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Passive smoking and chronic obstructive pulmonary disease: cross-sectional analysis of data from the Health Survey for England

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ABSTRACT

Objectives: There is increasing evidence that passive smoking is associated with chronic respiratory diseases, but its association with chronic obstructive pulmonary disease (COPD) requires more study. In this cross-sectional analysis of data from 3 years of the Health Survey for England, the association between passive smoking exposure and risk of COPD is evaluated.

Design: Cross-sectional analysis of the 1995, 1996 and 2001 Health Surveys for England including participants of white ethnicity, aged 40+ years with valid lung function data. COPD was defined using the lower limit of normal spirometric criteria for airflow obstruction. Standardised questions elicited self-reported information on demography, smoking history, ethnicity, occupation, asthma and respiratory symptoms (dyspnoea, chronic cough, chronic phlegm, wheeze). Passive smoking was measured by self-report of hours of exposure to cigarette smoke per week.

Results: Increasing passive smoke exposure was independently associated with increased risk of COPD, with adjusted OR 1.05 (95% CI 0.93 to 1.18) for 1–19 h and OR 1.18 (95% CI 1.01 to 1.39) for 20 or more hours of exposure per week. Similar patterns (although attenuated and non-significant) were observed among never smokers. More marked dose–response relationships were observed between passive smoking exposure and respiratory symptoms, but the most marked effects were on the development of clinically significant COPD (airflow obstruction plus symptoms), where the risk among never smokers was doubled (OR 1.98 (95% CI 1.03 to 3.79)) if exposure exceeded 20 h/week.

Conclusion: This analysis adds weight to the evidence suggesting an association between passive smoking exposure and COPD.

ARTICLE SUMMARY

Article focus

- Passive exposure to cigarette smoke is established as an important independent risk factor for the development of chronic conditions such as heart disease and lung cancer.
- Although there is growing evidence implicating passive smoking in asthma and other respiratory diseases, the evidence for its effect on chronic obstructive pulmonary disease (COPD) is inconsistent.
- Using cross-sectional data from the annual Health Survey for England, we examined the association between self-reported exposure to passive smoking and COPD.

Key messages

- We have demonstrated a significant dose–response relationship between hours of exposure to passive smoking and increasing risk of COPD.
- The most marked effects were observed on the development of clinically significant COPD (airflow obstruction plus symptoms), where the risk among never smokers was doubled (OR 1.98 (95% CI 1.03 to 3.79)) if exposure exceeded 20 h/week.
- Passive smoking is prevalent worldwide, and even after the 2007 public smoking ban in the UK, 20% of the adult English population are still exposed to up to 20 h of passive smoking per week, with 5% exposed to more than 20 h/week; further measures are needed to investigate and reduce exposures in the home and elsewhere.

INTRODUCTION

Active smoking is accepted as the most important risk factor for the development of chronic obstructive pulmonary disease (COPD), and is responsible for over 70% of cases in high income countries, although far fewer (~40%) in lower income countries.[1] The remaining risk is attributed to a number of environmental factors, including occupational exposure to dust and fumes, and indoor and outdoor air pollution.[2]

Passive exposure to cigarette smoke is accepted as an independent risk factor for heart disease and lung cancer,[3 4] and has also been implicated in the aetiology of COPD,[2 5] although the association between passive

ARTICLE SUMMARY

Strengths and limitations of this study

- Our study has the advantage of being a large sample representative of the English population (>21 000 participants), conducted over 3 separate years, with a standardised protocol and objective measure of lung function.
- However, due to the cross-sectional nature of the design, temporal associations cannot necessarily be inferred.
- The Health Survey for England was not designed for the specific analyses presented in this paper, and thus some of the measures are crude.
- Self-reported passive smoke exposure is only a proxy for true exposure levels, but is accepted as the most practical method of assessment.

smoking and COPD is less well defined. There is increasing evidence that passive smoking is an important risk factor in chronic respiratory diseases. A number of studies suggest that asthma can be induced by exposure to passive smoking,[6] and there is strong evidence to suggest that passive smoking increases the risk of general respiratory symptoms.[6–11] Many previous studies have also examined the relationship between passive smoking and lung function, but with inconsistent results.[6] Evidence is now emerging that COPD may be independently associated with passive smoking exposure,[7 11–18] although a recent editorial[19] highlighted that more studies were required before a causal role could be established.

One difficulty with evaluating such an association is the number of subjects required to demonstrate the relatively small RRs associated with passive smoking risks (in the region of 1.2–1.5).[12 13] The Health Survey for England (HSE) has the advantage of size (over 15 000 adults are surveyed every year), generalisability and a standard protocol which is repeated annually. In 1995, 1996 and 2001, lung function was measured, providing the opportunity to examine the association between passive smoking and COPD in large numbers of people. The large size also allows the analysis to be restricted to never smokers, thus reducing any misclassification of reported exposure to passive smoking which could occur among smokers.

METHODS

Study design

Cross-sectional analysis of data collected by the annual HSE in 1995, 1996 and 2001, was carried out to establish the association between passive smoking and COPD.

Setting

The HSE is part of a set of annual surveys designed to monitor population health. The dataset is publicly available and obtained from the UK Data archive.[20] Briefly, an independent general population sample was surveyed each year obtained by multistage stratified random sampling of private households in England.[21–23] Postcode sectors were sorted by health authority and, within each health authority by the percentage of households where the head of the household had a non-manual occupation. A total of 720 postcode sectors were selected, with the probability of selection proportional to the number of delivery points (or addresses) in each sector. Nineteen addresses were selected from each sector. (Selecting sectors and addresses in this way ensures each household has an equal probability of selection.) Up to three households could be selected in any one postcode address. Home interviews and health assessments by trained interviewers and nurses were carried out for over 15 000 different adults each year. In 1995, 1996 and 2001, an assessment of lung function (FEV_1, FVC and PEF) was included, and in 1995 and 1996, information on respiratory health was additionally collected. Data from all 3 years were combined with adjustment for year of study in the analyses.

Participants

Participants of white ethnicity, aged 40 years and above and with valid lung function tests and height data were included in the analysis. Because only pre-bronchodilator spirometry was available, participants reporting a diagnosis of asthma were excluded from the main analyses but included in sensitivity analyses. Analyses were carried out among all participants and separately among never smokers.

Questionnaire and procedures

All consenting participants within the HSE were administered a detailed standardised computer-assisted interview used in previous survey years and seeking information on demographic characteristics, smoking history, ethnicity, occupation and educational level. Socioeconomic status was grouped into non-manual occupations and manual/other occupations. Participants were specifically asked if they had ever been diagnosed with asthma and whether they experienced a range of respiratory symptoms. Standard questions about wheeze, dyspnoea, chronic cough and chronic phlegm were included:

Wheeze: yes to either of:

Have you had wheezing or whistling in the chest in the last 12 months?

In the past 12 months, have you been woken by an attack of shortness of breath?

Dyspnoea: yes to any of:

Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?

Do you get short of breath walking with other people of your own age on level ground?

Do you have to stop for breath when walking at your own pace on level ground?

Chronic cough: yes to either of the first two and yes to 3 months of coughing

Do you usually cough first thing in the morning in winter?

Table 1 Characteristics of included and excluded participants

	Included participants				Excluded participants*
	1995	1996	2001	Total	
n	7071	7565	6468	21 104	6549
Males	3303 (46.7%)	3551 (46.9%)	2993 (46.3%)	9847 (46.7%)	2616 (40.0%)
Mean age, years (SD)	58.4 (12.3)	58.1 (12.3)	57.9 (11.9)	58.1 (12.2)	63.9 (14.3)
Age group					
40–49	2129 (30.1%)	2409 (31.8%)	1872 (28.9%)	6410 (30.4%)	1389 (21.2%)
50–59	1812 (25.6%)	1876 (24.8%)	1954 (30.2%)	5642 (26.7%)	1290 (19.7%)
60–69	1598 (22.6%)	1695 (22.4%)	1380 (21.3%)	4673 (22.1%)	1317 (20.1%)
70–79	1158 (16.4%)	1186 (15.7%)	969 (15.0%)	3313 (15.7%)	1418 (21.7%)
80+	374 (5.3%)	399 (5.3%)	293 (4.5%)	1066 (5.1%)	1135 (17.3%)
Socioeconomic status†					
Non-manual occupations	3928 (56.4%)	4195 (56.2%)	3683 (57.8%)	11 806 (56.7%)	3117 (49.6%)
Manual occupations	3028 (43.4%)	3254 (43.6%)	2676 (42.0%)	8958 (43.1%)	3158 (50.2%)
Other	14 (0.2%)	17 (0.2%)	13 (0.2%)	44 (0.2%)	13 (0.2%)
Smoking status†					
Current	1612 (22.8%)	1806 (23.9%)	1327 (20.5%)	4745 (22.5%)	1536 (23.5%)
Ex-regular	2569 (36.4%)	2637 (34.9%)	2290 (35.4%)	7496 (35.5%)	2237 (34.2%)
Never regular	2887 (40.9%)	3122 (41.3%)	2850 (44.1%)	8859 (42.0%)	2775 (42.4%)
Pack years smoked					
0	3107 (44.0%)	3359 (44.4%)	3038 (47.0%)	9504 (45.0%)	2973 (45.4%)
1–19	1817 (25.7%)	1922 (25.4%)	1656 (25.6%)	5395 (25.6%)	1517 (23.2%)
20–49	1695 (24.0%)	1803 (23.8%)	1389 (21.5%)	4887 (23.2%)	1552 (23.7%)
50+	449 (6.4%)	481 (6.4%)	384 (5.9%)	1314 (6.2%)	506 (7.7%)
Exposure to passive smoking, h					
Mean hours per week (SD)	9.2 (18.4)	9.3 (18.3)	6.1 (13.8)	8.3 (17.1)	7.0 (16.3)
Median hours per week (IQR)	2 (0–8)	2 (0–8)	1 (0–5)	1 (0–7)	0 (0–5)
0	2859 (40.5%)	3111 (41.2%)	3167 (49.1%)	9137 (43.4%)	3425 (52.6%)
1–9	2540 (36.0%)	2665 (35.3%)	2193 (34.0%)	7398 (35.1%)	1883 (28.9%)
10–19	667 (7.9%)	560 (7.4%)	440 (6.8%)	1557 (7.4%)	425 (6.5%)
20+	1105 (15.7%)	1224 (16.2%)	657 (10.2%)	2986 (14.2%)	778 (12.0%)
Any respiratory symptoms‡	3120 (44.1%)	3340 (44.2%)	–	6460 (44.1%)	1823 (47.7%)
Physician-diagnosed asthma	692 (9.8%)	794 (10.5%)	801 (12.4%)	2287 (10.8%)	702 (10.7%)
COPD§					
LLN	675 (10.6%)	593 (8.8%)	414 (7.3%)	1682 (8.9%)	–
GOLD	1190 (18.7%)	1086 (16.1%)	757 (13.4%)	3033 (16.1%)	–
NICE	526 (8.3%)	443 (6.6%)	333 (5.9%)	1302 (6.9%)	–

*Excluded due to invalid lung function or height measurements.
†Missing data: 557/27 653 (2.0%) had missing socioeconomic status and 5/27 653 (0.02%) missing smoking history.
‡Dyspnoea, wheeze, chronic cough or phlegm. Not recorded in 2001.
§Among those without asthma: GOLD criteria[26]: FEV_1/FVC ratio <0.7; NICE criteria: $FEV_1/FVC<0.7$ and $FEV_1<80\%$ predicted (equivalent to GOLD stage II)[27]; lower limit of normal (LLN) criteria[24 25]: participants with FEV_1/FVC values >1.645 SD below the mean reference value.
COPD, chronic obstructive pulmonary disease.

Do you usually cough during the day or at night in the winter?
Do you cough like this on most days for as much as 3 months each year?

Chronic phlegm: yes to either of the first two and yes to 3 months of phlegm
Do you usually bring up any phlegm from your chest, first thing in the morning in winter?
Do you usually bring up any phlegm from your chest, during the day or at night in the winter?
Do you bring up phlegm like this on most days for as much as 3 months each year?

Smoking habit was defined as current, ex- and never regular smokers (where regular was defined as at least one cigarette per day). Passive smoking exposure was measured by self-report of the number of hours currently exposed to cigarette smoke per week: "Now, in most weeks, how many hours a week are you exposed to other people's tobacco smoke?" As there was no lifetime indicator of chronic passive smoking exposure, this was assumed to be indicative of past adult exposure, aided by the data being collected before the 2007 public smoking ban in England.

Among other measurements, pulmonary function tests, without reversibility, were performed according to a standard protocol[18–20] with a Vitalograph (Maids Moreton, Buckinghamshire, UK) Escort spirometer (Fleisch pneumotachograph flow head) which was

Table 2 Association between passive smoking and chronic obstructive pulmonary disease* by year among participants in the Health Survey for England

	All participants				Never smokers
	1995 Adjusted† OR (95% CI)	1996 Adjusted† OR (95% CI)	2001 Adjusted† OR (95% CI)	Combined years Adjusted‡ OR (95% CI)	Combined years Adjusted‡ OR (95% CI)
n	6278	6669	5571	18518	7789
Age (years)					
40–49	1.00	1.00	1.00	1.00	1.0
50–59	1.57 (1.22 to 2.02)	1.25 (0.97 to 1.61)	1.39 (1.04 to 1.87)	1.40 (1.20 to 1.63)	0.99 (0.73 to 1.34)
60–69	2.34 (1.82 to 3.02)	1.75 (1.35 to 2.25)	1.74 (1.27 to 2.40)	1.95 (1.67 to 2.28)	1.19 (0.88 to 1.62)
70–79	3.43 (2.62 to 4.48)	2.52 (1.92 to 3.31)	2.43 (1.75 to 3.39)	2.80 (2.38 to 3.31)	2.09 (1.54 to 2.84)
80+	4.22 (2.93 to 6.07)	3.52 (2.43 to 5.10)	4.18 (2.69 to 6.50)	3.92 (3.13 to 4.90)	3.18 (2.20 to 4.61)
Sex					
Male	1.00	1.00	1.00	1.00	1.00
Female	0.79 (0.67 to 0.94)	0.90 (0.75 to 1.08)	0.97 (0.79 to 1.21)	0.87 (0.78 to 0.97)	0.97 (0.79 to 1.20)
Smoking status					
Never regular smoker	1.00	1.00	1.00	1.00	–
Ex-regular smoker	1.43 (1.15 to 1.78)	1.46 (1.15 to 1.85)	1.64 (1.25 to 2.14)	1.50 (1.30 to 1.72)	–
Current smoker	3.78 (3.03 to 4.72)	4.07 (3.23 to 5.13)	3.99 (3.02 to 5.25)	3.94 (3.43 to 4.52)	–
Socioeconomic status (occupation)					
Non-manual	1.0	1.0	1.0	1.0	1.0
Manual/other	1.17 (0.99 to 1.39)	1.23 (1.03 to 1.47)	1.29 (1.05 to 1.60)	1.22 (1.10 to 1.36)	1.03 (0.83 to 1.27)
Passive smoking exposure (h/week)					
0	1.00	1.00	1.00	1.00	1.00
1–19	0.99 (0.82 to 1.20)	1.13 (0.92 to 1.38)	1.03 (0.82 to 1.30)	1.05 (0.93 to 1.18)	1.14 (0.91 to 1.42)
20+	1.07 (0.83 to 1.39)	1.27 (0.97 to 1.65)	1.28 (0.91 to 1.81)	1.18 (1.01 to 1.39)	1.17 (0.78 to 1.76)
Year					
1995	–	–	–	1.0	1.0
1996	–	–	–	0.79 (0.70 to 0.89)	0.75 (0.59 to 0.95)
2001	–	–	–	0.70 (0.62 to 0.80)	0.66 (0.51 to 0.85)

*Lower limit of normal (LLN) criteria[24 25]: participants with FEV_1/FVC values >1.645 SD below the mean reference value.
†Adjusted for age, sex, smoking status, socioeconomic status and passive smoking exposure.
‡Adjusted for age, sex, smoking status, socioeconomic status, year of study and passive smoking exposure.

calibrated daily at normal room temperature. The best FEV_1 and FVC measurements were used.

Outcomes

The main outcome measure was the presence of COPD, defined by using the reference equations from the European Community for Steel and Coal Study[24] and the lower limit of normal (LLN) criterion.[25] In this way, participants were classified as having obstructive airways disease if their pre-bronchodilator FEV_1/FVC values were below the lowest 5% of the frequency distribution of values found in the healthy reference population. Secondary outcome measures were the presence of any chronic respiratory symptoms (wheeze, dyspnoea, chronic cough, chronic phlegm, as detailed above) and the presence of clinically significant COPD (both airflow obstruction (LLN) and any of the above respiratory symptoms). Analyses were repeated using both pre-bronchodilator modified GOLD[26] (FEV_1/FVC ratio<0.7) and NICE criteria[27] (FEV_1/FVC<0.7 and FEV_1<80% predicted, equivalent to GOLD stage II). Because lung function data were pre-bronchodilator, some patients with airflow obstruction could have asthma rather than COPD. The analyses were initially undertaken excluding participants with asthma and then repeated including participants with asthma.

Statistical analysis

Univariate and multivariate logistic regression were undertaken in STATA V.10.0, in the latter adjusting for age, sex, smoking history, year of study, socioeconomic status and self-reported asthma where appropriate and adding significantly to the statistical model.

RESULTS

Study participants

Of 27 653 white adults aged 40 years and older, 6549 (23.7%) were excluded because they did not have valid lung function data or a reliable height measurement.

Table 1 describes the baseline characteristics of the remaining included participants, by year and compared with the excluded participants. The mean age of the participants was 58.1 years (SD 12.2) and 9847 (46.7%) were male. Age and sex distributions were similar for each year. Overall, 4745 (22.5%) were current smokers and 8859 (42.0%) had never smoked regularly; there were fewer current and more never smokers in 2001 compared with previous years. Most smokers had smoked less than 50 pack-years.

Exposure to passive smoking declined over time, with 2859 (40.5%) experiencing no exposure in 1995, which increased to 3167 (49.1%) by 2001. Numbers exposed in the highest exposure group (20 or more hours per week) dropped from 1105 (15.7%) in 1995 to 657 (10.2%) in 2001.

COPD prevalence among participants without asthma (LLN criteria) also decreased from 10.6% in 1995 to 7.3% in 2001. COPD measured by modified GOLD or NICE criteria showed similar patterns, although absolute prevalence values differed by definition.

Excluded participants (table 1) were more likely to be older, female, have manual occupations and greater respiratory symptoms but lower current exposure to passive smoking.

Table 3 Association between passive smoking and respiratory symptoms and clinically significant chronic obstructive pulmonary disease among never-smoking participants of the Health Survey for England, 1995/1996

	Respiratory symptoms* Adjusted OR‡ (95% CI)	Clinically significant COPD† Adjusted OR‡ (95% CI)
n	5441	5441
Age (years)		
40–49	1.00	1.00
50–59	1.37 (1.16 to 1.62)	1.46 (0.74 to 2.87)
60–69	2.03 (1.72 to 2.40)	2.73 (1.46 to 5.10)
70–79	3.06 (2.54 to 3.68)	6.33 (3.48 to 11.54)
80+	4.22 (3.29 to 5.40)	11.08 (5.76 to 21.30)
Sex		
Male	1.00	1.00
Female	1.32 (1.17 to 1.50)	0.99 (0.67 to 1.44)
Year		
1995	1.00	1.00
1996	0.97 (0.87 to 1.09)	0.61 (0.42 to 0.87)
Passive smoking exposure (h/week)		
0	1.00	1.00
1–19	1.20 (1.06 to 1.37)	1.52 (1.04 to 2.23)
20+	1.68 (1.36 to 2.08)	1.98 (1.03 to 3.79)

*Respiratory symptoms: any of dyspnoea, wheeze, chronic cough or phlegm.
†Lower limit of normal (LLN) criteria[24 25]: participants with FEV_1/FVC values >1.645 SD below the mean reference value plus respiratory symptoms.
‡Adjusted for age, sex, passive smoking exposure and year of study.
COPD, chronic obstructive pulmonary disease.

Association between passive smoking and COPD

For each of the 3 years, risk of COPD increased with increasing age and was highest among current smokers compared with never regular smokers, and manual workers compared with non-manual workers (table 2). Females had a lower risk (OR 0.87 (95% CI 0.78 to 0.97) for combined data). Exposure to passive smoking was associated with increased risk of COPD, although for individual years this effect was not statistically significant. For the combined data, increasing exposure to passive cigarette smoke was independently associated with increased risk of COPD, with OR 1.05 (95% CI 0.93 to 1.18) for up to 20 h and OR 1.18 (1.01 to 1.39) for 20 or more hours of exposure per week once year of study and socioeconomic status were taken into account. Restricting the analyses to never regular smokers resulted in similar patterns, but with a lower sample size (n=7789) the risk estimates became non-significant (OR

1.14 (0.91 to 1.42) and 1.17 (0.78 to 1.76) for 1–19 and ≥20 h of exposure, respectively).

Effect of passive smoking on respiratory symptoms

The effect of passive smoking exposure on respiratory symptoms is shown in table 3 (data from 1995 and 1996). Never-smoking participants with increasing exposure to passive smoking were at increased risk of reporting respiratory symptoms, with OR 1.20 (1.06 to 1.37) for exposure levels of 1–19 h/week and OR 1.68 (1.36 to 2.08) for ≥20 h of exposure. Notably, there was a significantly increased risk among females compared with males of reporting any respiratory symptoms (OR 1.32 (1.17 to 1.50)).

Furthermore, the risk of clinically significant COPD (ie, having both respiratory symptoms and evidence of airflow obstruction by spirometry) among never-smoking participants exposed to passive smoking was higher than the risk of symptoms alone (OR 1.52 (1.04 to 2.23) for 1–19 h and 1.98 (1.03 to 3.79) for ≥20 h/week of exposure).

Sensitivity analyses

Repeating the analyses with alternative spirometric criteria for COPD showed similar patterns (table 4). However, males and those who were older showed increased independent risks of COPD with both these definitions. Inclusion of participants reporting asthma (table 5) led to similar patterns of the effect of passive smoking on COPD and respiratory symptoms, although sometimes loss of significance for the two outcomes which included airflow obstruction.

DISCUSSION

In this population-based analysis of over 21 000 subjects, an independent dose–response relationship between exposure to passive smoking and chronic respiratory disease was observed. Results were not always statistically significant, but the effect was consistently present in each year of the survey, with different spirometric definitions of COPD, with and without respiratory symptoms, with respiratory symptoms alone, and among never smokers as well as the total population. The effect of passive smoking exposure was most marked among never smokers having clinically significant COPD, where never smokers exposed to between 1 and 19 h of passive smoking had a 52% excess risk and those exposed to ≥20 h had an excess risk of 98%.

Passive smoking was associated with higher risks of reporting respiratory symptoms (OR 1.68 (1.36 to 2.08)) among never smokers, which is consistent with other European studies,[7 8 11] although not with a large study in China.[12] However, it has been shown that symptoms are consistently less likely to be reported in South-East Asia than in Europe.[28] More women also reported respiratory symptoms, which is similar to findings elsewhere.[7]

It is difficult to compare the results for COPD with other studies as definitions for COPD differ, as do

Table 4 Association between passive smoking and chronic obstructive pulmonary disease among never-smoking participants of the Health Survey for England: effect of alternative spirometric definitions

	GOLD criteria*		NICE criteria*	
	COPD Adjusted OR (95% CI)‡	Clinically significant COPD† Adjusted OR (95% CI)‡	COPD Adjusted OR (95% CI)‡	Clinically significant COPD† Adjusted OR (95% CI)‡
n	7944	5443	7944	5443
Age (years)				
40–49	1.00	1.00	1.00	1.00
50–59	1.69 (1.32 to 2.16)	2.17 (1.27 to 3.71)	1.85 (1.00 to 3.44)	3.92 (1.06 to 14.53)
60–69	2.75 (2.17 to 3.49)	4.14 (2.50 to 6.85)	5.13 (2.96 to 8.91)	11.26 (3.33 to 38.05)
70–79	4.48 (3.51 to 5.72)	8.87 (5.40 to 14.58)	10.95 (6.35 to 18.86)	29.68 (8.96 to 98.30)
80+	9.00 (6.80 to 11.92)	21.81 (12.91 to 36.83)	18.42 (10.28 to 33.03)	60.77 (17.83 to 207.18)
Sex				
Male	1.00	1.00	1.00	1.00
Female	0.56 (0.48 to 0.66)	0.62 (0.48 to 0.82)	0.53 (0.40 to 0.70)	0.45 (0.29 to 0.69)
Year				
1995	1.00	1.00	1.00	1.00
1996	0.76 (0.64 to 0.90)	0.68 (0.52 to 0.88)	0.61 (0.44 to 0.84)	0.57 (0.37 to 0.88)
2001	0.67 (0.55 to 0.80)	–	0.61 (0.43 to 0.86)	–
Passive smoking exposure (h/week)				
0	1.00	1.00	1.00	1.00
1–19	1.11 (0.94 to 1.31)	1.31 (0.98 to 1.74)	1.10 (0.81 to 1.49)	1.09 (0.68 to 1.74)
20+	1.10 (0.81 to 1.49)	1.82 (1.12 to 2.97)	1.33 (0.74 to 2.38)	1.92 (0.88 to 4.23)

*GOLD criteria[26]: FEV_1/FVC ratio <0.7; NICE criteria: $FEV_1/FVC<0.7$ and $FEV_1<80\%$ predicted (equivalent to GOLD stage II).[27]
†Participants with airflow obstruction plus respiratory symptoms (any of dyspnoea, wheeze, chronic cough or phlegm).
‡Adjusted for age, sex, passive smoking exposure and year of study.

Table 5 Association between passive smoking, COPD, respiratory symptoms and clinically significant COPD among never-smoking participants of the Health Survey for England (data including participants reporting presence of asthma)

	COPD* Adjusted OR (95% CI)§	Respiratory symptoms† Adjusted OR (95% CI)§	Clinically significant COPD‡ Adjusted OR (95% CI)§
n	8849	6004	6004
Age (years)			
40–49	1.00	1.00	1.00
50–59	1.06 (0.82 to 1.35)	1.33 (1.14 to 1.56)	1.17 (0.75 to 1.82)
60–69	1.30 (1.02 to 1.67)	2.04 (1.74 to 2.39)	2.13 (1.41 to 3.22)
70–79	1.88 (1.45 to 2.46)	2.93 (2.45 to 3.50)	3.59 (2.35 to 5.49)
80+	2.98 (2.16 to 4.13)	3.90 (3.06 to 4.96)	5.54 (3.31 to 9.28)
Sex			
Male	1.00	1.00	1.00
Female	0.93 (0.78 to 1.12)	1.38 (1.22 to 1.56)	0.96 (0.71 to 1.28)
Asthma			
No	1.00	1.00	1.00
Yes	4.51 (3.70 to 5.50)	7.17 (5.85 to 8.80)	10.20 (7.65 to 13.59)
Year			
1995	1.00	1.00	1.00
1996	0.81 (0.66 to 0.98)	0.99 (0.88 to 1.10)	0.71 (0.54 to 0.94)
2001	0.64 (0.52 to 0.80)	–	–
Passive smoking exposure (h/week)			
0	1.00	1.00	1.00
1–19	1.16 (0.97 to 1.40)	1.19 (1.05 to 1.34)	1.19 (0.88 to 1.61)
20+	1.25 (0.90 to 1.72)	1.70 (1.39 to 2.08)	1.47 (0.89 to 2.43)

*Lower limit of normal (LLN) criteria[24 25]: participants with FEV_1/FVC values >1.645 SD below the mean reference value.
†Respiratory symptoms: any of dyspnoea, wheeze, chronic cough or phlegm.
‡Lower limit of normal (LLN) criteria[24 25]: participants with FEV_1/FVC values >1.645 SD below the mean reference value plus respiratory symptoms.
§Adjusted for age, sex, passive smoking exposure, year of study and history of asthma.
COPD, chronic obstructive pulmonary disease.

passive smoking exposure measures. However, previous studies[7 12–14 16–18] have indicated significant increased risk of COPD with increasing passive smoking exposure, with ORs between 1.31 and 2.24, depending on the definitions used and the study design. In China,[12] being exposed to ≥40 h of passive smoking per week for at least 5 years was associated with risks of spirometrically defined COPD among never smokers of 1.48 (1.18 to 1.85). Similarly, in Estonia, for those with more than 5 h of passive smoking exposure per day outside the home (ie, more than 35 h/week), the risk of physician-diagnosed chronic bronchitis or emphysema was 1.54 (1.13 to 3.00).[7] For those with 1–5 h/day (7–35 h/week), the risk was lower (OR 1.16 (0.88 to 1.53)). Passive smoking exposure is lower in England, and likely to have reduced further, subsequent to the collection of our data, since smoking in public places was banned in 2007. Nevertheless, we found that among never smokers with ≥20 h of exposure, there was a risk of 1.17 (0.78 to 1.76) for spirometrically defined COPD, which was between one-third and one-half of the excess risk observed in China or Estonia for approximately half of the reported exposure.

However, risks were highest for never smokers with clinically significant COPD (OR 1.98 (1.03 to 3.79) for ≥20 h of exposure). The greater risks are likely to reflect tighter disease definitions (and more severe disease) and less disease and exposure misclassification. The effects of passive smoking exposure seen with alternative definitions of COPD are similar, although lack of statistical significance and more extreme effects of age and sex are likely to reflect less accurate disease definitions. In particular, our results highlight the known age and sex bias inherent in the GOLD and NICE criteria for COPD.[29]

Nevertheless, our calculated risks all fall within the range reported in previous studies, and indicate that our analysis reflects, and adds to, the weight of previous evidence. Our results also accord with published excess risks of passive smoking for heart disease, lung cancer and asthma.[3 4 6]

Strengths and limitations

Our study has the advantage of being a large sample representative of the English population, conducted over 3 separate years, with a standardised protocol and objective measure of lung function. There were over 21 000 participants, including 8859 never smokers. In addition, we initially excluded those with physician-diagnosed asthma, as some studies have suggested that the effect of passive smoking on lung function is confined to those with increased susceptibility such as asthma,[30–32] and in order to reduce misclassification of COPD. Our sensitivity analyses demonstrated that the effects show a consistent pattern among those with and without asthma.

However, due to the cross-sectional nature of the design, temporal associations cannot be inferred and it is

possible that participants with either respiratory symptoms, poorer lung function or a respiratory diagnosis may have modified their exposure to passive smoking. Nonetheless, this would be most likely to manifest in a reduction of their exposure and thus have the effect of underestimating any positive associations between passive smoking exposure and COPD. Additionally, the HSE was not designed for the specific analyses presented in this paper, and thus some of the measures are crude. We used self-reported current passive exposure status, assuming that this would reflect past exposure, although this may have changed over time. In general, passive smoking exposure reduced between 1995 and 2001, and if past exposures were greater, then our analysis may have overestimated the effects at a given exposure level. Also, self-reported exposure is only a proxy for true exposure levels but is accepted as the most practical method of assessment. In addition, the spirometry, while undertaken to a standardised protocol, may not have reached currently recommended quality criteria[25] and could have misclassified some participants.

Recall bias of passive smoking exposure would not be likely as this is a large survey with many questions and no particular hypothesis associated with respiratory disease.

It is also possible that the observed effects of passive smoking in our analysis may be partly attributable to residual confounding, as any effects of indoor air pollution or occupational exposure could not be accounted for.

In summary, our study, although cross-sectional, lends weight to the argument that passive smoking is an important modifiable risk factor for COPD, and is consistent with other published evidence. In the UK (and many other western countries), passive smoking outside the home has been reducing, although, even after the public smoking ban, 20% of the adult English population are still exposed to up to 20 h of passive smoking per week, and 5% to more than 20 h/week (unpublished analysis of HSE 2007).[33] Further investigation is required to determine whether subgroups of the population are experiencing greater exposure in the home since the smoking ban, and what strategies might be effective in reducing their exposure. However, in developing countries, passive smoking is still a major problem in the workplace and policy makers should strive to reduce this.

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