for specialist paediatric care. Secondly, evidence of the effectiveness of this treatment came from Chinese sources that are not routinely searched when systematic reviews are carried out in the West. To determine whether the inclusion of Chinese and Russian trials would reinforce or change the conclusions of systematic reviews, Chinese and Russian trials of interventions should be reviewed and the results compared with currently available systematic reviews.

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Environmental tobacco smoke and mortality in Chinese women who have never smoked: prospective cohort study

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Abstract

Objective To evaluate the association of environmental exposure to tobacco smoke from husbands and from work, as well as from family members in early life, with all cause mortality and mortality due to cancer or cardiovascular disease in Chinese women.

Design Ongoing prospective cohort study in Shanghai, China.

Participants Of 72 829 women who had never smoked, 65 180 women provided information on smoking by their husbands, and 66 520 women provided information on exposure to tobacco smoke at work and in early life from family members.

Main outcome measures All cause mortality and cause specific mortality with the main focus on cancer and cardiovascular disease. Cumulative mortality according to exposure status, and hazard ratios.

Results Exposure to tobacco smoke from husbands (mainly current exposure) was significantly associated with increased all cause mortality (hazard ratio 1.15, 95% confidence interval 1.01 to 1.31) and with increased mortality due to cardiovascular disease (1.37, 1.06 to 1.78). Exposure to tobacco smoke at work was associated with increased mortality due to cancer (1.19, 0.94 to 1.50), especially lung cancer (1.79, 1.09 to 2.93). Exposure in early life was associated with increased mortality due to cardiovascular disease (1.26, 0.94 to 1.69).

Conclusions In Chinese women, exposure to environmental tobacco smoke is related to moderately increased risk of all cause mortality and mortality due to lung cancer and cardiovascular disease.

Introduction

The excess risk of coronary heart disease and lung cancer from environmental tobacco smoke is estimated at about 15-35%.[1-3] Studies on the relation between environmental tobacco smoke and mortality are few and most have limited statistical power.[4 5]

We used data from the Shanghai women's health study—an ongoing prospective cohort study—to examine the association of environmental tobacco smoke with mortality. The rate of smoking in Chinese men is high, but most Chinese women living in Shanghai do not smoke. For the causes of death, we focused on cancer, especially lung cancer, which is most closely related to tobacco smoke, and cardiovascular disease, particularly stroke, a leading cause of death and disability in China.

Methods

Shanghai women's health study

Eligible women (n = 81 170) were aged 40-70 and lived in seven areas of urban Shanghai.[6] In total, 75 220 women (92.7%) completed the baseline survey, which included a face to face interview and a self administered questionnaire, from March 1997 to May 2000. Subsequently, 278 (0.4%) women outside the eligible age range were excluded, leaving 74 942 women in the baseline cohort. The cohort has been followed up every two years by face to face interviews. Data are also obtained from the Shanghai Cancer Registry and

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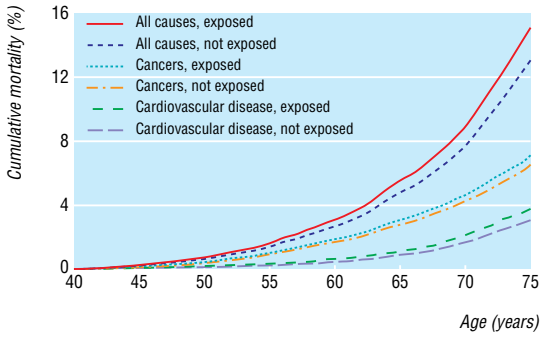

Mortality and exposure to husband's tobacco smoke in Chinese women who had never smoked

the Shanghai Death Registry at the end of each year as another way to obtain cancer and death outcomes. Only 2113 of these women (2.8%) had ever smoked, and they were excluded from our analysis.

The exposure and outcome

The baseline survey asked participants questions about smoking and other lifestyle factors, for themselves and for their husbands. Exposure was classified as current exposure (if the husband still smoked) or former exposure (if he had stopped smoking). Exposure was measured by the number of pack years that the husband had smoked during the marriage. The baseline survey also recorded demographic details and disease history, reproductive history, family history, and dietary history.

The first follow-up survey was conducted from 2000 to 2002 and the second from 2002 to 2004. Only 934 (1.3%) women were lost to follow-up at the end of the second follow-up survey. About 99.8% of the participants (or their next of kin if they had died) were interviewed to obtain information on death and common diseases diagnosed after the baseline survey. Of 72 983 living participants, 67 163 (92.1%) completed the lifestyle module of the questionnaire through a face to face interview. The status and total years of environmental exposure to tobacco smoke at work and in early life from family members were assessed (see bmj.com).

Statistical analysis

The end date of the observation was set as the date of death for participants who had died, or the date of the last follow-up or 30 June 2004 for those who were still alive—half a year ahead of the last annual linkage with the Shanghai Death Registry, whichever was later.

We used the Cox proportional hazards regression model to estimate hazard ratios and 95% confidence intervals. Survival was modelled as a function of age.

Association between mortality and exposure to environmental tobacco smoke in the Shanghai women's health study. Values are hazard ratios and 95% confidence intervals*

		Cause of death			
Type of exposure	**All causes of death**	**Cancer (all types)**	**Lung cancer**	**Cardiovascular disease**	**Stroke**
Husband† (n=65 180)					
Ever exposed	1.11 (0.99 to 1.25)	1.08 (0.91 to 1.27)	1.09 (0.74 to 1.61)	1.18 (0.92 to 1.51)	1.33 (0.96 to 1.84)
Former	1.01 (0.85 to 1.22)	1.15 (0.90 to 1.47)	1.15 (0.65 to 2.04)	0.75 (0.49 to 1.12)	0.92 (0.54 to 1.52)
Current	1.15 (1.01 to 1.31)	1.05 (0.88 to 1.26)	1.06 (0.69 to 1.63)	1.37 (1.06 to 1.78)	1.52 (1.08 to 2.15)
Pack years of exposure:					
<8.8	1.14 (0.94 to 1.38)	1.26 (0.98 to 1.63)	1.62 (0.86 to 2.70)	1.10 (0.72 to 1.69)	1.35 (0.79 to 2.31)
8.8-17.9	1.09 (0.91 to 1.31)	1.03 (0.80 to 1.33)	1.39 (0.81 to 2.39)	1.12 (0.76 to 1.64)	1.25 (0.76 to 2.07)
≥18.0	1.11 (0.97 to 1.28)	1.02 (0.84 to 1.25)	0.79 (0.48 to 1.31)	1.22 (0.93 to 1.61)	1.36 (0.94 to 1.96)
Trend test	P=0.155	P=0.811	P=0.589	P=0.080	P=0.029
Workplace† (n=66 520)					
Ever exposed	1.08 (0.91 to 1.28)	1.19 (0.94 to 1.50)	1.79 (1.09 to 2.93)	0.92 (0.64 to 1.32)	0.73 (0.44 to 1.20)
Years of exposure:					
<10	1.04 (0.79 to 1.37)	1.03 (0.70 to 1.51)	1.58 (0.73 to 3.45)	0.86 (0.47 to 1.57)	0.47 (0.17 to 1.29)
10-24	1.06 (0.82 to 1.38)	1.20 (0.85 to 1.70)	1.28 (0.57 to 2.87)	0.96 (0.56 to 1.65)	0.95 (0.47 to 1.90)
>24	1.14 (0.89 to 1.45)	1.31 (0.95 to 1.81)	2.45 (1.32 to 4.56)	0.93 (0.54 to 1.58)	0.74 (0.35 to 1.56)
Trend test	P=0.292	P=0.076	P=0.003	P=0.864	P=0.520
In early life† (n=66 520)					
Ever exposed	0.98 (0.85 to 1.14)	0.79 (0.63 to 0.98)	0.88 (0.55 to 1.43)	1.26 (0.94 to 1.69)	1.10 (0.74 to 1.63)
Years of exposure:					
<20	0.96 (0.80 to 1.14)	0.77 (0.59 to 1.01)	0.88 (0.50 to 1.55)	1.21 (0.86 to 1.70)	0.91 (0.56 to 1.47)
≥20	1.03 (0.83 to 1.28)	0.81 (0.59 to 1.11)	0.89 (0.45 to 1.77)	1.36 (0.90 to 2.05)	1.46 (0.86 to 2.45)
Trend test	P=0.807	P=0.077	P=0.980	P=0.029	P=0.342
All 3 sources (n=59 675)					
Ever exposed	1.15 (0.95 to 1.41)	1.06 (0.80 to 1.40)	1.03 (0.57 to 1.87)	1.45 (0.95 to 2.22)	1.64 (0.91 to 2.95)
Source of exposure:					
Husband only	1.26 (0.99 to 1.59)	1.25 (0.90 to 1.74)	0.89 (0.42 to 1.92)	1.52 (0.92 to 2.50)	2.21 (1.15 to 4.24)
Early life only	0.89 (0.61 to 1.28)	0.41 (0.20 to 0.82)	0.21 (0.03 to 1.61)	1.82 (0.96 to 3.43)	1.86 (0.77 to 4.46)
Workplace only	1.28 (0.92 to 1.79)	1.71 (1.13 to 2.58)	2.23 (0.95 to 5.27)	0.80 (0.32 to 1.96)	0.49 (0.11 to 2.18)
Husband+early life	1.07 (0.82 to 1.40)	0.91 (0.61 to 1.35)	0.81 (0.34 to 1.94)	1.42 (0.82 to 2.45)	1.25 (0.56 to 2.77)
Husband+workplace	0.99 (0.69 to 1.42)	0.81 (0.48 to 1.36)	1.24 (0.44 to 3.51)	1.30 (0.62 to 2.74)	1.47 (0.55 to 3.91)
Early life+workplace	1.13 (0.74 to 1.72)	1.00 (0.56 to 1.79)	0.77 (0.17 to 3.43)	1.11 (0.42 to 2.93)	0.88 (0.20 to 3.91)
All	1.38 (1.00 to 1.89)	1.14 (0.73 to 1.79)	2.17 (0.94 to 5.03)	1.74 (0.89 to 3.40)	2.16 (0.91 to 5.13)

*Derived from Cox regression models with age as the time to scale; also adjusted for education, occupation, family income, physical activity, body mass index, and intake of meat, vegetables, and fruit.
†Regardless of whether exposed to other sources.

Left truncation was set to the date of the baseline survey (for exposure to smoking by husbands) or the date of the first follow-up survey (for exposure at work, in early life from family members, and all three sources of exposure combined). Covariates included in the model were education, occupation, family income, physical activity, body mass index, and intake of meat, vegetables, and fruit. See bmj.com for details.

Results

Baseline characteristics of the 72 829 women who had never smoked are shown on bmj.com; 89% were married. The number of participants and number of deaths varied when different exposure variables were analysed. Cancer and cardiovascular disease were two of the most common causes of death. Stroke was the leading cause of death due to cardiovascular disease.

Exposure to environmental tobacco smoke was prevalent; 83.1% (49 563/59 675) of women were exposed to environmental tobacco smoke from their husbands, at work, or in early life (or a combination). Cumulative all cause mortality was significantly higher in women whose husbands smoked than in those whose husbands did not smoke (9.1% *v* 7.8% at age 70; P = 0.009), but other sources of environmental tobacco smoke were not associated with significantly higher all cause mortality. For cause specific mortality with the competing risk accounted for, cumulative mortality due to cardiovascular disease was significantly increased in women whose husbands smoked (2.2% *v* 1.7% at age 70; P = 0.048) (figure).

The hazard ratios adjusted for potential confounders were similar to those where only age was adjusted for (table 1). Exposure from husbands (mainly current exposure) was associated with significantly increased all cause mortality, and more strongly with increased mortality due to cardiovascular disease and stroke. Exposure to tobacco smoke at work was associated with increased mortality due to cancer, especially lung cancer. Exposure in early life from family members was associated with increased mortality due to cardiovascular disease. We also analysed the effect of duration of exposure to environmental tobacco smoke on mortality. Significant dose-response relations existed between mortality due to stroke and husband's number of pack years (P = 0.029), between mortality due to lung cancer and total years of exposure at work (P = 0.003), and between mortality due to cardiovascular diseases and total years of exposure in early life (P = 0.029) (table).

Discussion

In Chinese women who had never smoked, exposure to tobacco smoke from husbands was associated with increased all cause mortality, especially increased mortality due to cardiovascular disease. Exposure to environmental tobacco smoke at work was associated with increased mortality due to cancer, especially lung cancer.

Relation to other studies

Studies published in the past two decades have indicated that environmental tobacco smoke is associated with lung cancer and coronary heart disease. Recent studies reported an association with all cause mortality: a 15% increase in people aged 45-74 in New Zealand and a 34% increase in people aged 60 or more in Hong Kong.[4 5]

Most previous epidemiological studies on the relation between environmental tobacco smoke and cardiovascular disease have focused on coronary heart disease, a leading cause of death in Western countries. Only a few studies have examined the relation of environmental tobacco smoke to stroke, a leading cause of death and disability in China.[7 8] We found a positive association of stroke with exposure via husbands who smoke, especially current exposure. This finding confirmed our previous report, which was based on cross sectional survey data.[7] Exposure to environmental tobacco smoke was also associated with a moderately increased risk of all cause mortality and mortality due to lung cancer and cardiovascular diseases, which is generally consistent with most previous studies.

Strengths and limitations

The strengths of our study include a high participation rate (92.7%) at baseline recruitment, so that our data are representative of the general population; a large sample size with a low proportion of current smokers, divorced women, and unmarried women; and a high proportion of environmental exposure to tobacco smoke (83.1%). Other strengths are complete assessment of exposure to environmental tobacco smoke from husbands, at work, and in early life from family members; extensive baseline and follow-up data on smoking and potential confounders; and appropriate use of the Cox regression model with complete control for the effect of age and adjustment of important potential confounders. The inconsistency of previous findings has resulted, at least in part, from methodological problems. These include incomplete and inaccurate measurement of environmental tobacco smoke[1 9] and insufficient control of confounding due to a lack of information about confounders, such as dietary factors,[1] and the use of inappropriate regression models.[10 11]

Our results were based on data obtained after only a short follow-up time (average 5.7 years), so that the power of our study was not optimal, especially for the evaluation of cause specific mortality. Although our results generally show that environmental tobacco smoke has adverse effects on all cause mortality and mortality due to cancer and cardiovascular disease, associations with different sources of exposure varied. Exposure at different times of life may contribute to the risk of different diseases. Environmental tobacco smoke could also be a surrogate measure of other risk factors that were not adjusted for, such as carcinogens at work. As with previous studies, errors of measurement and in assessing environmental tobacco smoke may have occurred, especially for exposure at work, which is ubiquitous, and for exposure in early life, where long term recall might not be accurate. We tried to reduce potential recall bias by assessing exposure at the beginning of our study.

What is already known on this topic

The excess risk of coronary heart disease and lung cancer from exposure to environmental tobacco smoke is about 15-35%

Exposure to environmental tobacco smoke may also increase mortality, the risk of other cardiovascular diseases such as stroke, and other cancers, though evidence is scarce

What this study adds

Exposure to tobacco smoke from husbands was associated with increased all cause mortality and mortality due to cardiovascular disease

Exposure to environmental tobacco smoke at work was associated with increased mortality due to cancer, especially lung cancer

Conclusions

Environmental tobacco smoke was related to a moderately increased risk of all cause mortality and mortality due to lung cancer and cardiovascular disease. Given the high prevalence of exposure to environmental tobacco smoke (83.1%) in this population, a moderate association with a hazard ratio of 1.15 would yield an attributable risk in the population of 11.1%. The impact of environmental tobacco smoke on all cause mortality could be substantial.

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When going into urinary retention, call a … vet?

An 80 year old man, well known to the local urology department, called his local veterinary practice one Saturday afternoon. From previous painful experience, he knew he was going into urinary retention. He also knew that a hospital admission would mean that no one would be available to look after his Irish wolfhound. The veterinary practice had promised to look after the dog in such circumstances, but by the time the pair reached the surgery the elderly man was in acute urinary retention.

The on-duty vet called the out-of-hours medical services but was directed to the district nurse, who was unavailable for three hours. The round trip for the ambulance from the nearest emergency department would have been two hours. The gentleman was rapidly becoming more distressed so the vet called the senior partner, who lived near the surgery.

This experienced vet, familiar with the males of most species, from tom cats to stallions, arrived to find her client in extremis. En route, she had already contacted a nursing friend to ask what size catheter was appropriate for an 80 year old human male urethra. She prepared a "pencil sized" Foley catheter (which she would normally have used on a Labrador bitch) and asked the man for his preferred catheterisation position. He lay supine on the operating table in the small animal theatre, and the catheterisation was performed uneventfully. The patient was overjoyed at his relief.

After the hour long journey to hospital in the vet's truck, accompanied by two nurses, the patient was admitted to the urology ward. There were no postoperative complications, and the wolfhound enjoyed his brief holiday. Since the veterinary practice sent an itemised bill for their out-of-hours service to the local medical practice, subsequently passed on to the area ambulance service, there has been a change in paramedic training to incorporate urethral catheterisation. The (somewhat tongue-in-cheek) bill was settled amicably by the regional head of the ambulance service, with the replacement of the used Foley catheter.

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We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. Please submit the article on http://submit.bmj.com Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.