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Childhood exposure to environmental tobacco smoke and chronic respiratory symptoms in nonsmoking adults: The Singapore Chinese Health Study

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Abstract

Introduction—Childhood exposure to environmental tobacco smoke has been extensively associated with childhood respiratory illness; fewer data address adult effects.

Methods—We examined childhood environmental tobacco smoke exposure in relation to chronic cough, phlegm and asthma diagnosis among never smokers from a cohort of Singaporeans of Chinese ethnicity, aged 45–74 at enrollment from 1993 to 1998. From 1999 to 2004, subjects were interviewed regarding environmental tobacco smoke exposure before and after age 18 and the presence and duration of current symptoms of chronic cough and phlegm production and asthma diagnosis.

Results—Among 35,000 never smokers, fewer had smoking mothers (19%) than fathers (48%). Although few subjects currently lived (20%) or worked (4%) with smokers, 65% reported living with a daily smoker before age 18 years. Living with a smoker before age 18 increased the odds of chronic dry cough (149 cases, OR = 2.1, 95% CI 1.4–3.3) and, to a lesser extent, phlegm, after adjustment for age, gender, dialect group and current and past exposure to smokers at home and at work after age 18. Associations strengthened with higher numbers of smokers in childhood. There was no association with asthma or chronic bronchitis. We found suggestive evidence of a stronger association among subjects with lower adult intake of fiber, which we previously found to be protective for respiratory symptoms.

Conclusions—In this large study of nonsmokers, living with a smoker in childhood was associated with chronic dry cough and phlegm in adulthood, independently of later exposures to environmental tobacco smoke.

Keywords

Tobacco smoke pollution; asthma; Signs and Symptoms; Respiratory; Bronchitis; chronic; Dietary Fiber

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INTRODUCTION

Childhood exposure to environmental tobacco smoke (ETS) has been consistently associated with childhood respiratory illness and symptoms, including bronchitis, pneumonia, cough, phlegm, breathlessness, wheeze, and asthma (reviewed in [1]). Several studies have suggested that ETS exposure in adulthood is associated with nonmalignant respiratory disease (reviewed in [2]); however, data on the relation between childhood exposure to ETS and nonmalignant respiratory illness in adults are somewhat inconsistent.[3] [4] [5] [6] [7] [8] [9] [10] [11] [12] Most of these studies deal with asthma alone.

Findings on childhood ETS exposure in relation to adult respiratory symptoms and illnesses are likely confounded by the strong association between active smoking and these outcomes. Exposure to tobacco smoke in early life may influence smoking behavior later in life.[6] Of the studies with data on childhood ETS exposure and adult respiratory illness, most have a large proportion of smokers. The study of nonsmokers offers the advantage of virtually eliminating residual confounding by smoking. The usefulness of restricting the analysis to nonsmokers in the elimination of bias due to uncontrollable confounding by active smoking has been demonstrated for the study of leanness in relation to lung cancer.[13]

We examined whether exposure to ETS as a child increases the prevalence of adult chronic cough, phlegm and asthma in a population of 35,000 lifetime never smokers from the Singapore Chinese Health Study. We also considered whether the effects of childhood exposure to tobacco smoke in the home might be modified by intake of non-starch polysaccharides, a form of dietary fiber that is associated with reduced chronic respiratory symptoms in this cohort.[14]

METHODS

Study Population

The design of the Singapore Chinese Health Study has been previously described.[15] In brief, the cohort was drawn from the population of men and women of Chinese ethnicity, aged 45 to 74 years, who were permanent residents or citizens of Singapore and resided in government-built housing estates, which housed 86% of the population during the period of subject enrollment. The study was restricted to individuals belonging to the two major Chinese dialect groups in Singapore: Cantonese and Hokkien. Between April 1993 and December 1998, we enrolled 63,257 individuals (85% of those eligible). Baseline questionnaires were administered by trained interviewers in the subjects' homes and elicited information on demographics, active smoking, and medical history. The baseline interview included a 165-item quantitative food frequency questionnaire which was developed for and validated in this population[16], enabling estimation of intake of non-starch polysaccharides, a form of dietary fiber.[14]

Beginning in 1999, subjects were interviewed by telephone, regarding active smoking, history of ETS exposure before and after age 18, and the presence and duration of respiratory symptoms. The protocol was reviewed and approved by the Institutional Review Boards of the University of Southern California, the National University of Singapore, and the U.S. National Institute of Environmental Health Sciences.

As of December 31, 2004, there were 7,722 deaths in the overall cohort. The follow-up telephone interview was completed for 52,325 individuals. The average time between the baseline and follow-up interviews was 5.8 years. We restricted the analysis to the 35,000 never smokers defined by a "NO" response to the question at baseline about ever having smoked at least one cigarette a day for one year or longer and a further "NO" response to the question "Have you smoked at least 100 cigarettes in your lifetime?" on the follow-up interview. Smoking histories were not validated by biomarkers; however, studies comparing serum

cotinine data with self-reported tobacco use suggest that there is little misclassification of nonsmokers based on self-report in surveys of the general population. [17] [18]

We used American Thoracic Society questionnaire items (ATS-DLD-78-A) to assess history of asthma, cough, and phlegm production (see online supplement Appendix 1). [19] Because we were interested in the general question of whether early life exposure to smokers increased later risk of respiratory outcome, we wanted to compare these various outcomes to a common group of individuals who had no respiratory impairment. Therefore, we divided subjects into mutually exclusive categories based on asthma, cough and phlegm. Chronic was defined as occurring on most days for at least three months of the year, lasting more than two years in a row. We first classified subjects according to report of doctor-diagnosed asthma and then classified those without this report according to the presence of chronic cough and/or phlegm or if these symptoms did not meet the definition of chronic, into a combined recent symptoms category. The categories were doctor-diagnosed asthma (1504 cases), recent cough and/or phlegm (1320), chronic phlegm (700), chronic cough (185), chronic cough plus phlegm (chronic bronchitis) (149), and a referent group with no cough, phlegm, or asthma (31,142). The objective of this study was to examine the association between early life exposure to ETS and chronic respiratory symptoms in adults; therefore, analyses of the 1320 individuals reporting recent cough and/or phlegm are not included in the tables for space reasons, but can be found in the online supplement Appendix 2.

Statistical Analysis

We examined the odds of chronic respiratory symptoms in adults in relation to exposure to ETS in early life and adulthood. Odds ratios (ORs) and 95 percent confidence intervals (CIs) were calculated from unconditional logistic regression models (version 9.0; SAS Institute, Cary, NC). We represented childhood exposure, defined as birth to age 18, with a semi-quantitative variable representing the minimum number of smokers an individual lived with during childhood from the number of categories marked regarding who smoked in the household [father, mother, grandparent(s), sibling(s), and other person(s)] (see online supplement Appendix 1 for questionnaire items). Some of the categories could include more than one smoker; therefore, each marked category includes at least one smoker. We created a similar semi-quantitative variable for residential exposure after age 18, henceforth referred to as adult home exposure. Adult work exposure was modeled past or current versus none.

We adjusted all odds ratios presented for age, as a continuous variable, gender, and dialect group. The following covariates were assessed as potential confounders: education (completed ≤ grade 6, grade 7–10, grade 11 or higher), body mass index as a continuous variable (log transformed), occupation (10 categories), being born in Singapore versus elsewhere and dietary intake of non-starch polysaccharides (quartiles). None appreciably changed the associations so they were not included in the multivariate models.

RESULTS

Females outnumbered males in this population of lifetime non-smokers, as expected given the lower smoking rates among women (Table 1). Overall, 65% of subjects reported living with at least one daily smoker before age 18. Only 19% reported maternal smoking; 48% reported paternal smoking. Adult home exposure to ETS and work exposure were more common in the past than current (Table 1). These trends reflect prohibitive measures against smoking in public places and health education efforts established in the 1970s in Singapore.[20] [21] Chronic dry cough was reported by 0.5% of subjects and chronic phlegm without cough by 2%.

Subjects who lived with one or more daily smokers prior to age 18 had increased odds of chronic phlegm without cough (OR = 1.25, 95% CI 1.02–1.53) and chronic dry cough (OR =

2.14, 95% CI 1.39–3.29) (Table 2). We observed a significant trend of increasing odds ratios with higher number of smokers in the household in early life for both chronic phlegm (P, trend = 0.004) and chronic dry cough (P, trend = 0.005). We were unable to examine dose-response with duration of early life exposure because duration varied little; 91% of the subjects who reported exposure prior to age 18 indicated duration of 12 or more years, the top category. Early life ETS exposure was associated with chronic bronchitis when adjusting only for age, gender, and dialect (OR = 1.78, 95% CI 1.22–2.61); adjustment for exposure after age 18 virtually eliminated the association (OR = 1.18, 95% CI 0.76–1.82). Chronic phlegm production alone gave similar results to chronic bronchitis for early ETS. If these two categories are combined, the OR for living with a smoker before age 18 was 1.24 (95% CI 1.03–1.49). In the overall data, we found no appreciable relationship between early life ETS exposure and doctor-diagnosed asthma.

Chronic cough was equally strongly associated with early life exposure to maternal smoking (OR = 2.22, 95% CI 1.33 – 3.69) and with early life exposure to smoking by others (including father, grandparent, sibling, other) (OR = 2.11, 95% CI 1.36 – 3.28) (see online supplement, Appendix 3). Smoking by mothers alone was uncommon (4%) in this population.

Although, some of the categories become sparse, because of previous literature [10], we examined effects of parental smoking on adult respiratory health by gender (Table 3). ETS exposure in early life was similarly associated with increased risk of chronic dry cough in females and males although precision was lower in the smaller stratum of males. Living with at least one smoker in childhood raised the odds of doctor-diagnosed asthma among women (OR 1.17, 95% CI 1.01–1.36) but not in men (OR = 0.82, 95% CI 0.60–1.13; P value for difference in ORs = 0.05).

We found no appreciable difference in the association between early life exposure and chronic dry cough by age (</≥ median of 61 yrs) or dialect group (data not shown).

We also examined possible effect modification of the association between early ETS exposure and chronic cough or phlegm by intake of non-starch polysaccharides, a form of fiber related to reduced chronic respiratory symptoms in this cohort.[14] We examined only potential effect modification by fiber, because we previously found that fiber intake explained univariate associations with antioxidant micronutrients (such as vitamin C, vitamin E, and carotenoids) and food groups such as total fruit and soyfoods.[14] Although numbers become small on stratification, the association between early life ETS exposure and chronic dry cough or chronic phlegm was somewhat greater in subjects with a calorie-adjusted intake of fiber below the median of 7.5 grams/day for the entire Singapore Chinese Cohort than in subjects with higher intake (Table 4). There was a significant trend with increasing number of smokers in childhood in the lower fiber ($P < 0.01$), but not the higher fiber, category for both chronic phlegm (P, interaction = 0.27) and chronic cough (P, interaction = 0.05).

Adult ETS exposure at home was associated with various respiratory outcomes, independently of childhood exposure and adult work exposure. Adult past and current home exposure were positively associated with chronic bronchitis (Table 2). Doctor-diagnosed asthma was weakly related to exposure to 2 or more past smokers; a smaller association for currently living with 1 or more smokers might reflect the influence of an asthmatic on smoking behavior of household members. The similar association of past and current exposure with the outcomes could reflect resistance to ETS effect on the part of persons still living with smokers after many years. Chronic phlegm, but not chronic dry cough, was modestly associated with adult exposure to smokers in the home.

Exposure to ETS at work was associated with increased risk for all outcomes, after adjusting for adult home exposure and childhood exposure, with the weakest association being observed

for asthma (Table 2). We combined the small category of current work exposure with the larger past exposure category because odds ratios were in the same direction.

We explored the potential interaction between early life and adult ETS exposure and adult respiratory symptoms by examining odds ratios according to four categories of joint exposure: none, childhood only, adult only, and both childhood and adult. Although small numbers preclude precise estimation of the effects of exposure only in childhood, the associations with all outcomes were greater for exposure in both childhood and adulthood than for adult exposure alone (Table 5). Stronger associations observed in Table 5 compared to Table 2 most likely reflect the fact that subjects in the reference category for Table 5 had neither childhood nor adult ETS exposure.

We preferred mutually exclusive categories in order to compare each outcome group to the same reference category of individuals unaffected by any of the conditions. However, for comparison with studies that do not use mutually exclusive categories, we also compared each outcome group to all other subjects. Not surprising, the results were quite similar. For example, the odds ratio for living with a smoker before age 18 and chronic cough was 2.12 (95% CI 1.38–3.26) and for chronic phlegm was 1.24 (95% CI 1.01–1.52).

DISCUSSION

These data provide evidence that early life exposure to ETS has long-term effects on adult respiratory health. Living with a smoker in childhood was associated with chronic dry cough and, to a lesser degree, phlegm production in adult nonsmokers, independently of later life exposures to ETS at home and at work. We observed a dose-response relationship with increasing odds ratios for higher estimated number of smokers in the home in childhood. These associations suggest effects of postnatal exposure given the low rates of maternal smoking in this population. We also found some evidence that intake of fiber might modify the association between early life ETS and adult respiratory symptoms.

It is unclear how early life exposure to ETS contributes to adult chronic dry cough. However, exposure to tobacco smoke in childhood may lead to permanent structural changes in the lung. Elliot *et al* observed an increase in inner airway wall thickness of the larger airways in sudden infant death syndrome (SIDS) victims whose mothers smoked pre- and postnatally compared with unexposed SIDS infants.[22] In a larger subsequent study, significant inner wall thickening was seen in infants exposed only postnatally.[23] This finding is relevant to our study where relatively few subjects would have had prenatal exposure. A small increase in airway wall thickness may contribute to excessive airway narrowing in response to smooth muscle contraction.[24] Although studies in SIDS infants do not allow one to conclude whether the observed changes would have been permanent, lung function studies in adults provide suggestive evidence of long-lasting damage to airways from childhood exposure. [10] [11] [12]

Exposure to ETS during childhood may also enhance the excitability of the sensory nerves of the lung that are responsible for the cough reflex. Exposure of guinea pigs to ETS in the first five weeks of life (equivalent of human childhood) upregulates lung C-fiber central nervous system (CNS) reflex responses which could result in cough.[25] It is not known if these changes persist long-term. Alternatively, ETS exposure during childhood may influence adult respiratory symptoms via the well documented increased risk of acute lower respiratory illness. [1]

Early life exposure to ETS was weakly associated with adult chronic phlegm without cough. One would expect that cough would accompany phlegm as subjects were asked to exclude phlegm from the nose. Few other studies using similar questions have reported separate results

for phlegm, thus it is hard to know the worldwide prevalence of reports of phlegm without cough. However, some other cohorts have also reported a higher prevalence of phlegm than of cough. [26] [27] [28] [29] In our cohort about twice as many people reported phlegm only compared with cough or cough with phlegm. This might reflect practices in Chinese populations in which spitting of phlegm onto the street is a common problem.[30] Recognition of this habit resulted in the banning of public spitting in Singapore long ago. Ignoring phlegm from the nose in reporting may be difficult, especially in certain populations. Thus we present the chronic phlegm and chronic bronchitis outcomes separately. Results for early ETS were similar for the two outcomes although results for later ETS exposure differed somewhat. It is also possible that an increase in lung C-fiber CNS reflex responses to postnatal ETS exposure could also lead to mucus hypersecretion and thus, phlegm production.[25]

Data on early life exposure to ETS and adult respiratory symptoms or illness [3] [4] [5] [6] [7] [8] [9] [10] are not entirely consistent. In contrast with our study, most other studies examined only asthma. [3] [4] [5] [6] [8] [9] No association between childhood ETS exposure and asthma was observed in five studies [3] [5] [7] [8] [10]; positive associations were found in three others.[4] [6] [9] We found weak evidence for an association between early life ETS and asthma only in women. Of note, in the study of Larsson *et al.* [6], no association was found in subjects aged 40–69 years in the mid 1990s, a group of comparable birth cohort to our subjects. In the European Community Respiratory Health Survey (ECRHS) [10], parental smoking was associated with wheeze, with a composite variable of three or more asthma symptoms, and with chronic bronchitis, but not with current asthma. Unfortunately, we did not ask about wheeze. We did not observe an association with chronic bronchitis after adjusting for later life ETS exposure. Robbins *et al.* also found no association between childhood exposure and chronic bronchitis.[7]

The major limitation of this cross-sectional study is the potential for recall bias if individuals with respiratory symptoms are more likely to report ETS exposure. However, it seems reasonable that adults could remember if they lived with a smoker during childhood, especially after the age of six. Data from the Midspan family study shows that there is good agreement between parental reporting of maternal and paternal smoking and adult recall by offspring aged 30–59.[31] Further, if recall bias were playing a major role, we would expect to see associations between early life exposure to ETS and all outcomes, not just chronic dry cough and phlegm. In the ECRHS, differential recall bias would not explain the finding that parental smoking during childhood was associated with reduced lung function even in non-symptomatic subjects. [10] We did not ask about childhood exposure to ETS sources outside of the home as the accuracy of these data are less certain.

This study enhances the existing literature which is based mostly on Western populations. Our Chinese population also provides a large proportion of nonsmoking women. Restricting the analysis to nonsmokers virtually eliminates potential confounding by active smoking. This is also the largest study of adult respiratory effects of early exposure to ETS.

A strength of this study is the high quality dietary data that enabled us to examine possible confounding and effect modification by intake of non-starch polysaccharides, the major protective factor for respiratory symptoms in this cohort. [14] To our knowledge, this is the first study of childhood exposure to ETS to include examination of interaction by dietary factors. The dietary data were collected on the baseline questionnaire and thus were prospective with respect to the respiratory outcomes, eliminating the possibility of recall bias. We found suggestive evidence of a stronger association between chronic cough and phlegm and living with smokers in childhood among subjects with lower intake of non-starch polysaccharides, a form of dietary fiber. Fiber is a complex dietary mixture [32] [33] that may have beneficial effects on the lung by reducing blood glucose concentrations [34] [35] [36], reducing

inflammation [37] and enhancing antioxidant processes.[38] [39] Higher fiber intake may produce a state of lower inflammation and oxidative stress that may protect the lung against subsequent environmental insults which could trigger development of respiratory symptoms among those exposed to ETS in childhood. Our data suggest that poorer diet might accentuate deleterious effects of early life exposure to ETS. It is extremely difficult to obtain data on childhood diet from older adult subjects, and thus we are unable to directly test the possibility that adults with higher fiber intake also had higher fiber intake as children.

In conclusion, substantial evidence already exists that exposure to ETS in early life has adverse effects on childhood respiratory health. Our data from the Singapore Chinese Health Study add to a growing body of evidence that early life exposure may also adversely affect adult respiratory health. In this largest study to date, exposure, that was largely postnatal, was associated with chronic respiratory symptoms in adults. These results have major health implications given the widespread exposure of youth to ETS. In addition, other early life exposures, such as outdoor air pollution, may act through similar pathways resulting in long-term consequences on respiratory health. These results emphasize the importance of reducing the exposure of children to tobacco smoke and other environmental pollutants.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

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Table 1

Characteristics of the Singapore Chinese Health Study, never smokers

Characteristic	No Cough, Phlegm or Asthma N = 31,142	Recent Cough and/or Phlegm N = 1320	Asthma Diagnosis N = 1504	Chronic Bronchitis * N = 149	Chronic Phlegm without Cough N = 700	Chronic Dry Cough N = 185	Total Subjects † N = 35,000
% of Total †	89.0	3.8	4.3	0.4	2.0	0.5	100
% female	77.3	72.4	77.0	65.1	68.7	68.1	76.8
Age (years)							
Mean (SD)	60.7 (7.8)	61.5 (8.1)	60.2 (7.8)	62.1 (8.0)	60.1 (7.9)	60.7 (7.4)	60.7 (7.8)
Hokkien or Cantonese dialect							
% Hokkien	49.8	37.3	52.5	55.7	44.3	59.5	49.4
Childhood ETS exposure (%)							
None	35.4§	29.5	31.4§	24.2	26.1	19.5	34.7§
1 Smoker‡	34.0	35.8	34.2	39.6	35.1	40.5	34.2
2 Smokers	21.4	24.1	24.1	22.1	25.9	27.6	21.7
3+ Smokers	9.3	10.6	10.2	14.1	12.9	12.4	9.5
Adult ETS Exposure (%)							
Home							
None	27.7	23.1	24.5§	14.1§	21.1	20.5§	27.2
Past							
1 Smoker	30.7	32.0	28.3	35.6	34.7	35.7	30.8
2+ Smokers	21.4	23.6	24.9	26.2	23.6	27.6	21.7
Current							
1 Smoker	17.7	18.6	19.9	19.5	17.0	14.1	17.8
2+ Smokers	2.5	2.7	2.5	4.7	3.6	2.2	2.5
Work							
None	78.1	72.2	73.1	66.4§	66.4§	67.0	77.3
Past	17.7	23.1	21.4	29.5	25.4	26.5	18.3
Current	4.2	4.7	5.5	4.0	8.1	6.5	4.4

* Chronic bronchitis was defined as combined chronic cough and phlegm.

† Total includes all 35,000 nonsmoking subjects.

‡ Number of smokers for childhood and adult ETS exposure is a semi-quantitative variable representing the minimum number of smokers.

§ Some percentages do not add up to 100, because of rounding.

Table 2

Respiratory symptoms in relation to exposure to environmental tobacco smoke (ETS)

	No Cough, Phlegm or Asthma	Asthma Diagnosis		Chronic Bronchitis*		Chronic Phlegm without Cough		Chronic Dry Cough
	N	N	OR (95% CI)†	N	OR (95% CI)	N	OR (95% CI)	N	OR (95% CI)
Childhood home ETS exposure ‡									
None	11011	473	1.00	36	1.00	183	1.00	36	1.00
1 Smoker§	10599	515	1.06 (0.92–1.22)	59	1.21 (0.76–1.92)	246	1.18 (0.95–1.47)	75	2.07 (1.32–3.24)
2 Smokers	6649	362	1.13 (0.96–1.32)	33	1.01 (0.59–1.73)	181	1.33 (1.04–1.69)	51	2.24 (1.36–3.70)
3 or more Smokers	2883	154	1.06 (0.86–1.31)	21	1.42 (0.77–2.63)	90	1.48 (1.10–1.99)	23	2.36 (1.29–4.32)
P, trend			0.317		0.491		0.004		0.005
1 or more Smokers	20131	1031	1.08 (0.94–1.23)	113	1.18 (0.76–1.82)	517	1.25 (1.02–1.53)	149	2.14 (1.39–3.29)
Adult home ETS exposure ¶									
None	8635	369	1.00	21	1.00	148	1.00	38	1.00
1 Past Smoker	9558	425	0.98 (0.83–1.15)	53	2.13 (1.22–3.72)	243	1.30 (1.03–1.64)	66	1.04 (0.66–1.62)
2 or more Past Smokers	6655	374	1.22 (1.02–1.45)	39	2.37 (1.27–4.43)	165	1.26 (0.96–1.66)	51	1.06 (0.63–1.76)
1 or more Current Smokers	6294	336	1.16 (0.98–1.38)	36	2.69 (1.46–4.94)	144	1.33 (1.02–1.73)	30	0.75 (0.44–1.30)
P, trend			0.017		0.005		0.099		0.285
Past or Current	22507	1135	1.07 (0.93–1.24)	128	2.30 (1.36–3.91)	552	1.30 (1.04–1.62)	147	0.97 (0.64–1.49)
Work ETS exposure **									
None	24325	1099	1.00	99	1.00	465	1.00	124	1.00
Past or Current	6817	405	1.30 (1.15–1.47)	50	1.51 (1.05–2.17)	235	1.55 (1.31–1.83)	61	1.53 (1.10–2.11)

*Chronic bronchitis was defined as combined chronic cough and phlegm.

†All odds ratios (OR) and 95% confidence intervals (CI) are adjusted for age, gender, and dialect group plus ETS variables as indicated in subsequent footnotes.

‡ORs adjusted for adult home (no, 1 past, 2+ past, 1+ current smokers) and work (no, past or current) exposure.

§Number of smokers for childhood and adult ETS exposure is a semi-quantitative variable representing the minimum number of smokers.

¶ORs adjusted for childhood (no, 1, 2, 3+ smokers) and work (no, past or current) exposure.

**ORs adjusted for childhood (no, 1, 2, 3+ smokers) and adult home (no, 1 past, 2+ past, 1+ current smokers) exposure.

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Table 3

Respiratory symptoms in relation to environmental tobacco smoke exposure among men and women.

	No Cough, Phlegm, or Asthma	Asthma Diagnosis		Chronic Bronchitis*		Chronic Phlegm without Cough		Chronic Dry Cough	
	N	N	OR (95% CI)†	N	OR (95% CI)	N	OR (95% CI)	N	OR (95% CI)
Males (N = 7759)									
Childhood home ETS exposure ‡									
None	2292	112	1.00	11	1.00	50	1.00	11	1.00
1 or more Smokers §	4791	234	0.82 (0.60 – 1.13)	41	0.93 (0.39 – 2.23)	169	1.40 (0.92 – 2.11)	48	2.24 (1.00 – 5.03)
Adult home ETS exposure ¶									
None	2862	129	1.00	11	1.00	70	1.00	19	1.00
Past or Current	4221	217	1.27 (0.94 – 1.72)	41	2.52 (1.06 – 6.00)	149	1.16 (0.80 – 1.68)	40	0.87 (0.44 – 1.71)
Work ETS exposure **									
None	4229	195	1.00	25	1.00	112	1.00	32	1.00
Past or Current	2854	151	1.15 (0.92 – 1.43)	27	1.46 (0.84 – 2.53)	107	1.35 (1.03 – 1.77)	27	1.16 (0.69 – 1.94)
Females (N = 25921)									
Childhood home ETS exposure‡									
None	8719	361	1.00	25	1.00	133	1.00	25	1.00
1 or more Smokers §	15340	797	1.17 (1.01 – 1.36)	72	1.27 (0.77 – 2.10)	348	1.21 (0.96 – 1.52)	101	2.16 (1.31 – 3.55)
Adult home ETS exposure¶									
None	5773	240	1.00	10	1.00	78	1.00	19	1.00
Past or Current	18286	918	1.06 (0.90 – 1.25)	87	2.34 (1.15 – 4.76)	403	1.43 (1.09 – 1.88)	107	1.09 (0.63 – 1.88)
Work ETS exposure **									
None	20096	904	1.00	74	1.00	353	1.00	92	1.00
Past or Current	3963	254	1.40 (1.21 – 1.62)	23	1.60 (0.99 – 2.57)	128	1.70 (1.38 – 2.09)	34	1.86 (1.25 – 2.78)

*Chronic bronchitis was defined as combined chronic cough and phlegm.

†All ORs are adjusted for age and dialect group.

‡ORs adjusted for adult home (no, past or current) and work (no, past or current) exposure.

§Number of smokers for childhood ETS exposure is a semi-quantitative variable representing the minimum number of smokers.

¶ORs adjusted for childhood (no, 1+ smokers) and work (no, past or current) exposure.

**ORs adjusted for childhood (no, 1+ smokers) and adult home (no, past or current) exposure.

Table 4

Chronic phlegm and chronic cough in relation to childhood environmental tobacco smoke (ETS) stratified by fiber intake*

	No Cough, Phlegm, or Asthma	Chronic Phlegm		Chronic Cough	
		N	OR (95% CI)†	N	OR (95% CI)
Fiber intake below the median ‡					
Childhood home ETS exposure					
None	4616	75	1.00	12	1.00
1 Smoker	4453	100	1.28 (0.91–1.80)	34	3.12 (1.51–6.46)
2 Smokers	2891	72	1.38 (0.94–2.02)	30	4.59 (2.13–9.90)
3 or more Smokers	1247	49	2.13 (1.38–3.27)	13	4.99 (2.03–12.25)
1 or more Smokers	8591	221	1.39 (1.01–1.90)	77	3.63 (1.82–7.27)
Fiber intake above the median					
Childhood home ETS exposure					
None	6395	108	1.00	24	1.00
1 Smoker	6146	146	1.11 (0.84–1.48)	41	1.54 (0.86–2.74)
2 Smokers	3758	109	1.28 (0.94–1.76)	21	1.18 (0.59–2.36)
3 or more Smokers	1636	41	1.08 (0.72–1.63)	10	1.25 (0.53–2.93)
1 or more Smokers	11540	296	1.16 (0.89–1.51)	72	1.41 (0.81–2.48)

* Fiber is estimated by non-starch polysaccharide intake.

† Odds ratios adjusted for age, dialect group, gender and adult home exposure (no, 1 past, 2+ past, 1+ current smokers) and work exposure (none versus past or current).

‡ Median in the entire Singapore Chinese Cohort of calorie-adjusted non-starch polysaccharide intake = 7.5 grams/day.

Table 5

Respiratory symptoms in relation to environmental tobacco smoke (ETS) during early life and adulthood

	No Cough, Phlegm or Asthma	Asthma Diagnosis		Chronic Bronchitis*		Chronic Phlegm without Cough		Chronic Dry Cough
	N	N	OR (95% CI)†	N	OR (95% CI)	N	OR (95% CI)	N	OR (95% CI)
ETS exposure									
No childhood or adult exposure‡	5461	214	1.00	12	1.00	61	1.00	14	1.00
Childhood Only	1560	55	0.89 (0.66–1.21)	3	0.89 (0.25–3.14)	30	1.62 (1.04–2.52)	16	4.03 (1.96–8.29)
Adult Only	5550	259	1.19 (0.99–1.43)	24	2.06 (1.03–4.12)	122	2.11 (1.55–2.88)	22	1.57 (0.80–3.08)
Both child and adult	18571	976	1.32 (1.13–1.53)	110	2.87 (1.58–5.22)	487	2.38 (1.82–3.12)	133	2.80 (1.61–4.87)

* Chronic bronchitis was defined as combined chronic cough and phlegm.

† All odds ratios (OR) and 95% confidence intervals are adjusted for age, gender, and dialect.

‡ Adult exposure includes home and/or work exposure.