Published in final edited form as:
Am J Epidemiol. 2006 June 15; 163(12): 1118–1128.

Vapor, Dust and Smoke Exposure in relation to adult-onset asthma and chronic respiratory symptoms: The Singapore Chinese Health Study

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Abstract

Occupational factors contribute to a significant fraction of respiratory disease and symptoms. We evaluated the role of occupational exposures on asthma, chronic bronchitis, and respiratory symptoms in a population-based cohort, the Singapore Chinese Health Study. History of occupations, occupational exposures, and respiratory conditions were collected by interviews with 52,325 Singaporeans born 1918–1953. Exposure to dusts, from cotton, wood, metal, mineral and/or asbestos, was associated with non-chronic cough and/or phlegm (OR = 1.19, 95% CI = 1.08, 1.30), chronic bronchitis (OR = 1.26, 95% CI = 1.01, 1.57) and adult-onset asthma (OR = 1.14, 95% CI = 1.00, 1.30). Cotton dust was the major component contributing to respiratory symptoms. Vapor exposure, from chemical solvents, dyes, cooling oils, paints, wood preservatives and/or pesticides, was associated with non-chronic cough or phlegm (OR = 1.14, 95% CI = 1.03, 1.27), chronic dry cough (OR = 1.55, 95% CI = 1.19, 2.01) and adult-onset asthma (OR = 1.34, 95% CI = 1.15, 1.56). Chemical solvents, cooling oils and pesticides were the major sources contributing to respiratory symptoms. These data support the role of occupational exposures in the etiology of respiratory illness in a population-based cohort in Singapore with a low prevalence of atopic illness.

Keywords

Asthma; Chronic Bronchitis; Occupational Disease; Occupational exposure; Chronic obstructive pulmonary disease

Abbreviations

OR - odds ratio; CI - confidence interval; COPD - chronic obstructive pulmonary disease

There is growing recognition that occupational exposures make a substantive contribution to adult-onset asthma, chronic bronchitis and other respiratory symptoms [1,2]. The American Thoracic Society recently estimated that 15% of these conditions are work related [3]. More

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than 300 different workplace exposures have been associated with occupational asthma or chronic bronchitis [4–6]. Exposures can be classified according to the possible pathogenic mechanisms. Sensitizing substances cause asthma by inducing specific IgE antibodies [1,7]. These are mostly high-molecular weight allergens of animal or vegetable origin. Others are low-molecular weight compounds, such as organic and inorganic compounds that act as haptens interacting with a protein to form a complete antigen. Notably, an IgE mechanism has not been demonstrated consistently for exposure to agents such as diisocyanates, western red cedar and acrylates. The reaction between the antigen and specific IgE antibodies initiates a cascade of events that result in an allergic inflammatory reaction in the airways. Sensitizing agents induce respiratory symptoms after a latent interval of several months- occasionally weeks or years following exposure [8]. Agents that contribute to adverse respiratory symptoms by non-immunological mechanisms, mostly irritant vapors such as chlorine, sulfur dioxide, combustion products and ammonia, may lead to bronchial epithelial cell damage, release of inflammatory mediators and airway remodeling [9,10]. An irritant-induced respiratory symptom typically develops within hours of an acute inhalation, although irritants can lead to chronic respiratory impairment [8].

Much of the data relating occupational exposure and respiratory disease and symptoms come from cross-sectional studies conducted in industrial groups with high levels of exposure [11, 12]. However, these studies may suffer from the healthy worker effect, selection bias due to affected workers leaving highly exposed jobs and this tends to result in underestimation of the true risk [13,14]. Because workers who have changed employment remain in the general population, this bias may be minimized by sampling subjects from the general population [15]. Community-based studies have supported a role for occupational exposures in respiratory disease but nearly all of these studies have been conducted in Western populations [16]. We examined the relationship between self-reported exposure to occupational dust (cotton, wood, metal, mineral), smoke (welding, burning coal, burning wood) or vapor (chemical solvents, dyes, cooling oils, paints, wood preservatives, pesticides) and adult-onset doctor-diagnosed asthma and symptoms of cough and phlegm in a large population-based cohort of adult Singaporeans of Chinese ethnicity, the Singapore Chinese Health Study.

Materials and Methods

Study population

The design of the Singapore Chinese Health Study has been described [17]. Briefly, the cohort was recruited between 1993 and 1998, drawn from permanent residents or citizens of Singapore who resided in government-built housing (86% of the Singapore population reside in such facilities). Men and women of Chinese ethnicity (Cantonese or Hokkien dialect), aged 45 to 74 years were eligible. A total of 63,257 individuals (~ 85% of eligible subjects) were enrolled. A baseline questionnaire was administered in-person by trained interviewers to collect data on smoking history, diet, medical history and occupations. A follow-up telephone interview was completed between 1999 and 2004 for 52,325 cohort members. The average time between the baseline and follow-up interviews was 5.8 years. The follow-up interview included detailed information on environmental tobacco smoke exposure, medical history including, doctor diagnosis of hay fever, allergic rhinitis, sinusitis or eczema, and items on asthma, cough and phlegm from the American Thoracic Society questionnaire (ATS-DLD-78-A). These items were not obtained on the baseline questionnaire and items on occupation were not obtained on the follow-up questionnaire. The Institutional Review Boards at the University of Southern California (Los Angeles, CA), the National University of Singapore and the National Institute of Environmental Health Sciences (Research Triangle Park, NC) have approved this study. Informed written consent was obtained from all participants. As of December 31, 2004, there were 7,722 deaths in the overall cohort.

We categorized the 52,316 individuals with complete respiratory outcome data (nine individuals had missing data on duration of symptoms) into mutually exclusive categories consisting of doctor-diagnosed adult-onset asthma (n = 1,426) (defined as onset of symptoms after age 18), doctor-diagnosed childhood asthma (878), chronic dry cough (364), chronic phlegm (1,475), chronic cough plus chronic phlegm (chronic bronchitis, 417), or non-chronic cough and/or phlegm (2,652). The referent group consisted of subjects who did not report any of the following – cough, phlegm or asthma (whether childhood or adult onset) (45,104). We do not present data for the 878 childhood-onset asthmatics because the illness would likely have begun prior to occupational exposures. Chronic was defined as occurring on most days for at least three months of the year and lasting more than two years in a row. We first classified subjects according to doctor-diagnosed asthma and then classified those without this report according to the presence of chronic cough and/or phlegm. Thus, subjects with asthma were classified as asthmatic, even if they also had cough and/or phlegm. The use of mutually exclusive categories allows all subjects to be compared to a common reference category of subjects free from asthma, cough or phlegm and thus better answers the question of whether occupational exposures are related to respiratory outcomes in general.

Occupation was assessed by 45 questions on the baseline questionnaire regarding current or previous jobs and exposures plus corresponding questions on duration of exposure. Subjects were asked if they worked in any of the following industries for one year or longer – manufacture and repair of boots, shoes or other leather goods, manufacture of furniture and cabinets, cotton textile, electrical and electronic industry, lumber and sawmill, carpentry or joinery, rubber and tire manufacturing, manufacture of dyes or dyestuffs, manufacture of paints, manufacture of plastics, petroleum, metal production or processing, construction work, urea formaldehyde manufacturing. With respect to specific job categories, subjects were asked if they had a job lasting one year or longer as a welder, textile machine mechanic, other mechanic, cotton spinner or weaver, painter, textile dyer, machinist, printer, tailor or seamstress, janitor or cleaner, vocational driver or food hawker, cook or other kitchen worker. Subjects were also asked if they were exposed on the job (lasting for one year or longer) to any of the following substances: cotton dust, wood dust, wood preservatives, metal dust or fumes, rock or mineral dust, smoke (all types), smoke from welding, smoke from burning coal or coke, smoke from burning wood, other smoke, pesticides, asbestos, coal tar, soot, pitch, acid or alkali solutions, chemical solvents, dyes or dyestuffs, cutting, cooling or lubricating oils, paints, or formaldehyde. For each question, duration was also asked in categories of 1–4 years, 5–9 years, 10–14 years, 15–19 years, 20+ years. Dust exposure included cotton dust, wood dust, metal dust or mineral dust (including asbestos) in a job lasting one year or longer. Smoke derived from welding, burning coal, burning wood, or “other” sources was used as the composite smoke variable. Vapor exposure included wood preservatives, pesticides, chemical solvents, dyes, cutting, cooling or lubricating oils, or paints. We did not ask information on the year in which exposure began or ended.

Statistical analyses

We examined exposures at baseline, in relation to the outcome of respiratory symptoms reported on the follow-up questionnaire. Adjusted odds ratios (ORs) and 95% confidence intervals (CI) were calculated from polytomous logistic regression models using PROC CATMOD [18] (version 9.1; SAS Institute, Cary, NC), except that when the expected cell number was ≤ 5, ORs were not calculated. We adjusted all models for age, gender and dialect, although they did not necessarily alter the exposure-outcome associations. For most of the respiratory outcomes, individuals were more likely to be current smokers and to smoke more than those in the referent group; therefore, we carefully controlled for potential confounding by including terms for smoking status (never, current, past), and among smokers, age at starting to smoke and amount smoked in adjusted models [19]. The inclusion of current age and age at

starting to smoke captures smoking duration. Individuals were categorized as never smokers if they had never smoked at least one cigarette per day for 1 year or longer. We created a variable for atopy based on yes or no responses to questions on ever diagnosis of allergic rhinitis, sinusitis, hay fever or eczema. Inclusion of terms for education, exposure to environmental tobacco smoke as a child or adult (at work and/or at home) or for atopy did not alter the odds ratios (change of less than 10%) and thus were not included in the final models.

Results

Study demographics

At follow-up, 2.7% of the cohort reported adult-onset asthma diagnosis, 0.7% reported chronic dry cough, 2.8% reported chronic phlegm, 0.8% reported the combination of chronic cough and phlegm (the definition of chronic bronchitis), and 5.1% reported cough and/or phlegm that did not meet the definition of chronic (Table 1). Compared with individuals without asthma, cough or phlegm, individuals in all outcome categories were more likely to be current smokers, to have started smoking before the age of 20 years, and to smoke more than subjects without asthma or symptoms (Table 1). Level of education differed modestly by case status. Not surprisingly, symptomatic and asthmatic individuals were more likely to be atopic, although the prevalence of atopy was low, consistent with other studies in Chinese populations [20].

Association between exposure and respiratory symptoms

Exposure to dust, a composite variable combined from reported exposure to cotton, wood, metal, mineral and/or asbestos, was associated with non-chronic cough and/or phlegm (OR = 1.19, 95% CI = 1.08, 1.30), chronic bronchitis (OR = 1.26, 95% CI = 1.01, 1.57) and adult-onset asthma (OR = 1.14, 95% CI = 1.00, 1.30) (Table 2). Individuals exposed to smoke from welding, coal burning, wood burning or other sources had an increased odds for non-chronic cough or phlegm compared to controls (OR = 1.12, 95% CI = 1.01, 1.25) (Table 2). Vapor exposure, derived from chemical solvents, dyes, cutting, cooling or lubricating oils, paints, formaldehyde, wood preservatives and/or pesticides, was associated with non-chronic cough and/or phlegm (OR = 1.14, 95% CI = 1.03, 1.27), chronic dry cough (OR = 1.55, 95% CI = 1.19, 2.01) and adult-onset asthma (OR = 1.34, 95% CI = 1.15, 1.56) (Table 2). No differences were observed when individuals reporting “never worked” were excluded from the analyses (data not shown).

We examined possible effect modification of the association between dust, smoke and vapor exposure and respiratory symptoms by gender, smoking (ever, never), atopy and fiber intake, a protective factor for chronic bronchitis symptoms in this cohort [21]. No appreciable differences were found for gender or fiber intake (data not shown). Given the low prevalence of atopy, power to detect differences by atopic status was limited, and none were observed (data not shown) [20]. Smoking status (never vs. ever) significantly modified the association between exposure to dust, smoke or vapors and chronic dry cough (Table 3). For chronic dry cough, the effects of all three broad exposure groups were greater in nonsmokers than in smokers, with p-values for interaction all less than 0.04.

Although numbers become small, we looked at individual sources of dust and found the strongest association for the cotton dust variable (Table 4). Cotton dust exposure was associated with non-chronic cough or phlegm (OR = 1.40, 95% CI = 1.19, 1.64), chronic phlegm (OR = 1.36, 95% CI = 1.09, 1.69) and chronic bronchitis (OR = 1.71, 95% CI = 1.13, 2.60). The duration of exposure was associated with a significant trend for all three outcomes ($p < 0.03$). Individuals reporting exposure to cotton dust more often indicated work in the cotton textile industry (14%) or as a tailor or seamstress (71%) than the other industries or occupations on our questionnaire. There was an association between being a tailor or seamstress and chronic

bronchitis (OR = 1.35, 95% CI = 0.88, 2.06), chronic phlegm (OR = 1.39, 95% CI = 1.14, 1.70) or chronic dry cough (OR = 1.32, 95% CI = 0.88, 1.99).

Although we did not find any statistically significant relationship between the individual smoke components and respiratory symptoms (Table 5), chronic phlegm was associated with coal burning (OR = 1.45, CI = 0.95, 2.20) and adult-onset asthma was associated with wood burning (OR = 1.48, CI = 0.92, 2.40). Of note, individuals reporting exposure to smoke from coal or wood were frequently employed as food hawkers (41% and 26%, respectively) who cook food in covered outdoor or indoor stalls.

Given the association with vapor exposure, we examined the individual sources of vapors (Table 6). Exposure to chemical solvents was associated with an increased odds for non-chronic cough or phlegm (OR = 1.20, 95% CI = 1.05, 1.37), chronic dry cough (OR = 1.63, 95% CI =1.17, 2.27) and adult-onset asthma (OR = 1.44, 95% CI = 1.19, 1.74). The duration of exposure was associated with a significant trend in all cases ($p < 0.03$). Subjects reporting exposure to vapors were often in the construction (9%), metal processing (7%) and electronic (9%) industry and/or had occupations such as a mechanic (18%), vocational driver (9%) or painter (7%). In addition, being a cleaner or janitor, an occupation often exposed to chemical solvents, was significantly associated with adult onset asthma (OR = 1.24, 95% CI = 1.03, 1.50).

Another source of vapors –cutting, cooling or lubricating oils- was associated with chronic dry cough (OR = 1.46, 95% CI = 1.02, 2.08; trend, $p = 0.05$). Of those exposed to cutting, cooling or lubricating oils, 12% reported working as a machinist and an additional 9% had worked in the metal processing industries.

Occupational pesticide exposure was limited in this general population cohort, yet we observed a moderate association with adult-onset asthma (OR = 1.69, 95% CI = 1.13, 2.52). Among individuals reporting pesticide exposure, the most commonly reported types of work were construction (22%), vocational drivers (10%) and/or janitor/cleaner (9%). We observed an increased odds, albeit of borderline statistical significance, between exposure to dyes/dyestuffs and non-chronic cough or phlegm (OR = 1.46, 95% CI = 0.97, 2.20), chronic phlegm (OR = 1.57, 95% CI = 0.94, 2.61) and adult-onset asthma (OR = 1.61, 95% CI = 0.92, 2.82). Individuals with exposure to dyes were most often printers (16%).

Discussion

These data from the Singapore Chinese Health Study support the role of occupational exposures in the etiology of doctor-diagnosed adult-onset asthma, symptoms of chronic bronchitis, chronic dry cough or phlegm as well as less than chronic cough and/or phlegm. Exposure to dust and vapors were especially important. Population-based studies from Westernized countries have consistently demonstrated an increased relative risk for respiratory symptoms associated with occupational exposure to dusts, gases and/or fumes [22–31]; there are few studies in Asian populations [32].

Cotton dust exposure was the primary source of the association with dusts in this cohort. Work as a tailor/seamstress was much more common than work in the textile industry among subjects reporting exposure to cotton dust. Extensive literature links exposure to cotton dust, from work in the textile industry, to chronic respiratory symptoms and illness [33]. Our study extends these observations to include an increased risk for chronic bronchitis, chronic phlegm and chronic dry cough among individuals exposed primarily as tailors and seamstresses who would be expected to have much lower levels of exposure than workers in the textile industry.

Although sensitizations to wood dust, metal dust or mineral dust are well-recognized causes of respiratory symptoms, our population included few exposed subjects and thus we were not

well powered to explore these associations. It is also possible that exposures may be lower in this population than others, the component exposures may differ and/or our questions on occupational history may have lacked specificity to capture these exposures.

In our Singapore cohort, we found an association between chemical solvent exposure and non-chronic cough or phlegm, chronic dry cough and adult-onset asthma. We did not ask about the type of solvent used; therefore we are limited in our ability to identify the potential etiologic agents. However, chemical solvents are known respiratory irritants affecting the mucosa [34, 35]. Chemical solvents have a wide variety of industrial applications, including the manufacture of paints, inks, cleaning products, adhesives and petrochemicals. Of note, we found that individuals having an occupation as a janitor or cleaner had higher odds of adult-onset asthma compared to those without that job history. A modest increased risk from working as a janitor or cleaner is supported in other population-based studies [36] from the European Community Respiratory Health Survey (ECRHS) study [37], the US [38] and Finland [11]. While asthma was not related to occupation as a cleaner in a community-based study involving seven French cities, exposure to industrial cleaning agents was [15]. Our results add to the growing body of evidence for a role of cleaning agents in asthma.

Exposure to cooling, lubricating and cutting oil aerosols occurs at highest levels in the automotive industry and has been found to be associated with a variety of respiratory symptoms including cross-shift changes in pulmonary function, asthma, cough and phlegm [39–41]. Our self-reported exposure category no doubt was subject to a high level of misclassification. Nonetheless, in our Singapore cohort, individuals reporting exposure to cutting, cooling or lubricating oils reported an increased prevalence of symptoms of non-chronic cough or phlegm, chronic dry cough and doctor-diagnosed adult-onset asthma. Microbial growth in the predominant aqueous component of cooling and lubricating oils may drive the association with respiratory illness [39,42].

Pesticide exposure in this cohort was associated with adult-onset asthma. We did not have information regarding the use of specific pesticides; however various pesticides, including organophosphates, carbamates, fungicides, fumigants and paraquat, can produce adverse respiratory effects [43]. Pesticide exposure contributes to wheeze in agricultural workers [44] and other outdoor workers [45]. Agriculture is not a major industry in the small city state of Singapore: individuals exposed to pesticides most commonly worked in the construction industry or as a janitor/cleaner. Exposure to pesticides in the construction industry may be a result of chemical pesticide treatment to wood for outdoor construction projects [45]. We suggest that construction workers and cleaners may be exposed through pesticide treatment of homes or buildings.

This study enhances the existing literature because there are few data on occupational exposure in general population studies in Asia. Singapore provides a unique opportunity within Asia because ambient air pollution levels are lower than most Asian cities and comparable to US cities [46,47]. Smoking is illegal in the workplace in Singapore [48] and there is a relatively high proportion (72%) of never smokers. In addition, since Singapore is tropical, our cohort was not exposed to coal or wood burning in the home for heating, unlike in China. Thus, confounding by these exposures is minimized.

The magnitude of the effects we observed were modest and there is always the theoretical concern that unknown biases may contribute to modest associations. Nonetheless, the large size of this dataset gave us excellent power to detect associations of the modest size that we would expect between occupational exposure and chronic respiratory symptoms.

We lacked information on the precise timing of exposure in relation to the start of respiratory symptoms; therefore we are unable to address the impact of exposures on work-aggravated

asthma, which is a significant component of the effects of occupation [49]. In addition, we are unable to address whether individuals may have selected “cleaner” occupations because of the early presence of respiratory symptoms. If such a bias were operating, the impact would be that we underestimated the associations.

Although we lacked data on precise timing of exposures, occupational exposures were assessed on the baseline questionnaire and respiratory symptoms were assessed five years later at follow-up thereby reducing biased recall of occupational exposure contingent on respiratory symptoms or vice-versa. Differential misclassification of self-reported exposures may occur by asthma diagnosis [50]. However, we found associations for symptoms not closely related to asthma in this cohort, rather than simply asthma diagnosis.

A total of 5,339 subjects died prior to the follow-up interview creating the possibility for bias. However, a strength of this study is that we had cause of death data on decedents and found that among subjects who died without follow-up, exposure to dust, fumes or vapors was not related to death from respiratory disease as opposed to other causes, suggesting that we do not have significant nonresponse bias due to death.

We first categorized subjects based on asthma and then secondarily on cough and phlegm. Thus there will be a slight loss of power for analyses of cough and phlegm. However, the overlap was minimal; among the asthmatics, 6% reported chronic bronchitis, 2% reported chronic dry cough and 6% reported chronic phlegm.

A self-report of doctor-diagnosed asthma was used to classify subjects as asthmatic. It has been found that questions on “reported asthma” and “doctor-diagnosed” asthma had good positive predictive value and specificity in predicting current asthma [51]. We re-interviewed 331 of 406 individuals who reported incident asthma on the follow-up questionnaire (82%) and among these, 313 (95%) reconfirmed asthma diagnosis or asthma symptoms.

There is growing evidence from large population-based studies that a sizable proportion of chronic obstructive pulmonary disease may be attributable to workplace exposures [52]. Although we did not investigate chronic obstructive pulmonary disease (COPD) *per se* because of the unreliability of self-report of chronic obstructive pulmonary disease in the general population [53], we included chronic bronchitis, a symptom complex of COPD defined by questionnaire responses alone. However, some people with COPD may not have symptoms, thus were included in the referent group of “no symptoms”. Inclusion of people with COPD in the referent group would tend to attenuate associations.

In this cohort of Chinese Singaporeans, we found significant associations, independent of tobacco smoking or environmental tobacco smoke exposure, between occupational exposures and adult-onset asthma, chronic bronchitis, chronic cough and/or phlegm and non-chronic cough/and/or phlegm. Specific exposures included cotton dust, chemical solvents, cooling and lubricating oils and pesticides. Given that a substantial proportion (42.5%) of our subjects were over the common Singaporean retirement age of 62, our data suggest that occupational exposures can have long-lasting effects on respiratory symptoms.

Acknowledgements

This research was funded by the National Institutes of Health (NCI RO1 CA80205, NIEHS Z01 ES43012 and NIEHS KO1 ES00386). We thank Siew-Hong Low of the National University of Singapore for supervising the fieldwork of the Singapore Chinese Health Study, Kazuko Arakawa of the University of Southern California for the development and management of the cohort study database, and Marsha Shepherd of Westat, Inc. for programming.

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TABLE 1

Characteristics of participants in the Singapore Chinese Health Study by respiratory symptom status

	No Symptoms n = 45104		Non-Chronic Cough or Phlegm[#] n = 2652		Chronic Phlegm n = 1475		Chronic Dry Cough n = 364		Chronic Bronchitis n = 417		Adult- Onset Asthma[§] n =1426	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Age, mean yr (SD)	55.7 (7.7)		57.1 (7.9)		55.8 (8.1)		55.9	(7.4)	57.5 (8.2)		57.4 (8.0)	
Baseline [*]												
Sex, Male	18319	41	1528	58	921	62	215	59	282	68	626	44
Dialect												
Cantonese	21659	48	1526	58	773	52	144	40	168	40	652	46
Hokkien	23445	52	1126	42	702	48	220	60	249	60	774	54
Education												
≤ grade 6	11989	27	519	20	244	17	85	23	87	21	386	27
grade 7–10	19972	44	1290	49	702	48	169	46	214	51	651	46
≥ grade 11	13143	29	843	32	529	36	110	30	116	28	389	27
Smoking												
never	33335	74	1440	54	780	53	212	58	170	41	962	67
past	4591	10	323	12	202	14	36	10	54	13	175	12
current	7178	16	889	34	493	33	116	32	193	46	289	20
Age started smoking [‡]												
≤ 14 yr	1264	18	173	19	94	19	34	29	53	27	70	24
15–19	2598	36	343	39	214	43	42	36	68	35	105	36
≥ 20 yr	3316	46	373	42	185	38	40	34	72	37	114	39
Cigarettes smoked per day [‡]												
≤ 12	3105	43	308	35	163	33	36	31	55	28	120	42
13–22	2897	40	402	45	219	44	51	44	103	53	108	37
≥ 23	1176	16	179	20	111	23	29	25	35	18	61	21
ETS												
Child < 18 yr	31327	69	2035	77	1176	80	303	83	343	82	1050	74
Adult ≥ 18 yr	36476	81	2333	88	1326	90	324	89	388	93	1232	86
Atopy [†]	3493	8	337	13	232	16	63	17	63	15	260	18

Definition of abbreviation: SD = standard deviation, ETS = environmental tobacco smoke

[*] Baseline questionnaire administered 1993–1998

[†] Atopy as self-reported hay fever, allergic rhinitis, sinusitis or eczema.

[‡] Percent of current smokers

[§] 878 subjects with childhood-onset asthma were not reported

[#] Answered yes to at least one of the following questions: Do you usually cough when you get up in the morning? Do you usually cough at all during the rest of the day or at night? Do you usually bring up phlegm from your chest when you get up in the morning? Do you usually bring up phlegm from your chest during the rest of the day or at night?

TABLE 2

Odds ratios by exposure to dust, smoke or vapors in relation to respiratory symptoms: The Singapore Chinese Health Study

	No Symptoms	Non-Chronic Cough or Phlegm			Chronic Phlegm			Chronic Dry Cough			Chronic Bronchitis			Adult-Onset Asthma		
	n	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI
Dust †	35967	1922	1.0		1082	1.0		271	1.0		296	1.0		1110	1.0	
	9137	730	1.19 #	1.08, 1.30	393	1.08	0.95, 1.22	93	1.15	0.90, 1.46	121	1.26	1.01, 1.57	316	1.14	1.00, 1.30
Smoke ‡	39368	2164	1.0		1212	1.0		305	1.0		338	1.0		1220	1.0	
	5736	488	1.12	1.01, 1.25	263	1.01	0.87, 1.16	59	1.00	0.75, 1.34	79	1.07	0.83, 1.38	206	1.13	0.97, 1.33
Vapors §	39184	2134	1.0		1196	1.0		282	1.0		345	1.0		1189	1.0	
	5920	518	1.15	1.03, 1.27	279	1.03	0.89, 1.18	82	1.55	1.19, 2.01	72	0.93	0.71, 1.22	237	1.34	1.15, 1.56

Definition of abbreviations: CI = confidence interval; OR = odds ratio.

* Models are adjusted for age, gender, dialect, smoking status (never, former, current), age at starting to smoke (≥ 20, 15–19, ≤ 14 years), and cigarettes per day (≤ 12, 13–22, ≥ 23).

† Dust = cotton, wood, metal, mineral, asbestos

‡ Smoke = welding, coal burning, wood burning, other

§ Vapors = chemical solvents, dyes, cutting, cooling or lubricating oils, paints, formaldehyde, wood preservatives, pesticides

Odds ratio relative to the reference category equal to OR 1.0 for unexposed individuals

Am J Epidemiol. Author manuscript; available in PMC 2006 July 17.

TABLE 3

Odds ratios by exposure to dust, smoke or vapors in relation to respiratory symptoms: stratification by smoking

	No Symptoms	Non-Chronic Cough or Phlegm			Chronic Phlegm			Chronic Dry Cough			Chronic Bronchitis			Adult-Onset Asthma		
	n	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI
Dust †																
never smoker	27719	1123	1.0		621	1.0		160	1.0		137	1.0		791	1.0	
	5616	317	1.30 #	1.14, 1.48	159	1.11	0.93, 1.33	52	1.57	1.13, 2.16	33	1.15	0.78, 1.70	171	1.11	0.93, 1.32
ever smoker	8248	799	1.0		461	1.0		111	1.0		159	1.0		319	1.0	
	3521	413	1.08	0.95, 1.23	234	1.04	0.88, 1.22	41	0.82	0.57, 1.19	88	1.31	1.00, 1.72	145	1.20	0.97, 1.47
Smoke ‡																
never smoker	30309	1272	1.0		696	1.0		182	1.0		154	1.0		870	1.0	
	3026	168	1.20	1.01, 1.42	84	0.97	0.77, 1.23	30	1.41	0.94, 2.12	16	0.86	0.51, 1.47	92	1.07	0.85, 1.34
ever smoker	9059	892	1.0		516	1.0		123	1.0		184	1.0		350	1.0	
	2710	320	1.09	0.95, 1.25	179	1.02	0.85, 1.22	29	0.75	0.50, 1.14	63	1.15	0.85, 1.54	114	1.21	0.97, 1.51
Vapors §																
never smoker	30186	1265	1.0		681	1.0		168	1.0		152	1.0		842	1.0	
	3149	175	1.15	0.96, 1.36	99	1.09	0.87, 1.36	44	2.33	1.62, 3.35	18	0.94	0.56, 1.57	120	1.43	1.16, 1.76
ever smoker	8998	869	1.0		515	1.0		114	1.0		193	1.0		347	1.0	
	2771	343	1.14	0.99, 1.31	180	0.98	0.82, 1.17	38	1.07	0.73, 1.57	54	0.92	0.67, 1.26	117	1.25	1.00, 1.57

Definition of abbreviations: CI = confidence interval; OR = odds ratio.

* Models are adjusted for age, gender, dialect, smoking status (never, former, current), age at starting to smoke (≥ 20, 15–19, ≤ 14 years), and cigarettes per day (≤ 12, 13–22, ≥ 23).

† Dust = cotton, wood, metal, mineral, asbestos

‡ Smoke = welding, coal burning, wood burning, other

§ Vapors = chemical solvent, dyes, cutting, cooling or lubricating oils, paints, formaldehyde, wood preservatives, pesticides

Odds ratio relative to the reference category equal to 1.0 for unexposed individuals

TABLE 4

Odds ratios by exposure to sources of dust in relation to respiratory symptoms: The Singapore Chinese Health Study

	No Symptoms	Non-Chronic Cough or Phlegm			Chronic Phlegm			Chronic Dry Cough			Chronic Bronchitis			Adult-Onset Asthma		
	n	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI
Cotton dust	42330	2472	1.0		1383	1.0		342	1.0		392	1.0		1345	1.0	
	2774	180	1.40 §	1.19, 1.64	92	1.36	1.09, 1.69	22	1.39	0.89, 2.16	25	1.71	1.13, 2.60	81	1.05	0.84, 1.33
Wood dust	43004	2470	1.0		1373	1.0		339	1.0		381	1.0		1351	1.0	
	2100	182	1.06	0.91, 1.25	102	1.01	0.82, 1.25	25	1.11	0.74, 1.69	36	1.25	0.88, 1.78	75	1.09	0.85, 1.38
Metal dust	42089	2379	1.0		1314	1.0		332	1.0		376	1.0		1317	1.0	
	3015	273	1.09	0.95, 1.25	161	1.11	0.93, 1.32	32	1.03	0.71, 1.50	41	1.04	0.74, 1.45	109	1.15	0.94, 1.42
Mineral dust	41969	2371	1.0		1307	1.0		330	1.0		375	1.0		1312	1.0	
†	3135	281	1.08	0.94, 1.23	168	1.11	0.93, 1.32	34	1.06	0.73, 1.53	42	1.02	0.73, 1.42	114	1.16	0.95, 1.43

Definition of abbreviations: CI = confidence interval; OR = odds ratio.

*Models are adjusted for age, gender, dialect, smoking status (never, former, current), age at starting to smoke (≥ 20, 15–19, ≤ 14 years), and cigarettes per day (≤ 12, 13–22, ≥ 23).

†Mineral dust includes asbestos

§Odds ratio relative to the reference category equal to OR 1.0 for unexposed individuals

Am J Epidemiol. Author manuscript; available in PMC 2006 July 17.

TABLE 5

Odds ratios by exposure to sources of smoke in relation to respiratory symptoms: The Singapore Chinese Health Study

	No Symptoms	Non-Chronic Cough or Phlegm			Chronic Phlegm			Chronic Dry Cough			Chronic Bronchitis			Adult-Onset Asthma		
	n	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI
Welding	42829	2427	1.0		1361	1.0		341	1.0		386	1.0		1345	1.0	
	2275	225	1.13 ‡	0.97, 1.32	114	0.94	0.76, 1.15	23	0.95	0.61, 1.48	31	1.03	0.70, 1.51	81	1.17	0.92, 1.48
Coal Burning	44728	2618	1.0		1451	1.0		360			413			1411	1.0	
	376	34	1.14	0.80, 1.64	24	1.45	0.95, 2.20	4	†		4	†		15	1.23	0.73, 2.07
Wood Burning	44745	2622	1.0		1459	1.0		360			411			1408	1.0	
	359	30	1.02	0.70, 1.50	16	0.96	0.58, 1.60	4	†		6	†		18	1.48	0.92, 2.40
Other	41474	2359	1.0		1312	1.0		324	1.0		364	1.0		1296	1.0	
	3630	293	1.12	0.98, 1.27	163	1.06	0.89, 1.25	40	1.09	0.78, 1.52	53	1.13	0.84, 1.52	130	1.09	0.90, 1.31

Definition of abbreviations: CI = confidence interval; OR = odds ratio.

*Models are adjusted for age, gender, dialect, smoking status (never, former, current), age at starting to smoke (≥ 20, 15–19, ≤ 14 years), and cigarettes per day (≤ 12, 13–22, ≥ 23).

† OR was not calculated

‡ Odds ratio relative to the reference category equal to OR 1.0 for unexposed individuals

Am J Epidemiol. Author manuscript; available in PMC 2006 July 17.

TABLE 6

Odds ratios by exposure to sources of vapors in relation to respiratory symptoms: The Singapore Chinese Health Study

	No Symptoms	Non-Chronic Cough or Phlegm			Chronic Phlegm			Chronic Dry Cough			Chronic Bronchitis			Adult-Onset Asthma		
	n	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI	n	OR*	95% CI
Chemical Solvents	42093	2384	1.0		1333	1.0		321	1.0		382	1.0		1298	1.0	
	3011	268	1.20‡	1.05, 1.37	142	1.08	0.90, 1.29	43	1.63	1.17, 2.27	35	1.02	0.71, 1.45	128	1.44	1.19, 1.74
Dyes	44827	2626	1.0		1459	1.0		359			416			1413	1.0	
	277	26	1.46	0.97, 2.20	16	1.57	0.94, 2.61	5	†		1	†		13	1.61	0.92, 2.82
Cooling Oils	42532	2403	1.0		1336	1.0		326	1.0		387	1.0		1331	1.0	
	2572	249	1.12	0.97, 1.30	139	1.07	0.89, 1.29	38	1.46	1.02, 2.08	30	0.81	0.55, 1.19	95	1.15	0.92, 1.43
Paints	43163	2459	1.0		1385	1.0		336	1.0		386	1.0		1352	1.0	
	1941	193	1.16	0.99, 1.36	90	0.88	0.71, 1.10	28	1.34	0.90, 2.00	31	1.12	0.76, 1.63	74	1.19	0.93, 1.52
Wood Preservatives	44724	2621	1.0		1452	1.0		357			412			1410	1.0	
	380	31	0.97	0.67, 1.41	23	1.24	0.81, 1.90	7	†		5	†		16	1.30	0.78, 2.15
Pesticides	44647	2622	1.0		1461	1.0		358			412			1400	1.0	
	457	30	0.98	0.67, 1.42	14	0.78	0.46, 1.34	6	†		5	†		26	1.69	1.13, 2.52

Definition of abbreviations: CI = confidence interval; OR = odd ratio.

*Models are adjusted for age, gender, dialect, smoking status (never, former, current), age at starting to smoke (≥ 20, 15–19, ≤ 14 years), and cigarettes per day (≤ 12, 13–22, ≥ 23).

†OR was not calculated

‡Odds ratio relative to the reference category equal to OR 1.0 for unexposed individuals