Passive smoking exposure and risk of COPD among adults in China: the Guangzhou Biobank Cohort Study

P Yin, C Q Jiang, K K Cheng, T H Lam, K H Lam, M R Miller, W S Zhang, G N Thomas, P Adab

Summary

Background **Chronic obstructive pulmonary disease (COPD) is a leading cause of mortality in China, where the population is also exposed to high levels of passive smoking, yet little information exists on the effects of such exposure on COPD. We examined the relation between passive smoking and COPD and respiratory symptoms in an adult Chinese population.**

Methods **We used baseline data from the Guangzhou Biobank Cohort Study. Of 20 430 men and women over the age of 50 recruited in 2003–06, 15 379 never smokers (6497 with valid spirometry) were included in this cross-sectional analysis. We measured passive smoking exposure at home and work by two self-reported measures (density and duration of exposure). Diagnosis of COPD was based on spirometry and defined according to the GOLD guidelines.**

Findings **There was an association between risk of COPD and self-reported exposure to passive smoking at home and work (adjusted odds ratio 1·48, 95% CI 1·18–1·85 for high level exposure; equivalent to 40 h a week for more than 5 years). There were significant associations between reported respiratory symptoms and increasing passive smoking exposure (1·16, 1·07–1·25 for any symptom).**

Interpretation **Exposure to passive smoking is associated with an increased prevalence of COPD and respiratory symptoms. If this association is causal, we estimate that 1·9 million excess deaths from COPD among never smokers could be attributable to passive smoking in the current population in China. Our findings provide strong evidence for urgent measures against passive smoking in China.**

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Department of Public Health and Epidemiology, University of Birmingham, UK (P Yin MSc, Prof K K Cheng PhD, K H Lam MPhil, P Adab MD)**; Guangzhou Number 12 People's Hospital, Guangzhou, China** (Prof C Q Jiang MD, W S Zhang PhD)**; Guangzhou Medical College, Guangzhou, China** (Prof C Q Jiang)**; School of Public Health, Department of Community Medicine, University of Hong Kong, Hong Kong, SAR China** (Prof T H Lam MD, G N Thomas PhD)**; and University Hospital Birmingham NHS Foundation Trust, Birmingham, UK** (M R Miller MD)

Correspondence to:
Prof K K Cheng, Department of Public Health and Epidemiology, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK
k.k.cheng@bham.ac.uk

Introduction

Chronic obstructive pulmonary disease (COPD) is a leading cause of mortality and morbidity worldwide, and is estimated to be the third most common cause of death by 2020.[1] Active smoking is well established as the predominant risk factor. However, less than a quarter of all smokers develop COPD and more than 15% of COPD occurs in never smokers, suggesting that other factors also play a part.[2]

China has one of the largest populations of tobacco consumers worldwide, with smoking rates as high as 70% among men during the 1990s.[3] Also, the proportion of never smokers who develop COPD in China is much higher than that in most other countries.[4] Compared with women in Europe, the USA, and Canada, Chinese women have a higher risk of respiratory disease, although their smoking rates are lower.[5] The proportion of COPD deaths in never smokers is as high as 81% in rural Chinese women.[6]

Passive smoking has been implicated as a potential risk factor. There is strong evidence of an association between passive smoking exposure in adulthood and respiratory symptoms.[7–10] The evidence on effects of passive smoking exposure and lung function is less consistent.[8] A comprehensive review of 23 published studies[8] available in Medline up to 2000, and a further eight studies published subsequently,[11–18] show mixed results. Some of these studies included smokers in the analysis[14,15,19] and only one was from China.[20] Furthermore, most previous studies included small samples.

The prevalence of passive smoking exposure is high in China[4] and there is not much legislation to restrict exposure in the workplace and public areas. However, there is little information on the effects of passive smoking on lung function in Chinese populations. In this study, we analysed baseline data from 15 379 never smokers among 20 430 participants in the Guangzhou Biobank Cohort Study to examine the relation between passive smoking, self-reported respiratory symptoms, and COPD, based on spirometry.

Methods

Participants

The Guangzhou Biobank Cohort Study is a continuing prospective study among older individuals (50 years and older) that aims to examine environmental and genetic determinants of several chronic diseases in a southern Chinese population. Details of recruitment and description of phase I participants are reported elsewhere.[21] Briefly, a community social and welfare association was chosen as a sampling frame. This association, with around 100 000 members, has branches throughout Guangzhou and its membership is open to anyone over the age of 50 years for a nominal fee. People were randomly recruited from the association's membership list. We only included those who were ambulatory and not receiving treatment for life threatening diseases, such as cancer. However, a cultural unwillingness of Chinese men to give blood due to the belief of an associated loss of so-called life energy has

resulted in fewer men than women recruits. The participants received a full medical check-up including lung function assessment. A detailed questionnaire was given by interview to assess a range of measures including smoking, passive smoking and occupational exposures, and personal disease history. This report is based on data from 20 430 participants, including 10 413 from phase I (September, 2003, to November, 2004) and 10 017 from phase II of recruitment (April, 2005, to May, 2006).

The study received ethics approval from the Medical Ethics Committee of the Guangzhou Medical Association. All participants gave written, informed consent before participating in the study.

Procedures

The questionnaire included a detailed assessment of passive smoking exposure both at home and at work. Two separate self-reported measures were used. The first measure was based on density, and defined as the presence of none, one, or more smokers living in the same household as the participant recorded separately for childhood and adulthood exposure, or co-workers smoking nearby while indoors. The second measure was based on duration of exposure in adulthood (since age 18 years). Participants were asked to report the number of hours per week of usual exposure to passive smoking at home and at work, and the number of years of such exposure for each. We calculated total hours of adulthood exposure at home and work separately and combined, based on 52 weeks per year. The level of exposure was categorised into low (less than 2 years of 40 h a week), medium (2–5 years of 40 h a week), and high (more than 5 years of 40 h a week). We used 40 h as the typical number of working hours in a week, but repeated the analyses using different cutoffs (30 h or 50 h per week) for level of exposure, to assess the robustness of our findings.

The Medical Research Council respiratory questionnaire was used to assess respiratory symptoms and the following definitions were used: cough or phlegm (usually having cough or phlegm first thing in the morning or either during the day or at night); shortness of breath (troubled by shortness of breath when hurrying on level ground or walking up a slight hill); or any symptoms (having any one of the symptoms defined above).

Spirometry was done with a pneumotachograph (Chestgraph HI-701, Chest MI Inc, Tokyo, Japan) in phase I and a turbine flowmeter (Cosmed microQuark, Rome, Italy) in phase II. The test was done in a standing position without nose clips. At least three manoeuvres were done and the best measure of forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and FEV_1/FVC were recorded. Predicted values for lung function were derived using the equations of Ip and colleagues.[22] To classify tests for reliability and validity, a numerical quality-check algorithm was developed according to European Respiratory Society recommendations using criteria used in the BRONCUS trial.[23] The remaining results were assessed by visual inspection of flow-volume and volume-time curves by two authors (PY, KHL). In those patients with no reported physician-diagnosed asthma, we defined COPD as FEV_1/FVC of less than 0·70, according to GOLD guidelines,[24] but without the use of a bronchodilator. We repeated the analyses in men and women separately, on the basis of data from phase I and then II, and using the 5th percentile of the lower limit of normal as the basis for cutoff in defining COPD.

	N (%)
Sex	
Men	1777 (11·6)
Women	13 602 (88·4)
Age (years)	
50–59	6717 (43·7)
60–69	6607 (42·9)
≥70	2055 (13·4)
Education	
<Primary school	1768 (11·5)
Primary school	5405 (35·2)
Middle school	6922 (45·0)
>Middle school	1277 (8·3)
Occupational dust exposure	
Yes	6147 (40·3)
No	9113 (59·7)
Indoor air pollution exposure	
Yes	5134 (33·4)
No	10 245 (66·6)
Passive smoking exposure	
Childhood home exposure	
Yes	8283 (54·1)
None	7029 (45·9)
Adulthood home exposure	
Yes	8846 (57·7)
None	6487 (42·3)
Work exposure	
Yes	6848 (44·7)
None	8465 (55·3)
COPD status*	
No COPD	6068 (93·4)
COPD GOLD I	140 (2·2)
COPD GOLD II	222 (3·4)
COPD GOLD III	55 (0·8)
COPD GOLD IV	12 (0·2)

*Based on 6497 participants with valid spirometry data and no self-reported asthma. GOLD I=FEV_1/FVC <0·70 and FEV_1 ≥80% predicted. GOLD II=FEV_1/FVC <0·70 and 50%≤FEV_1<80% predicted. GOLD III=FEV_1/FVC<0·70 and 30% ≤FEV_1 <50% predicted. GOLD IV=FEV_1/FVC <0·70 and FEV_1 <30% predicted.

Table 1: **Baseline characteristics of never smoking participants (N=15 379)**

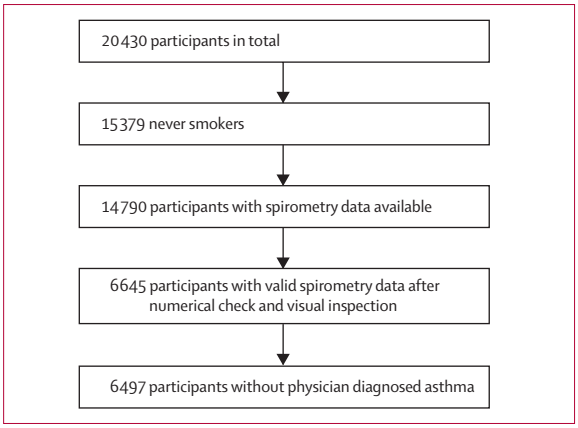

Figure: **Flow diagram of the study population**

A detailed smoking history was obtained and used to classify individuals as never smokers and ever smokers, if they had ever smoked each day. We measured urinary cotinine levels in the phase I participants, by gas chromatograph mass spectrometry, to validate smoking status. We also obtained a detailed occupational history, including exposure to dust and to indoor pollutants (household biomass fuels). Reported educational level was used as proxy for socioeconomic status. Interviewers who obtained data for passive smoking and other exposures, symptoms, and medical history were blinded to the spirometry findings, as were the participants at the time of interview.

Statistical analysis

For all analyses of the relation between passive smoking exposure and respiratory symptoms or COPD, we used only never smokers. Logistic regression models were done to assess the association between passive smoking exposure (measured as density and duration of exposure at home or work and density of exposure during childhood) and respiratory symptoms and COPD. The models were adjusted for age, sex, educational level, and exposure to occupational dust and indoor air pollutants. Odds ratios and 95% CI were computed. The models assessing the relation between passive smoking exposure and COPD included participants with valid spirometry (n=6497). The models assessing the relation between passive smoking exposure and respiratory symptoms included all never smokers (n=15 379). We calculated a p value for trend to assess any dose-response relation for each measurement of passive smoking exposure. We tested for interaction between passive smoking exposure and sex in relation to COPD prevalence.

Role of the funding source

The funding source had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

The baseline characteristics of the participants are shown in table 1. The mean age was 61·7 years (range 50–93). There were only 18 people (0·3%) who said they had never smoked who had urinary cotinine levels above 50 ng/mL. Of the 20 430 participants, 15 379 (75·3%) were never smokers. Spirometry data were available for 14 790 (96·1%). Application of the quality algorithm for spirometry measures and visual inspection resulted in rejection of data from 8145 participants. Of the remainder, 148 (2·2%) reported physician-diagnosed asthma and were excluded from analysis. Therefore, 6497 never smokers with spirometry data were available for use in the analysis for this report

	N (%) valid	N (%) invalid
Sex		
Men	688 (10·4)	1016 (12·4)
Women	5957 (89·6)	7161 (87·6)
Age (years)		
50–59	2961 (44·6)	3456 (42·3)
60–69	2803 (42·2)	3587 (43·8)
≥70	881 (13·2)	1134 (13·9)
Education		
<Primary school	735 (11·1)	963 (11·8)
Primary school	2309 (34·8)	2909 (35·6)
Middle school	3053 (46·0)	3620 (44·3)
>Middle school	545 (8·1)	681 (8·3)
Occupational dust exposure		
Yes	2696 (40·9)	3221 (39·7)
No	3899 (59·1)	4891 (60·3)
Indoor air pollution exposure		
Yes	2068 (31·1)	2852 (34·9)
No	4577 (68·9)	5325 (65·1)
Passive smoking exposure		
Childhood home exposure		
Yes	3657 (55·2)	4339 (53·3)
None	2963 (44·8)	3798 (46·7)
Adulthood home exposure		
<2 years of 40 h per week	4498 (67·7)	5642 (69·0)
2–5 years of 40 h per week	1177 (17·7)	1321 (16·2)
>5 years of 40 h per week	970 (14·6)	1214 (14·8)
Work exposure		
<2 years of 40 h per week	4896 (73·7)	6029 (73·7)
2–5 years of 40 h per week	842 (12·7)	1026 (12·6)
>5 years of 40 h per week	907 (13·6)	1122 (13·7)
Respiratory symptoms		
Cough	986 (11·3)	1358 (12·5)
Phlegm	1320 (15·4)	1694 (15·7)
Shortness of breath	3035 (35·1)	3794 (35·1)

Table 2: **Comparison of baseline data between participants with valid and invalid spirometry among never smokers**

(figure). Participants who had never smoked and whose spirometry data were included were similar to those excluded in terms of age, sex, educational level, occupational exposure, reporting of respiratory symptoms, and passive smoking exposures (table 2). The prevalence of COPD defined by spirometry was higher among smokers (n=342, 16·7%) compared with never smokers (n=429, 6·6%; odds ratio 2·85, 95% CI 2·44–3·31), and in men (n=354, 15·1%) compared with women (n=417, 6·7%; 2·48; 2·13–2·88).

Among never smokers, 98 (5·5%) men and 1667 (12·3%) women had never worked indoors. Among men and women combined, 13·7% reported high levels (more than 5 years of 40 h a week) of exposure to passive smoking at work and 14·5% at home. There were significant but weak associations between exposure to dust and indoor pollutants and risk of COPD. We therefore adjusted for these factors in subsequent analyses.

	N (%) without COPD	N (%) with COPD	Crude odds ratio (95% CI)	*Adjusted odds ratio (95% CI)
Childhood home exposure				
Number of smokers living in the same household				
0	2680 (92·3)	225 (7·7)	1	1
1	2534 (94·2)	157 (5·8)	0·74 (0·60–0·91)	0·89 (0·72–1·10)
≥2	829 (94·6)	47 (5·4)	0·68 (0·49–0·93)	0·81 (0·58–1·12)
p			0·002	0·14
Adulthood home exposure				
Number of smokers living in the same household				
0	2567 (92·9)	195 (7·1)	1	1
1	3011 (93·7)	201 (6·3)	0·88 (0·72–1·08)	0·96 (0·77–1·20)
≥2	490 (93·7)	33 (6·3)	0·89 (0·61–1·30)	0·92 (0·62–1·36)
p			0·26	0·63
Adulthood hours of exposure at home				
<2 years of 40 h per week	4129 (93·8)	273 (6·2)	1	1
2–5 years of 40 h per week	1082 (93·7)	73 (6·3)	1·02 (0·78–1·33)	1·11 (0·84–1·47)
>5 years of 40 h per week	857 (91·2)	83 (8·8)	1·47 (1·13–1·89)	1·60 (1·23–2·10)
Work exposure				
Number of smokers exposed to indoors at work				
0	3328 (93·3)	240 (6·7)	1	1
1	251 (94·4)	15 (5·6)	0·83 (0·48–1·42)	0·88 (0·51–1·52)
≥2	2465 (93·5)	172 (6·5)	0·97 (0·79–1·19)	0·97 (0·78–1·20)
p			0·73	0·76
Hours of exposure at work				
<2 years of 40 h per week	4501 (94·0)	286 (6·0)	1	1
2–5 years of 40 h per week	757 (92·1)	65 (7·9)	1·35 (1·02–1·79)	1·35 (1·01–1·80)
>5 years of 40 h per week	810 (91·2)	78 (8·8)	1·52 (1·17–1·97)	1·50 (1·14–1·97)
p			0·001	0·002
Total hours of adulthood home and work exposure				
<2 years of 40 h per week	2999 (94·0)	191 (6·0)	1	1
2–5 years of 40 h per week	1409 (94·5)	82 (5·5)	0·91 (0·70–1·19)	0·95 (0·72–1·24)
>5 years of 40 h per week	1660 (91·4)	156 (8·6)	1·48 (1·18–1·84)	1·48 (1·18–1·85)
p			0·001	0·001

*Adjusted for age, sex, educational level, occupational dust exposure, and indoor air pollution.

Table 3: **Relation between self-reported passive smoking exposure (using different measures) and COPD in never smokers**

The relation between the categories of passive smoking exposure and COPD is shown in table 3. There was no significant relation between risk of COPD and passive smoking exposure assessed by density of exposure during childhood or adulthood. However, using the duration of exposure, there was a significantly increased risk with increasing levels of adulthood exposure both at home and at work.

After adjustment for potential confounding factors, COPD prevalence among never smokers was significantly greater with high-level exposure to passive smoking at home (OR 1·60, 95% CI 1·23–2·10) and at work (1·50, 1·14–1·97) than those who had low-level exposure. When home and work exposures were combined, there was also a significantly increased risk of COPD with high exposure (1·48, 1·18–1·85 for longest duration of total adulthood exposure).

All measurements of passive smoking exposure, including childhood exposure, were significantly associated with reporting of any respiratory symptom (table 4). Adult passive smoking exposure was significantly associated with shortness of breath. Work, but not home, exposure was found to be associated with cough. No significant associations were seen between any exposure and phlegm.

We recorded no significant interaction between passive smoking exposure and sex (data not shown). We repeated the analyses on participants from phase I and II separately, since different spirometers were used in each phase. The direction of results for both phases was the same as the combined analysis, although the results were generally less significant because of smaller numbers of participants (and the association between duration of work exposure and COPD was not significant in phase I participants). When the lower limit of normal was used to define COPD, the proportion of those classified as having COPD reduced, but the direction and magnitude of results remained similar. When different cutoffs were used for categorising levels of passive smoking exposure, the direction of results remained the same. The magnitude of effect differed according to whether fewer or more people were included in the high-exposure category. Repeating the analyses on symptoms and passive smoking exposure separately for those with valid and invalid spirometry results showed that the direction of results was the same for both, and the magnitude of effect almost identical (results not shown).

Discussion

In this study, older adults who were exposed to passive smoking at work and at home, particularly higher level exposure, were associated with higher prevalence of COPD than those not exposed. Passive smoking exposure was also associated with respiratory symptoms. These results were particularly seen when exposure assessment

	Cough		Shortness of breath		Any symptoms	
	N (%)	Odds ratio	N (%)	Odds ratio	N (%)	Odds ratio
Childhood home exposure						
Number of smokers living in the same household						
0	695 (10·0)	1	2373 (34·4)	1	2981 (43·3)	1
1	636 (10·1)	1·08 (0·96–1·21)	2180 (34·8)	1·01 (0·94–1·09)	2796 (44·8)	1·07 (1·00–1·15)
≥2	191 (10·1)	1·06 (0·90–1·26)	695 (36·9)	1·10 (0·99–1·23)	879 (46·9)	1·16 (1·05–1·29)
P		0·27		0·13		0·003
Adulthood home exposure						
Number of smokers living in the same household						
0	654 (10·1)	1	2078 (32·3)	1	2718 (42·3)	1
1	737 (9·9)	1·02 (0·91–1·15)	2676 (36·2)	1·10 (1·02–1·19)	3348 (45·4)	1·09 (1·02–1·17)
≥2	138 (10·8)	1·06 (0·87–1·30)	511 (40·4)	1·31 (1·16–1·49)	615 (48·7)	1·24 (1·09–1·40)
P		0·57		<0·0001		<0·0001
Duration of exposure at home						
<2 years of 40 h per week	1047 (10·1)	1	3424 (33·2)	1	4434 (43·1)	1
2–5 years of 40 h per week	225 (8·8)	0·88 (0·76–1·03)	979 (38·4)	1·19 (1·08–1·30)	1188 (46·8)	1·13 (1·03–1·24)
>5 years of 40 h per week	257 (11·4)	1·18 (1·02–1·37)	862 (38·6)	1·20 (1·09–1·32)	1059 (47·6)	1·17 (1·07–1·29)
P		0·13		<0·0001		<0·0001
Work exposure						
Number of smokers exposed to at work						
0	792 (9·5)	1	2819 (33·9)	1	3576 (43·1)	1
1	62 (9·9)	1·11 (0·85–1·47)	217 (34·9)	1·10 (0·93–1·31)	277 (44·5)	1·10 (0·94–1·30)
≥2	669 (10·9)	1·19 (1·06–1·33)	2208 (36·2)	1·19 (1·10–1·27)	2801 (46·1)	1·17 (1·09–1·25)
P		0·003		<0·0001		<0·0001
Duration of exposure at work						
<2 years of 40 h per week	1087 (9·7)	1	3834 (34·5)	1	4870 (43·9)	1
2–5 years of 40 h per week	210 (10·9)	1·15 (0·98–1·35)	680 (35·7)	1·11 (1·0–1·23)	855 (45·0)	1·07 (0·97–1·18)
>5 years of 40 h per week	232 (11·0)	1·15 (0·98–1·34)	751 (36·0)	1·16 (1·05–1·28)	956 (46·1)	1·13 (1·03–1·25)
P		0·038		0·002		0·007
Total duration of adulthood home and work exposure						
<2 years of 40 h per week	736 (9·8)	1	2474 (33·1)	1	3204 (43·0)	1
2–5 years of 40 h per week	314 (9·4)	0·98 (0·85–1·13)	1195 (36·1)	1·13 (1·03–1·23)	1474 (44·5)	1·06 (0·97–1·15)
>5 years of 40 h per week	479 (11·0)	1·15 (1·01–1·30)	1596 (37·0)	1·19 (1·10–1·29)	2003 (46·6)	1·16 (1·07–1·25)
P		0·04		<0·0001		<0·0001

*All odds ratios are adjusted for age, sex, educational level, occupational dust exposure, and indoor air pollution.

Table 4: **Relation between self-reported passive smoking exposure and respiratory symptoms in never smokers**

was based on duration of exposure rather than density (as measured by numbers of smokers they were exposed to).

Our study has several strengths. The study population included in the analysis, with almost 6500 individuals, represents one of the largest studies addressing the relation between objectively assessed COPD and passive smoking exposure among never smokers. We were able to examine this group separately without the major confounding factor of active smoking, and self-report of smoking status was verified by urinary cotinine measures. We also had several measures of self-reported passive smoking exposure. Repeating analyses with different definitions of COPD and various cutoffs for passive smoking exposure all yielded similar results, with a significant association between passive smoking exposure and COPD.

One limitation of our study is the cross-sectional nature. Temporal associations cannot be inferred, and those with impaired lung function might have altered their exposure pattern. The study population might not be representative of the population of Guangzhou, since our participants were drawn from an association concerned with health and welfare, although it was open to all older residents in the city for a low yearly fee. Our exclusion criteria also meant that the participants represent a relatively healthy population of survivors compared with the general population, and those with the most severe COPD did not participate. Bias arising from differential recall of passive smoking in participants with COPD or respiratory symptoms could also generate a spurious association. In this connection, one important advantage of our study is that it is based on a large cohort

study with many objectives. In fact, COPD was not explicitly mentioned to participants at any stage. Furthermore, during interviews, questions on smoking and passive smoking were asked early on, after which participants were asked a large number of questions concerning sociodemographic and other lifestyle factors before they came to the section on symptoms. Also, respiratory symptoms were only a subset in the symptomatology section. The risk was further reduced by blinding the participants and the interviewers to spirometry findings. Also, few participants had a physician diagnosis of COPD. Finally, a survey in China showed that only about a third of respondents believed that passive smoking is harmful to health.[25] The combination of these features makes recall bias an unlikely explanation for the observed association between passive smoking and COPD.

The overall quality of spirometry was fairly poor, resulting in a large proportion of tests having to be discarded, which indicates the difficulty in doing lung function tests in large-scale studies, where a vast number of measurements are taken. However, by using a comprehensive and stringent quality check, we included only those data which were valid and reliable. Furthermore, baseline characteristics of those with valid and invalid spirometry were similar. Also the relation between exposure and respiratory symptoms were the same in both groups. Thus exclusion of those with invalid spirometry is unlikely to have resulted in any bias.

Our results of increased risk of COPD with high levels of adulthood exposure to passive smoking concur with some previous studies,[8,12] including a study showing excess mortality from COPD among those with passive smoking exposure in Hong Kong.[26] However, previous studies have shown mixed results,[8,11,13,16,17] and most have examined lung function rather than COPD as the outcome. One study that examined the risk of physician-diagnosed COPD, verified by spirometry in a small sample, reported that only those with long duration of passive smoking exposure (42 years or more at home and 23 years or more at work) were at increased risk (odds ratio 1·68; 95% CI 1·19–2·38, and 1·60; 1·20–2·14, respectively).[27] Some studies have shown that the relation between passive smoking exposure and poorer lung function is confined to those with increased susceptibility (bronchial hyper-responsiveness or asthma).[14,16,18] We specifically excluded those patients with physician-diagnosed asthma from this analysis. We noted that only duration of exposure, but not the number of smokers around the individual, was associated with COPD. This finding might mean that duration of exposure is a more accurate indicator of true total exposure.

Our finding of a dose-response relation between passive smoking exposure at work and home and any respiratory symptoms is consistent with several other studies,[8,10] although we did not find any association with phlegm. The magnitude of excess risk is generally lower than in previously reported studies, being between 13 (for any symptom in relation to work exposure) and 31% (for breathlessness in relation to home exposure).

Although there was a decrease in odds ratios for COPD with childhood passive smoking exposure in the unadjusted analysis, this effect disappeared after adjustment for age and other factors. The lack of association between childhood passive smoking exposure and COPD is in keeping with reports from a longitudinal study, when the analysis was confined to never smokers.[28] However, another large cross-sectional study which included smokers showed some association between parental smoking in childhood and respiratory symptoms and impaired lung function in adulthood.[29]

The prevalence of COPD among never smokers, particularly women, in this population is high, in keeping with other studies from China.[4,6] The prevalence of physician-diagnosed asthma, although lower than in most European and North American populations, is also in keeping with previous Chinese studies.[30]

In summary, passive smoking exposure in adulthood both at home and at work is related to COPD and respiratory symptoms in this southern Chinese population. However, the magnitude of increased risk is fairly small and the high prevalence of disease among never smokers in this population cannot be wholly explained by this exposure. Therefore other risk factors still need to be explored and identified.

Over 60% of adults in China are never smokers.[31] This population is exposed to high levels of passive smoking, since there is little restriction on smoking in indoor places. In our study population, more than half of never smokers reported exposure to passive smoking in their workplace and at home, with 28% reporting high levels of total adult exposure (more than 5 years with 40 h exposure per week). Of all deaths in China, around 11·6% among never smokers are attributable to COPD.[6] If our risk estimates are correct, and assuming that current mortality and passive smoking exposure patterns continue, of the 240 million people aged over 50 years alive today in China, high exposure to passive smoking would result in about 1·9 million (95% CI 0·9–2·8 million) excess deaths from COPD among never smokers. This finding has serious implications for population health, health services, and the economy, and lends further support to strong measures to ban smoking in public places and workplaces, and to increase availability of smoking cessation services in this region.

Contributors

All authors contributed to the study design, development of study instruments, and took part in the interpretation of findings and contributed to the final manuscript. PY undertook data cleaning checking and coding, did the analysis for the study, and wrote the first draft. CQJ, KKC, and THL originally designed the idea of the cohort study, and have been responsible for obtaining funding. CQJ oversaw data collection and facilities for participant recruitment, and has contributed to the final manuscript. KKC contributed to the design of the study, the decisions on the strategy for analysis, paper writing, and the amendment of the final manuscript. MRM developed the quality

check algorithm, checked the spirometry data, and contributed to the final manuscript. GNT contributed to data checking, writing the methods section, and approving the final manuscript. THL contributed to amendment of the manuscript and suggestions for data analysis. WSZ and KHL have contributed to data cleaning and checking, and have contributed to the final manuscript. PA conceived the idea for this paper, designed the study, contributed to the strategy for analysis, supervised and checked the analysis, and wrote the final draft.

Conflict of interest statement

We declare that we have no conflict of interest.

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