews (for 10% of hospital subjects), interviewing the husband to validate the data from the wife (for one third of hospital subjects), and single blind reinterview by a second interviewer who was not aware of the case-control status of the subjects (for 30% of hospital subjects). Furthermore, 26 patients had initially been diagnosed as having coronary heart disease and were interviewed before coronary arteriography but were subsequently confirmed by coronary arteriography to be normal. This group of "misdiagnosed" patients was accepted as controls and their exposure to passive smoking was compared with that of controls and cases who had not had coronary arteriography to check for subjective bias due to interviewers.

For the calculation of sample size required, it should be noted that the crude odds ratio for passive smoking from husband found in our previous study was 3.0.⁸ Assuming the proportion of exposure to passive smoking at work in the controls was 35%, for a significance of 5% and a power of 80%, 58 cases and 116 controls (one case to two controls) were required to detect a crude odds ratio of 2.5.

The data were managed and analysed by using the computer packages of Epi-Info (5.0) and SPSS-PC (3.1). The statistical procedures used included κ , t test, χ^2 test, χ^2 test for trend, and standard multivariate techniques for unmatched case-control studies (stratified analysis and multiple logistic regression analysis).¹¹

The present study included 59 cases of coronary heart disease (34 were confirmed by coronary arteriography and 25 with myocardial infarction) and 126 controls (61 confirmed as negative on coronary arteriography, 28 outpatients in cardiology departments, and 37 from community screening). Ages of cases ranged from 37 to 67 years and of controls from 42 to 66 years. About 70% of all eligible cases of coronary heart disease treated in the three hospitals were included.

Results

The characteristics of the controls from the three sources were compared and no significant differences were found. The controls were therefore combined in subsequent analyses. Table I shows that the cases and controls were similar in marital status, occupation, and education but there were more older subjects among the cases. The mean (SD) age of the cases (58.0 (5.39) years) was greater than that of the controls (55.0, (5.05) years; $t=3.69$, $P=0.002$). All the subjects had lived in Xi'an for more than 20 years.

Results on single blind test-retest by two interviewers on 35 hospital subjects (16 cases and 19 controls) showed good agreement, ranging from 75% to 95% for the 10 risk factors tested, with κ values ranging from 0.4 to 0.8 (nine κ values with $P < 0.01$ and one with $P < 0.05$; data not shown).

Table II shows that the exposure to passive smoking in the misdiagnosed controls was similar to that of the controls shown not to have coronary heart disease but different from that of the cases. There was no history of excess exposure in the misdiagnosed groups, suggesting that there was no subjective bias in the interviews.

For passive smoking from husband, the crude odds ratio was 2.12 (1.06 to 4.25) (table III). The sizes of the living quarters of cases and controls who were exposed to passive smoking from husband were not significantly different. Table III also shows that the crude odds ratio for passive smoking at work was 2.45 (1.23 to 4.88). The statistical power, calculated from table III with a significance level of 5%, was 79%.

Table IV shows the results of stratified analysis for the two sources of passive smoking. The odds ratio for

TABLE IV—Stratified analysis for passive smoking from husband and at work

Passive smoking		No of cases	No of controls	Crude odds ratio (95% confidence interval)	χ^2 (corrected)	P Value
From husband	At work					
No	No	11	50	1.00		
Yes	No	15	33	2.07 (0.78 to 5.55)	1.91	0.17
No	Yes	10	18	2.53 (0.82 to 7.83)	2.42	0.12
Yes	Yes	23	25	4.18 (1.63 to 10.92)	9.83	0.002

Overall $\chi^2=11.27$, $df=3$, $P=0.01$.

Test for trend: $\chi^2=10.95$, $P<0.001$.

combined exposure to both sources, 4.18, was slightly higher than expected from the additive model ($2.07 + 2.53 - 1 = 3.60$) but much less than expected from the multiplicative model ($2.07 \times 2.53 = 5.24$). The crude odds ratio for any exposure (from husband or at work, or both) was 2.87 (1.28 to 6.55).

Table V shows the final model of logistic regression analysis which included passive smoking from husband and at work as the base (these two variables were included in the model before other variables were entered and tested) and age, history of hypertension, type A personality, and high density lipoprotein cholesterol concentration. Other risk factors had also been tested by forward and backward stepwise procedures but none affected the model significantly. The interaction term of the two passive smoking risk factors (passive smoking from husband multiplied by passive smoking at work) was also tested but was not significant. The adjusted odds ratio for passive smoking from husband was 1.24 (0.56 to 2.72) and at work was 1.85 (0.86 to 4.00). Both adjusted odds ratios were smaller than the respective crude odds ratios and became non-significant after the other five risk factors were included in the final model. When passive smoking was removed from the logistic model the adjusted odds

ratio for passive smoking at work was slightly higher (1.92) but was still not significant. However, the adjusted odds ratio for any exposure (from husband or at work, or both) was significant (2.36; 1.01 to 5.55).

The relation between odds ratios and amount of exposure to passive smoking from husband and at work were examined. For passive smoking from husband, the crude odds ratios showed significant linear trends with amount smoked daily by husband, duration of exposure, and cumulative exposure (amount daily multiplied by duration), but the trends became non-significant after passive smoking at work and the other five risk factors were adjusted for in the final model (data not shown). However, for passive smoking at work, table VI shows that significant linear trends were found for the crude odds ratios in amount smoked daily, duration of exposure, number of smokers, exposure time daily, and cumulative exposure. For adjusted odds ratios, the linear trends were significant for all the variables except duration of exposure. When passive smoking from husband was removed from the logistic model, the odds ratios for passive smoking at work were only slightly higher, suggesting that the effects of multicollinearity of the two exposure variables were small. Table VII shows similar results when these variables were analysed as continuous variables, and table VIII shows that there were significant trends for the two indices of combined exposure from husband and at work.

TABLE V—Models of logistic regression analysis of risk factors

	Adjusted odds ratio (95% confidence interval)*	P value
Final model (factors 1 to 7):		
1 Age (years)	1.13 (1.04 to 1.22)	0.003
2 History of hypertension	2.47 (1.14 to 5.36)	0.022
3 Type A personality	2.83 (1.31 to 6.37)	0.008
4 Total cholesterol (mg/dl)	1.02 (1.01 to 1.03)	0.0006
5 High density lipoprotein cholesterol (mg/dl)	0.94 (0.90 to 0.98)	0.003
6 Passive smoking from husband	1.24 (0.56 to 2.72)	0.60
7 Passive smoking at work	1.85 (0.86 to 4.00)	0.12
Other model (factors 1 to 5 and passive smoking at work)	1.95 (0.90 to 4.10)†	0.087
Other model (factors 1 to 5 and passive smoking from husband or at work, or both)	2.36 (1.01 to 5.55)†	0.049

*Adjusted for the other variables in the final model.

†Adjusted for the first five variables above; odds ratios for these variables in the other models were essentially the same as those shown above and are not shown.

Discussion

The outcome or end point of this study is non-fatal incident cases of coronary heart disease. This is similar to those in the previous four case-control studies on passive smoking and coronary heart disease.^{4,6,8} Most previous prospective studies used mortality as the outcome measure. Despite the difference in end point, the relative risks for passive smoking were quite similar.² Although there may be prevalence-incidence bias in using non-fatal cases, the obvious advantage is that the patients can provide more and better information on exposure and other confounding factors.

Questionnaires or interviews are the key instruments in studies on passive smoking. Cummings *et al*

TABLE VI—Passive smoking at work and odds ratios of coronary heart disease

	No of cases	No of controls	Crude odds ratio (95% confidence interval)	First adjusted odds ratio (95% confidence interval)*	P value (global test)	P value (linear trend)	Second adjusted odds ratio†
Passive smoking at work							
No of cigarettes/day smoked by coworkers:							
0-5 (actually zero)	26	83	1.00				
6-10	10	25	1.28 (0.50 to 3.24)	0.87 (0.30 to 2.53)			0.94
11-20	15	14	3.42 (1.35 to 8.74)	2.95 (1.05 to 8.28)	0.019	0.022	3.04
≥ 20	8	4	6.38 (1.57 to 27.84)	3.56 (0.81 to 15.58)			3.68
Test for trend			$\chi^2 = 14.15, P < 0.001$				
Duration of exposure (years):							
0-5 (actually zero)	26	83	1.00				
6-15	8	12	2.13 (0.70 to 6.40)	3.08 (0.90 to 10.58)			3.20
≥ 16	25	31	2.57 (1.23 to 5.42)	1.56 (0.67 to 3.64)	0.24	0.12	1.62
Test for trend			$\chi^2 = 7.73, P < 0.005$				
No of smokers:							
0	26	83	1.00				
1-2	16	36	1.42 (0.64 to 3.15)	1.16 (0.48 to 2.82)			1.23
3	12	6	6.38 (1.97 to 21.51)	5.06 (1.42 to 18.02)	0.019	0.024	5.17
≥ 4	5	1	15.96 (1.67 to 378.10)	4.11 (0.39 to 43.68)			4.50
Test for trend			$\chi^2 = 17.54, P < 0.001$				
Exposure time daily (hours):							
0	26	83	1.00				
1-2	8	30	0.85 (0.31 to 2.25)	0.62 (0.22 to 1.80)			0.65
3-4	15	11	4.35 (1.164 to 11.73)	4.03 (1.33 to 12.25)	0.001	0.002	4.16
≥ 5	10	2	15.96 (2.98 to 113.32)	21.32 (2.71 to 168.00)			22.49
Test for trend			$\chi^2 = 21.58, P < 0.0001$				
Cumulative exposure (cigarettes/day × years × smokers × hours):							
0	26	83	1.00				
1-2000	13	32	1.30 (0.55 to 3.03)	1.00 (0.39 to 2.57)			1.05
2001-4000	5	8	2.00 (0.51 to 7.55)	2.05 (0.47 to 8.87)	0.005	0.003	2.11
≥ 4001	15	3	15.96 (3.89 to 75.27)	9.23 (2.01 to 42.25)			9.63
Test for trend			$\chi^2 = 20.76, P < 0.001$				

*Adjusted for age, history of hypertension, personality type, total cholesterol, high density lipoprotein cholesterol, and passive smoking from husband.

†Adjusted for variables above, except for passive smoking from husband. The 95% confidence intervals are all slightly higher and the P values are all slightly lower than those for first adjusted odds ratio and are not shown.

TABLE VII—Passive smoking at work as continuous variables and odds ratios of coronary heart disease

Passive smoking at work as continuous variables	Crude odds ratio (95% confidence interval)	First adjusted odds ratio* (95% confidence interval)	Second adjusted odds ratio†
Amount daily (cigarettes/day)	1.068 (1.035 to 1.106)	1.050 (1.007 to 1.094)	1.051
Duration of exposure (years)	1.047 (1.018 to 1.076)	1.027 (0.993 to 1.061)	1.028
No of smokers	1.633 (1.268 to 2.104)	1.425 (1.048 to 1.937)	1.440
Exposure time daily (hours)	1.480 (1.234 to 1.775)	1.447 (1.145 to 1.829)	1.454
Cumulative exposure (cigarettes/day × years × smokers × hours/100)	1.038 (1.020 to 1.056)	1.031 (1.011 to 1.052)	1.032

*Adjusted for age, history of hypertension, personality type, total cholesterol, high density lipoprotein cholesterol, and passive smoking from husband.

†Adjusted for variables above, except for passive smoking from husband. The 95% confidence intervals are all slightly higher than those for the first adjusted odds ratios and are not shown.

TABLE VIII—Combined exposure to passive smoking from husband and at work and odds ratios of coronary heart disease

Indices of combined exposure	Adjusted odds ratio (95% confidence interval)*	P value (global test)	P value (linear trend)
Index A (cigarettes/day × years)†:			
0	1.00		
1-499	1.79 (0.65 to 4.92)		
500-999	2.55 (0.92 to 7.10)	0.14	0.022
≥ 1000	3.95 (1.03 to 15.23)		
Index B‡:			
Score 0 (nil)	1.00		
Score 1 (low exposure)	1.75 (0.68 to 4.46)		
Score 2 (moderate exposure)	3.11 (1.06 to 9.12)	0.059	0.008
Score 3 (high exposure)	7.61 (1.15 to 50.18)		

*Adjusted for age, history of hypertension, personality type, total cholesterol, and high density lipoprotein cholesterol.

†Sum of cumulative exposure from husband (cigarettes/day × years) and an index of exposure at work (amount exposed/day × duration of exposure).

‡Cumulative exposure from husband (cigarettes/day × years) was recoded into four categories (score 0=no exposure, score 1=1-399, score 2=400-799, score 3≥800), and cumulative exposure at work (cigarettes/day × years × smokers × hours) was recoded into four categories (score 0=no exposure, score 1=1-2000, score 2=2001-4000, score 3≥4001). The two scores were added (range 0-6) and recoded into 4 categories (score 0=no exposure, score 1=1 to 2, score 2=3 to 4, score 3=5 to 6) to form Index B.

showed that subjects were able to report an accurate history of exposure to passive smoking: the level of agreement between subjects and surrogates on exposure to tobacco smoke at work was 78% ($\kappa=0.5$); the level of agreement for exposure to spouse smoking was 86% ($\kappa=0.7$).¹² We used the test-retest method with two interviewers: the agreement for passive smoking at work was 74.3% ($\kappa=0.5$, $P<0.01$), and for passive smoking from husband the agreement was 91.4% ($\kappa=0.8$, $P<0.01$). We did not interview subjects again on quantity of exposure, but for this aspect the agreement should be lower.

Validation by cotinine testing was not possible for past exposures in case-control studies. The analysis for trend should yield better evidence for causal inference of whether or not there is increased risk due to exposure but would not be precise enough for risk assessment for unit dose of exposure.

Because different sources of subjects might be a potential source of bias in the present study, exposures to passive smoking in each of the two series of cases (those confirmed by coronary arteriography and by myocardial infarction) were compared with exposures in each of the three sources of controls (cases confirmed negative on coronary arteriography, outpatients, and community controls). The crude odds ratios for passive smoking from husband ranged from 2.0 to 2.3 for cases confirmed by coronary arteriography and from 2.0 to 2.2 for those confirmed by myocardial infarction; for passive smoking at work, the crude odds ratios ranged from 1.8 to 3.6 and from 1.9 to 3.6 respectively. As the results of separate analysis were consistent, pooled analysis was justified.

Results of various quality control measures on selected subjects suggested that the data should be reasonably reliable and free from bias. Moreover, people in Xi'an were unaware of the issue of passive smoking, and bias due to overreporting of exposure in coronary heart disease patients was unlikely. The bias

due to misclassification of current and ex-smokers as lifelong non-smokers should be small because the prevalence of female smokers in China was very low as compared with Western countries. The prevalence of regular smokers in females aged 40-65 was 8.6% in the province of Shaanxi, of which Xi'an is the capital.¹³

Both this and our previous studies showed that passive smoking from husband was associated with coronary heart disease. The crude odds ratio of 2.12 (1.06 to 4.25) in the present study was smaller but consistent with the value of 3.00 (1.26 to 7.17) in the previous study.⁸ Although the adjusted odds ratio of 1.24 (0.56 to 2.72) was not statistically significant, it was consistent with the previous adjusted odds ratio of 1.50 (1.28 to 1.77). The variables adjusted were not the same in the two studies, and passive smoking at work was adjusted in the present study but not in the previous one. Thus the results of these two studies, together with the evidence from other countries, are strong evidence suggesting that passive smoking from the husband is likely to be a causal factor for coronary heart disease in women in Xi'an who have never smoked.

To the best of our knowledge, this is the first study showing an increased risk of coronary heart disease with increasing exposure to passive smoking at work (crude and adjusted odds ratios were 2.45 (1.23 to 4.88) and 1.85 (0.86 to 4.00) respectively). With the exception of number of years of exposure, the linear trends for amount exposed daily, number of smokers, number of hours exposed daily, and cumulative exposure were all statistically significant. These results strongly suggest that passive smoking at work is a risk factor for coronary heart disease, even after passive smoking from husband is taken into account. When passive smoking from husband was not included, the adjusted odds ratios were slightly higher.

Comparing the odds ratios for the two sources of environmental tobacco smoke, the crude and adjusted odds ratios for passive smoking at work were slightly greater than those for passive smoking from husband (although the 95% confidence intervals overlapped). If these were not due to chance, the higher risk for passive smoking at work may be explained by the fact that exposures to tobacco smoke at work are higher than exposures at home because the density of smokers is higher at work, and the length of stay at work for a working woman is longer than the time she is exposed to her husband's smoking at home. Stratified analysis in the present study suggests that the effects of the two are additive and that there is no significant interaction. Because the prevalence of smoking among men is high in China and most men smoke freely at home and at work, women are heavily exposed to environmental tobacco smoke and the magnitude of the risks could be quite high.

To conclude, the present study provides further evidence that environmental tobacco smoke, including smoke at work, is likely to be a cause of coronary heart disease. The weaknesses of the present study are recognised and further studies are required to investigate the role of passive smoking at work and its interaction with passive smoking at home and from other sources. However, China is the largest producer and consumer of tobacco in the world—30% of the world's cigarettes are consumed by China's 300 million smokers. In 1984, the prevalence of smoking in the population aged 15 and above was 61% in men and 7% in women. Forty per cent of non-smokers were exposed to environmental tobacco smoke for over 15 minutes a day: 67% of the passive smokers were exposed at home, 14% at work or in public places, and 19% both at home and at work.¹³ Smoking among young people has increased in recent years.¹⁴ Despite the efforts to control smoking, most women are still

Clinical implications

- Whether passive smoking is a cause of coronary heart disease is still controversial
- Previous studies have shown that coronary heart disease is associated with passive smoking at home but not with passive smoking at work
- This study shows that for women's passive smoking at work there were significant linear trends of increased risks of coronary heart disease with increasing exposures, even after adjustment for major risk factors and passive smoking from husband
- Urgent public health measures are needed to reduce smoking in China

exposed to tobacco smoke at home and at work. They are also unaware of the hazards of passive smoking and are in a disadvantaged position to protect themselves from environmental tobacco smoke. Urgent public health measures are required to reduce smoking in China so as not only to protect women from environmental tobacco smoke but also men from the hazards of active smoking.

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Passive exposure to tobacco smoke in children aged 5-7 years: individual, family, and community factors

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Abstract

Objective—To examine the importance of parental smoking on passive exposure to tobacco smoke in children and the social and geographical patterns of exposure.

Design—Cross sectional study.

Setting—Schools in 10 towns in England and Wales; five towns with high adult cardiovascular mortality and five with low rates.

Subjects—4043 children aged 5-7 years of European origin.

Main outcome measures—Salivary cotinine concentration and parents self reported smoking habits.

Results—1061 (53.0%) children were exposed to cigarette smoke at home or by an outside carer. Geometric mean cotinine rose from 0.29 (95% confidence interval 0.28 to 0.31) ng/ml in children with no identified exposure to 4.05 (3.71 to 4.42) ng/ml in households where both parents smoked and 9.03 (6.73 to 12.10) ng/ml if both parents smoked more than 20 cigarettes a day. The effect of mothers' smoking was greater than that of fathers', especially at high levels of consumption. After adjustment for known exposures geometric mean cotinine concentrations rose from 0.52 ng/ml in social class I to 1.36 ng/ml in social class V ($P < 0.0001$); and were doubled in high mortality towns compared with the low mortality towns ($P = 0.002$). In children with no identified exposure similar trends by social class and town were observed and the cotinine concentrations correlated with the prevalence of parental smoking, both between towns ($r = 0.69$, $P = 0.02$) and between schools within towns ($r = 0.50$, $P < 0.001$).

Conclusions—Mothers' smoking is more important than fathers' despite the lower levels of smoking by mothers. Children not exposed at home had low cotinine concentration, the level depending on the prevalence of smoking in the community.

Introduction

In the past decade many studies have investigated the health effects of passive exposure to tobacco smoke in non-smoking children.¹ Most studies have relied on questionnaire measures of parental smoking as the indicator of exposure. Such measures ignore exposure by people from outside the household, the extent to which parents smoke in the presence of the child, and other modifying factors such as the ventilation of the room. These studies may therefore have underestimated the real effect of passive smoking in children. Biochemical measures allow recent exposure to be estimated directly.

Cotinine, a metabolite of nicotine, is the best biochemical marker for quantifying passive exposure to smoke.² It is specific to tobacco, has a half life of about 20 hours, and can be detected at low concentrations by gas-liquid chromatography.³ Salivary concentrations are in approximate equilibrium with those in the blood⁴ and provide a non-invasive way of measuring passive smoke exposure. We present an analysis of the relation between cotinine concentration and questionnaire data in children aged 5-7 from 10 towns in England and Wales. We examine the importance of parental exposure as opposed to other sources; exposure among children from non-smoking households; and the social and geographical

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