Lung function and exposure to workplace second-hand smoke during exemptions from smoking ban legislation: an exposure–response relationship based on indoor $PM_{2.5}$ and urinary cotinine levels

Hak-Kan Lai,[1] Anthony J Hedley,[1] James Repace,[2,3] Ching So,[1] Qiu-Ying Lu,[1] Sarah M McGhee,[1] Richard Fielding,[1] Chit-Ming Wong[1]

[1]School of Public Health, The University of Hong Kong, Hong Kong SAR, China
[2]Department of Public Health & Community Medicine, Tufts University School of Medicine, Boston, Massachusetts, USA
[3]Repace Associates Inc, 101 Felicia Lane, Bowie, Maryland, USA

Correspondence to
Anthony J Hedley, The University of Hong Kong, 5/F William MW Mong Block, 21 Sassoon Road, Pokfulam, Hong Kong, China;
commed@hkucc.hku.hk

Received 8 February 2011
Accepted 24 March 2011
Published Online First 6 May 2011

ABSTRACT

Background The effects of workplace second-hand smoke (SHS) on lung function remain uncertain because of a lack of objective measures for SHS exposures.

Objective To determine whether an exposure–response association exists between lung function and two different markers of SHS based on indoor fine particulate ($PM_{2.5}$) and urinary cotinine levels in non-smoking catering workers.

Design A cross-sectional study during a 1.5-year exemption of licensed catering premises from smoke-free legislation.

Participants 186 non-smoking catering workers aged 18–65 years in Hong Kong were recruited. A declared non-smoking status was accepted in workers with exhaled breath carbon monoxide levels <6 ppm and urinary cotinine levels <100 ng/ml.

Main outcome measures Lung function measures of forced expiratory volume in 1s (FEV_1 in litres), forced vital capacity (FVC in litres) and forced expiratory flow as 25–75% of FVC (FEF_{25-75} in l/s) were recorded.

Results Indoor fine particulate ($PM_{2.5}$) concentrations were 4.4 times as high in smoking premises (267.9 $\mu g/m^3$) than in non-smoking premises (60.3 $\mu g/m^3$) and were strongly associated with the probability of permitted smoking (R^2=0.99). Smoking was the dominant source of particulates (R^2=0.66). Compared with workers exposed to the lowest indoor $PM_{2.5}$ stratum (<25 $\mu g/m^3$), lung function was lower in the three higher $PM_{2.5}$ strata (25–75, 75–175, >175 $\mu g/m^3$) with FEV_1 −0.072 (95% CI −0.123 to −0.021), −0.078 (95% CI −0.132 to −0.024), −0.101 (95% CI −0.187 to −0.014); FEF_{25-75} −0.368 (95% CI −0.660 to −0.077), −0.489 (95% CI −0.799 to −0.179), −0.597 (95% CI −0.943 to −0.251); and FEV_1/FVC (%) −2.9 (95% CI −4.8 to −1.0), −3.2 (95% CI −5.1 to −1.4) and −4.4 (95% CI −7.4 to −1.3), respectively. Urinary cotinine was associated positively with indoor $PM_{2.5}$ but negatively with lung function. Consistently lower values for lung function per unit increase of indoor $PM_{2.5}$ were found.

Conclusion Lung function is inversely associated with workplace SHS. Workplace exemptions and delays in implementing smoke-free policies and current moves to relax legislation are a major threat to the health of workers.

Key messages

What is the key question?
- Does second-hand smoke exposure cause measurable changes in adult lung function?

What is the bottom line?
- Incomplete or delayed workplace smoking bans damage respiratory health.

Why read on?
- This report provides the first complete analysis on the association between $PM_{2.5}$, urinary cotinine and lung function.

INTRODUCTION

Exposure to second-hand smoke (SHS) in catering workers is among the highest of all occupations,[1] with increased risks for respiratory and cardiovascular diseases.[2] A recent Cochrane review of 49 studies of smoking bans in 13 countries from 1988 to 2007 concluded that a reduction of SHS in indoor workplaces, especially among bar workers, is effective in reducing adverse health outcomes.[2]

With increasing evidence showing the benefits of smoking bans, the Hong Kong Special Administrative Region amended its Smoking (Public Health) Ordinance to ban smoking in indoor public and workplace areas on 1 January 2007 with exemptions until 1 July 2009 for many indoor areas,[3] postponing public and workers' health protection by 2.5 years.

Cigarette smoke particulates cause damage to the arterial endothelium[4] and pulmonary epithelium.[5] The 2006 US Surgeon General's Report[6] concluded that the evidence is sufficient to infer that exposure to SHS causes lung cancer and coronary heart disease morbidity and mortality. However, causality for effects on lung function in healthy non-smoking adults has been described as less clear,[2 6] inconsistent[7 8] and unlikely to be clinically significant.[8] While the gap in evidence is acknowledged, many jurisdictions allow exemptions or partial bans[2] and vested interests lobby for repeal of existing legislation.

A PubMed search for the period from January 1960 to September 2010 found several relevant reports on lung function changes in workers before and after smoke-free legislation,[2 9–11] but none showed a clear exposure–response relationship. In this study we examined the associations between lung function in non-smoking catering workers and

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

their exposures to workplace SHS using the biomarker cotinine and $PM_{2.5}$, a marker of tobacco aerosols with high penetration of airways[12] and a strong association with mortality.[13 14]

METHODS

Recruitment and selection

From February to June 2008 we surveyed 495 randomly selected catering premises, including Chinese, western, Hong Kong style (cha-chaan-teng) and fast-food restaurants. The sampling frame was a list of licensed premises in 19 districts, from the Food and Environmental Hygiene Department, and we randomly selected seven for the survey. We recruited a total of 222 workers from 99 premises after excluding premises with undefined indoor areas (figure 1). Unrestricted indoor smoking was allowed in 36 of these 99 venues and, in 63 venues, indoor smoking was prohibited by law including 12 premises with alfresco seating. Participants in the study were either never-smokers (85%) or ex-smokers who had quit for at least 3 months (15%). The purpose of the study was explained to the owner or manager and the benefits of participation were explained to the workers, including reports on lung function tests and urine cotinine levels and payment of HK$100 (US$12.8) as compensation for their time.

Assessment

All recruited workers were interviewed using a standard schedule to collect demographic and socioeconomic data, smoking history, SHS exposure and work patterns. Forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1) and forced expiratory flow as 25–75% of FVC (FEF_{25-75}) were measured using a portable spirometer (MicroLoop, Micro-Medical, Basingstoke, UK) following an American Thoracic Society/European Respiratory Society protocol. All pulmonary function tests were conducted by the same trained researcher and a demonstration video was shown to all subjects before the tests. We accepted declared non-smoking status in workers with exhaled breath carbon monoxide levels <6 ppm[15] (Micro 4 Smokerlyzer, Bedfont Scientific, Maidstone, Kent, UK) and urinary cotinine levels <100 ng/ml[16] (liquid-chromatography-tandem-mass-spectrometry, UCSF Clinical Pharmacology Laboratory, USA). The final data analysis included 186 of the 222 recruited workers (table 1) after exclusions for inadequate spirometry readings and failure to meet the criteria for non-smoking status (figure 1).

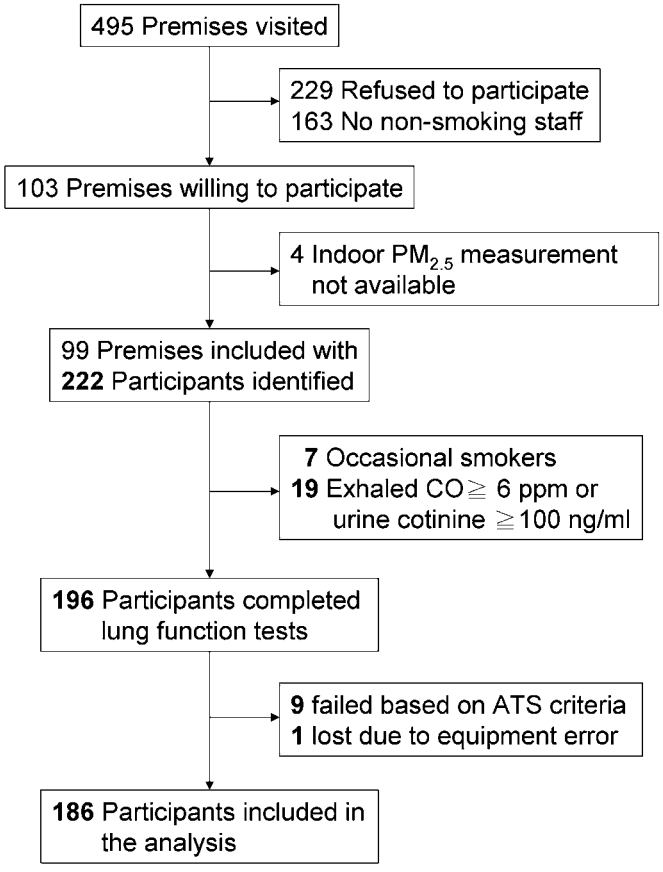


Figure 1 Participant recruitment, flow and dropouts. Sample sizes of the participants are shown in bold.

Measurement of $PM_{2.5}$

Two portable SidePak particulate monitors based on laser photometry (SidePak Aerosol Monitor AM510, USA) were used to measure $PM_{2.5}$ concentrations indoors and outdoors simultaneously for approximately 30 min at each location. For indoor measurements, the monitor was placed at least 1.1 m above the floor and away from vertical surfaces, ventilation units, direct sunlight, hallways or passageways, elevators and outside doors. For outdoor measurements, the monitor was carried while constantly walking around the catering premises. The relative humidity (RH), temperature (Oregon Scientific Wireless Weather Station, Tualatin, Oregon, USA), dimensions of the room (Leica Disto laser meter A2, Switzerland), number of smokers, burning cigarettes, other burning objects, ventilation type, cooking activities and the separation of the kitchen from the rest of the premises were recorded.

Table 1 Characteristics of recruited workers

	In non-smoking premises (n=140)	In smoking premises (n=46)	p Value
Male, n (%)	42 (30)	20 (43)	0.092
Age, mean (SD), years	39.1 (12.6)	30.5 (10.1)	<0.001
Height, mean (SD), cm	160.7 (7.0)	164.2 (9.3)	0.008
SHS exposure			
At work, mean (SD), years	6.84 (7.61)	6.71 (7.36)	0.999
At home, mean (SD), h/day	0.13 (0.31)	0.16 (0.62)	0.542
In other places, mean (SD), h/day	1.29 (2.43)	2.77 (4.63)	0.014
Education level, n (%)			
Primary or below	16 (11)	1 (2)	<0.001
Secondary	106 (76)	29 (63)	
Above secondary	18 (13)	16 (35)	
Monthly income, n (%) (US$1=HK$7.8)			
<HK$7000	47 (34)	7 (15)	0.008
HK$7001–10 000	57 (41)	19 (41)	
HK$10 001–20 000	32 (23)	18 (39)	
≥HK$20 001	4 (3)	2 (4)	
Current job position, n (%)			
Serving	58 (41)	13 (28)	0.013
Cleaning	9 (6)	0 (0)	
Cashier	9 (6)	0 (0)	
Kitchen	18 (13)	7 (15)	
Reception	4 (3)	1 (2)	
Bar	5 (4)	6 (13)	
Management	30 (21)	18 (39)	
Others	7 (5)	1 (2)	

Student t test was used for age and height, Savage two-sample test for SHS exposure duration in different places, Pearson χ^2 test for gender and job position, Mantel–Haenszel χ^2 test for education and income.
SHS, second-hand smoke.

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

All indoor and outdoor SidePak readings were adjusted for RH to account for aerosol hygroscopic growth.[17] For calibration, we placed one SidePak at a Hong Kong government Environmental Protection Department particulate monitor (http://www.epd-asg.gov.hk/english/backgd/monitor.html) for 2 weeks in April and confirmed a high correlation between the SidePak readings adjusted for RH and government monitor concentrations (R=0.91). The calibration factor of the other SidePak was adjusted to match the one tested. Before each sampling day, each monitor was cross-calibrated for 30 min in the laboratory to rectify any systematic differences.

Statistical analysis

Distribution plots were constructed for all data to assess normality, logarithmically-transformed indoor $PM_{2.5}$ data and square root-transformed outdoor $PM_{2.5}$ data, and descriptive analyses were tabulated for smoking and non-smoking premises. We examined whether there were differences in pulmonary function between ex-smokers and never-smokers among the recruited non-smokers.

Regression-based determinant analysis was used to examine whether smoking was the major determinant of indoor $PM_{2.5}$ concentration compared with a number of a priori selected determinants including outdoor $PM_{2.5}$ concentration, indoor volume of premises, type of kitchen and ventilation, and the presence of burning objects other than cigarettes.

To determine the relationship between indoor $PM_{2.5}$ concentrations and the probability of permitted indoor smoking, we divided the 99 premises into categories by unit increment of natural log indoor $PM_{2.5}$ concentration and calculated the probability of permitted smoking by using the number of smoking premises divided by the total sum of smoking and non-smoking premises in each category. We used a non-linear regression to achieve a better fit to the data.

Multiple regression based on generalised estimating equations was used to analyse the associations between the lung function variables FEV_1 (l), FEF_{25-75} (l/s), FEV_1/FVC (%) and FVC (l) and indoor $PM_{2.5}$ exposure levels with adjustment for clustering effects of premises and for possible modifying factors (table 2). Analyses of FEV_1 and FEF_{25-75} were additionally adjusted for FVC.[18] To satisfy the normality assumption for the regression model, we divided the indoor $PM_{2.5}$ distribution into four strata based on a natural log scale with $PM_{2.5}$ $\leq 25\ \mu g/m^3$ as the referent stratum. Regression was used to test the linear trend between lung function and $PM_{2.5}$ strata, and sensitivity analyses were performed for the less exposed subgroups (never-smokers, those not exposed to co-workers' SHS and workers in non-smoking premises) and the older subgroup. We analysed the regression coefficients for continuous variables of lung function and natural log indoor $PM_{2.5}$ concentration and then predicted lung function using the average indoor $PM_{2.5}$ levels in smoking and non-smoking premises.

Table 2 Lung function indices (with 95% CI) of non-smoking catering workers and indoor workplace $PM_{2.5}$ concentrations expressed with the associated probability of permitted indoor smoking

$PM_{2.5}$ ($\mu g/m^3$)	n_0	n_1	FEV_1 (l)	FEF_{25-75} (l/s)	FEV_1/FVC (%)
All workers (n=186)					
≤25	30	0	0	0	0
25–75	52	3	−0.072 (−0.123 to −0.021)**	−0.368 (−0.660 to −0.077)*	−0.029 (−0.048 to −0.010)**
75–175	44	19	−0.078 (−0.132 to −0.024)**	−0.489 (−0.799 to −0.179)**	−0.032 (−0.051 to −0.014)***
>175	14	24	−0.101 (−0.187 to −0.014)*	−0.597 (−0.943 to −0.251)**	−0.044 (−0.074 to −0.013)**
			p for trend=0.025	p for trend=0.003	p for trend=0.007
Workers who never smoked (n=156)					
≤25	25	0	0	0	0
25–75	41	3	−0.092 (−0.150 to −0.035)**	−0.414 (−0.714 to −0.113)**	−0.035 (−0.058 to −0.012)**
75–175	40	15	−0.093 (−0.153 to −0.034)**	−0.482 (−0.814 to −0.150)**	−0.037 (−0.057 to −0.017)***
>175	14	18	−0.106 (−0.218 to 0.006)	−0.563 (−0.990 to −0.136)**	−0.043 (−0.082 to −0.005)*
			p for trend=0.030	p for trend=0.006	p for trend=0.014
Workers aged ≥30 (n=113)					
≤25	26	0	0	0	0
25–75	37	2	−0.082 (−0.146 to −0.018)*	−0.548 (−0.807 to −0.289)***	−0.032 (−0.054 to −0.009)**
75–175	27	8	−0.070 (−0.137 to −0.004)*	−0.629 (−0.956 to −0.302)***	−0.030 (−0.051 to −0.008)**
>175	6	7	−0.220 (−0.365 to −0.074)**	−1.242 (−1.773 to −0.710)***	−0.097 (−0.150 to −0.044)***
			p for trend<0.001	p for trend<0.001	p for trend<0.001
Workers in non-smoking premises (n=140)					
≤25	30	0	0	0	0
25–75	52	0	−0.088 (−0.140 to −0.035)***	−0.460 (−0.745 to −0.175)**	−0.040 (−0.061 to −0.019)***
75–175	44	0	−0.097 (−0.160 to −0.033)**	−0.544 (−0.928 to −0.161)**	−0.040 (−0.062 to −0.019)***
>175	14	0	−0.183 (−0.320 to −0.046)**	−0.888 (−1.381 to −0.395)***	−0.081 (−0.128 to −0.034)***
			p for trend=0.029	p for trend=0.011	p for trend=0.008
Workers not exposed to co-workers' SHS (n=72)					
≤25	15	0	0	0	0
25–75	23	0	−0.099 (−0.183 to −0.015)*	−0.392 (−0.755 to −0.028)*	−0.045 (−0.076 to −0.013)**
75–175	22	4	−0.144 (−0.217 to −0.070)***	−0.705 (−1.039 to −0.371)***	−0.064 (−0.086 to −0.043)***
>175	8	0	−0.231 (−0.400 to −0.061)**	−1.186 (−1.801 to −0.570)***	−0.100 (−0.155 to −0.045)***
			p for trend<0.001	p for trend<0.001	p for trend<0.001

*p<0.05; **p<0.01; ***p<0.001 (F-test).
Estimates were adjusted for gender, age, height, duration of SHS exposure at work, daily duration of SHS exposure at home and in other places, education, income, job position, the effect of patio seating and FVC where appropriate.
FEV_1, forced expiratory volume in 1 s; FEF_{25-75}, forced expiratory flow as 25–75% of FVC; FVC, forced vital capacity; n_0, number of workers from non-smoking premises; n_1, number of workers from smoking premises; SHS, second-hand smoke.

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

The same approach was used to examine associations between workers' lung function and urinary cotinine levels. To satisfy the assumption of normality for regression, we divided the cotinine values into four strata based on the unit intervals of a natural log-log scale with a reference cotinine stratum of ≤0.8 ng/ml[3], a reported geometric mean for non-smokers in a non-occupational setting.[19] We tested the linear trend between lung function and cotinine strata.

A partial residual plotting method[20] was used to quantify the associations between levels of indoor $PM_{2.5}$ and urinary cotinine, a biomarker of SHS exposure,[2] taking into account possible modifying factors (table 3).

SPSS Version 16 and SAS Version 9.1.3 were used for analyses with statistical significance defined at an α level of 0.05 (two-tailed) and estimates are presented with 95% CIs.

RESULTS

Recruited catering workers

There were more female (67%) than male workers in both smoking and non-smoking premises. Among the 186 non-smokers, lung function indices in ex-smokers adjusted for age, sex and height (FEV_1 2.59 l, FEF_{25-75} 3.30 l/s, FEV_1/FVC 92%), were not statistically different (p=0.40–0.46) from never-smokers (FEV_1 2.65 l, FEF_{25-75} 3.44 l/s, FEV_1/FVC 91%). Compared with workers in non-smoking premises, those in smoking premises were younger (p<0.001), taller (p=0.008), better educated (p<0.001), with more income (p=0.008) and more exposure to SHS in other places (p=0.014) (table 1).

Indoor and outdoor $PM_{2.5}$ concentrations

The indoor $PM_{2.5}$ concentrations ranged from 2.6 to 847.2 μg/m[3]. In smoking premises the indoor $PM_{2.5}$ geometric mean (GM) (211.6 μg/m[3] (95% CI 161.1 to 277.8)) was 3.5 times as high as that in non-smoking premises (60.3 μg/m[3] (95% CI 48.0 to 75.8)), but with no differences (p=0.109) in their corresponding outdoor $PM_{2.5}$ concentrations (figure 2A). In smoking premises with active smoking during our visits, the $PM_{2.5}$ GM (267.9 μg/m[3] (95% CI 199.4 to 359.9)) was 4.4 times as high (p<0.001) as the GM (60.3 μg/m[3] (95% CI 47.8 to 76.1)) in non-smoking premises without active smoking (figure 2B).

Association between indoor $PM_{2.5}$ and indoor smoking

The multiple regression model indicated that, in terms of variation explained, the most important determinant of indoor $PM_{2.5}$ was smoking (57.2%, p<0.0001), followed by ventilation type (10.1%, p=0.014), outdoor $PM_{2.5}$ (7.0%, p<0.0001), presence of burning objects (2.1%, p=0.026), indoor volume of the premises (2.0%, p=0.046) and kitchen type (0.1%, p=0.803; table 4). Higher indoor $PM_{2.5}$ concentrations were strongly associated with the probability of permitted indoor smoking, ranging from 0 to 1 for concentrations of 20–400 μg/m[3], respectively (figure 3).

Association between indoor $PM_{2.5}$ and biomarker of SHS exposure

Workers' urinary cotinine levels were strongly associated with indoor particulate concentrations. Indoor $PM_{2.5}$ concentrations in all premises were positively associated with cotinine residuals (p<0.0001), with 45% of the variation explained after removal of the effects attributable to gender, age, height, duration from the last work shift, exposure to co-workers' SHS, daily SHS exposure duration at home and in other places and socioeconomic status (figure 4).

Association between lung function and SHS exposure

There was a strong exposure–response relationship between the strata of indoor $PM_{2.5}$ concentrations and the three lung function indices measured (table 2). When the analysis was restricted to subgroups that were older or less exposed to smoking, greater negative values in these lung function indices were observed with clear exposure–response relationships. Tests for interactions between indoor $PM_{2.5}$ and sex, age, smoking history and exposure to co-workers' SHS for effects on lung function were not significant. Workplace exposure to SHS measured as urinary cotinine levels was inversely associated with lung function (table 3), including FEV_1 and FEF_{25-75} but not FEV_1/FVC, as there was also an inverse association between FVC and cotinine (p=0.041). When compared with the referent group, the point estimates for FVC among the urinary cotinine strata showed a significant trend (p=0.026) from the lowest to the highest stratum (−34 ml (95% CI −202 to 134), −55 ml (95% CI −217 to 108), −157 ml (95% CI −308 to −6)).

Lung function indices per unit of natural log indoor $PM_{2.5}$ concentration

Indoor $PM_{2.5}$ concentrations were consistently negatively associated with lung function indices (p=0.001–0.021, figure 5). When the analysis was restricted to the older workers, larger negative effects were observed in FEF_{25-75} (p=0.006), FEV_1 (p=0.068) and FEV_1/FVC (p=0.005). Tests for interactions between indoor $PM_{2.5}$ and age group for effects on lung function were not significant. Based on the lung function indices for all workers in the sample, the predicted lung function for exposure to the average indoor $PM_{2.5}$ levels in smoking premises compared with non-smoking premises were lower by 42 ml (FEV_1), 253 ml/s (FEF_{25-75}) and 2.0% (FEV_1/FVC). These apparent reductions in lung function were lower by 26–50% in the older subgroup compared with the average for all workers. No associations were found between FVC and natural log indoor $PM_{2.5}$ concentrations (p=0.156).

Table 3 Lung function indices (with 95% CI) of non-smoking catering workers for different ranges of urinary cotinine (ng/ml)

Cotinine (ng/ml)	n_0	n_1	FEV_1 (l)	FEF_{25-75} (l/s^{-1})	FEV_1/FVC (%)
≤0.8	37	1	0	0	0
0.8–1.6	27	2	−0.067 (−0.197 to 0.063)	−0.065 (−0.389 to 0.260)	0.019 (−0.007 to 0.044)
1.6–3.2	46	1	−0.093 (−0.235 to 0.049)	−0.329 (−0.659 to 0.000)*	−0.011 (−0.041 to 0.019)
>3.2	30	42	−0.143 (−0.260 to −0.026)*	−0.335 (−0.635 to −0.036)*	−0.001 (−0.028 to 0.026)
			p for trend=0.017	p for trend=0.015	p for trend=0.528

*p<0.05.
Estimates were adjusted for gender, age, height, duration from the last shift of work, exposure to co-workers' SHS, daily duration of SHS exposure at home and in other places, education and income.
FEV_1, forced expiratory volume in 1 s; FEF_{25-75}, forced expiratory flow as 25–75% of FVC; FVC, forced vital capacity; n_0, number of workers from non-smoking premises; n_1, number of workers from smoking premises; SHS, second-hand smoke.

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

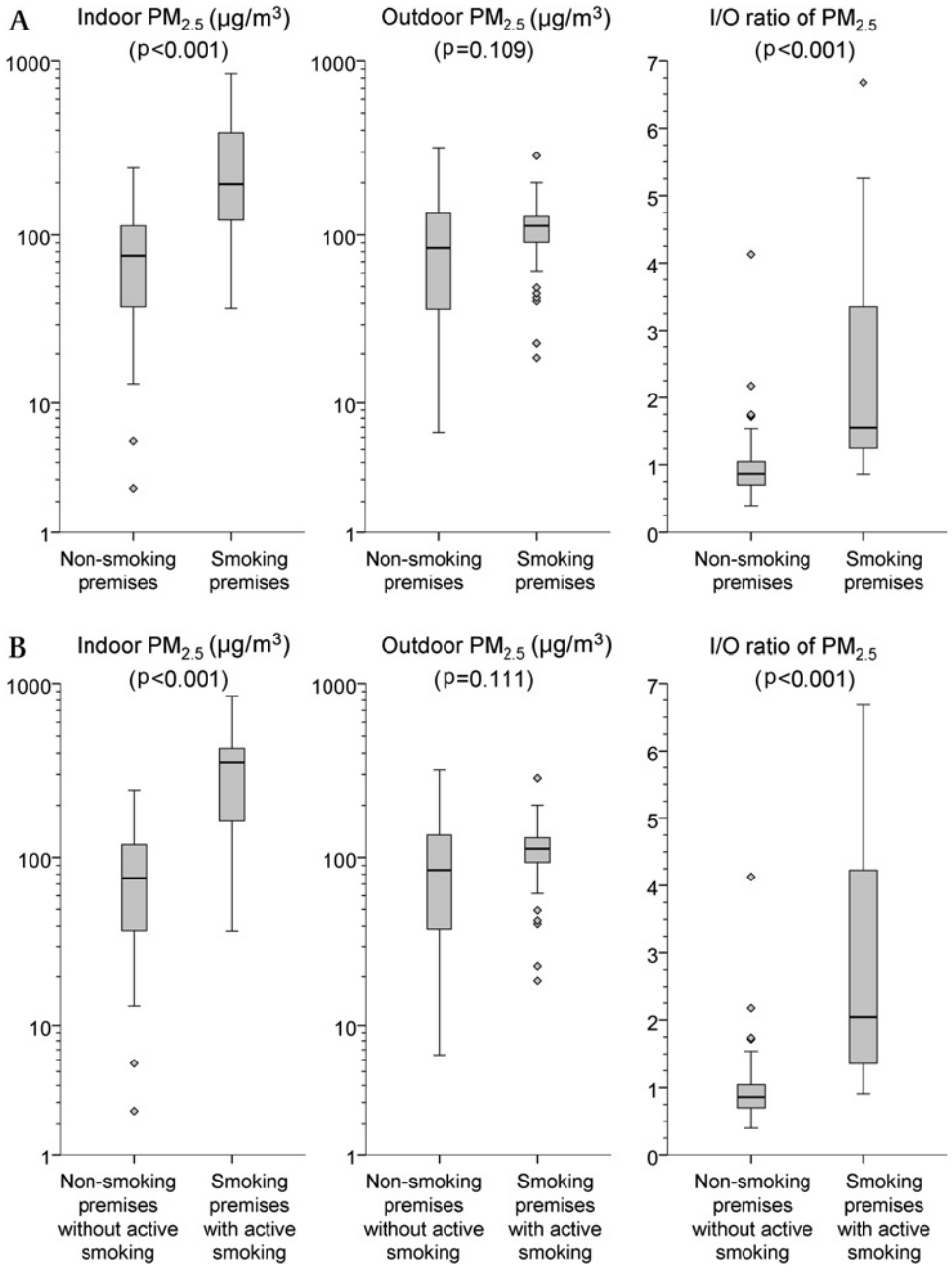


Figure 2 Boxplots of $PM_{2.5}$ concentrations and their indoor-outdoor (I/O) ratios. (A) All smoking (n=36) and non-smoking (n=63) premises. (B) Premises with (n=27) and without (n=62) observed indoor smoking. The boxes show the medians, interquartile ranges and the bars that were away from the box by 1.5 times the interquartile range; p values were obtained by the ANOVA F-test (two-tailed).

DISCUSSION

This is the first study on non-smoking catering workers which clearly shows an inverse exposure–response relationship between lung function and workplace SHS levels quantified by both indoor fine particulate concentrations and urinary cotinine levels. The lack of a consistent relationship in previous studies may be explained by insufficient precision of exposure measurements to allow detection of changes in lung function. Most studies[21–23] have measured SHS by questionnaires in which the participants self-reported exposures as yes/no answers, exposure duration and number of smokers or cigarettes. However, these measures may be biased by either over-reporting or limited awareness of exposure by the participants.[21] Self-reported SHS exposure of 3534 adult never-smokers in Switzerland showed no associations between workplace exposure and lung function except for subjects with asthma and women.[21] A questionnaire survey in 7882 never-smoking adults in 16 European countries reported no significant associations between FEV_1 and SHS exposure at work.[22] In 1033 Chinese subjects, a questionnaire survey found significant associations mimicking an exposure–response relationship between FEV_1 and categories of SHS exposures based on exposure 'only at work or at home' and 'both at work and at home' in never-smokers.[23]

The evidence for the effects of SHS on lung function is supported when cotinine is used as the exposure measure. For example, a salivary cotinine study of 1623 non-smokers demonstrated an exposure–response relationship in the highest two of five levels of cotinine, but no association among the never-smoker subgroup.[24] A study of a composite score for both self-reported exposure data and serum cotinine samples in 301 never-smokers in Scotland reported an exposure–response association between both FEV_1 and FVC and SHS exposure, but the association was less clear when using either self-reported data or cotinine levels separately.[25] Another study in the USA of self-reported exposure data and serum cotinine levels in 10 581 non-smokers found a clear exposure–response relationship for

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

Table 4 Smoking as a key determinant of natural log indoor $PM_{2.5}$ concentrations ($\mu g/m^3$) in catering premises (n=99) compared with other variables

Determinants	Partial η^2	β (95% CI)
Intercept	–	3.011 (2.542 to 3.479)****
Number of burning cigarettes	57.2%	0.314 (0.227 to 0.402)****
Ventilation type	10.1%	
Air-conditioned only		0.000
Air-conditioned and natural ventilation		−0.218 (−0.495 to 0.058)
Natural ventilation only		−1.128 (−2.022 to −0.233)*
Outdoor $PM_{2.5}$ concentrations†	7.0%	0.159 (0.119 to 0.200)****
Burning objects	2.1%	
Absence		0.000
Presence		0.418 (0.052 to 0.785)*
Volume of the premise (m^3)	2.0%	−0.001 (−0.001 to 0.000)*
Kitchen type	0.1%	
Closed kitchen		0.000
Open kitchen		−0.013 (−0.574 to 0.548)

Model R^2=0.66.
*p<0.05; ****p<0.0001.
†Square root-transformed.

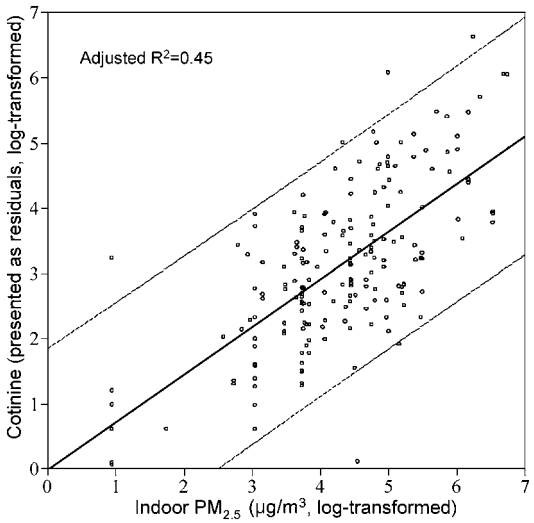


Figure 4 Partial residual plot for association between urinary cotinine level (ng/ml) and indoor $PM_{2.5}$ concentration ($\mu g/m^3$).

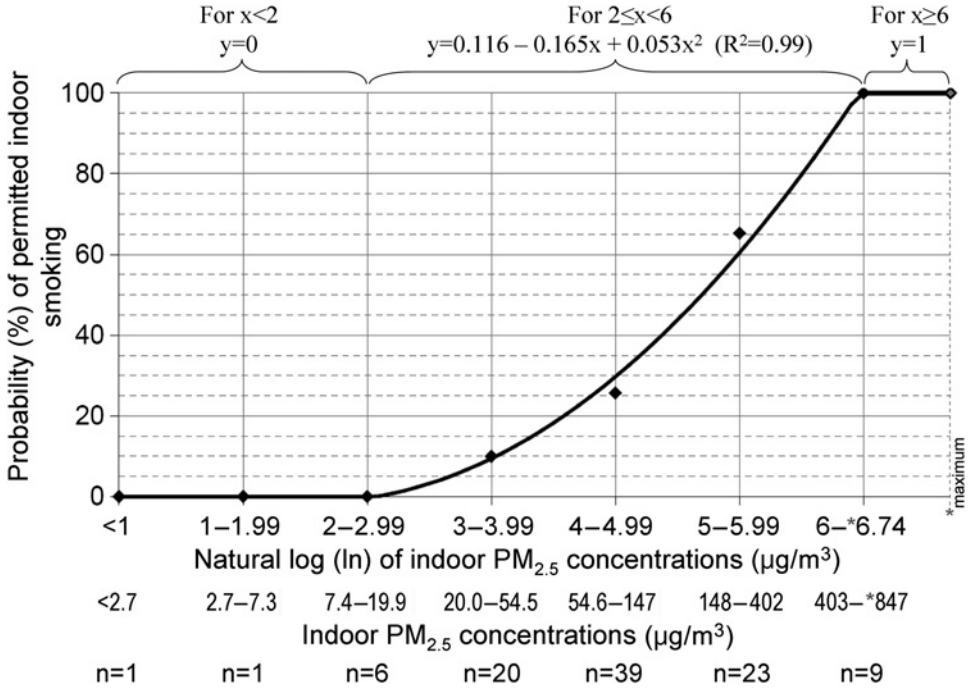
the FEV_1/FVC ratio, but only for FEV_1 among the female subgroup.[26]

One of the major limitations of using cotinine as a biomarker of exposure to nicotine in SHS is the inter-individual variation in cotinine metabolism, with a delay of 4–6 h to the peak plasma cotinine level after SHS exposure followed by a reduction at different individual removal rates.[27] Plasma cotinine concentrations could vary at different times of the day,[27] so heterogeneity in the sample collection timing from the last exposure could lead to marked variation in estimates of SHS exposures. Our urine samples were collected in workplaces during evening business hours with the advantage that the times elapsed since previous workplace exposures were similar. Our regression adjustments for SHS from sources outside workplaces also reduced residual effects due to multiple sources.

Measurement of both indoor $PM_{2.5}$ concentrations and lung function at the same time and location, and analysis of their associations with adjustments for long-term exposure[7] and other microenvironmental sources of SHS, can eliminate errors in the exposure timing and source. There are few reports on lung function and indoor $PM_{2.5}$ as a measure of SHS level in the workplace. Non-tobacco $PM_{2.5}$ sources were unlikely to have affected our exposure estimates as we found smoking was the dominant determinant of indoor $PM_{2.5}$ with eight times the contribution from outdoor $PM_{2.5}$. Since reverse causality is impossible given the involuntary nature of SHS exposure, the strong exposure–response relationships with triangulated linear associations between lung function and two different exposure measures (indoor $PM_{2.5}$ and cotinine) provide the strongest evidence so far reported for impairment of lung function by SHS.

Our study showed that the differences in lung function between smoking and non-smoking premises (figure 5) are similar to those from the pre- and post-smoking ban studies.[2 9–11] This finding, together with the well-established ageing effects on lung function[28] consistent with the greater negative coefficients in our older subgroup, support the validity of our results. A cotinine

Figure 3 Probability of permitted indoor smoking in Hong Kong (y) versus natural log indoor $PM_{2.5}$ concentration (x) among all smoking (n=36) and non-smoking (n=63) catering premises.



Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

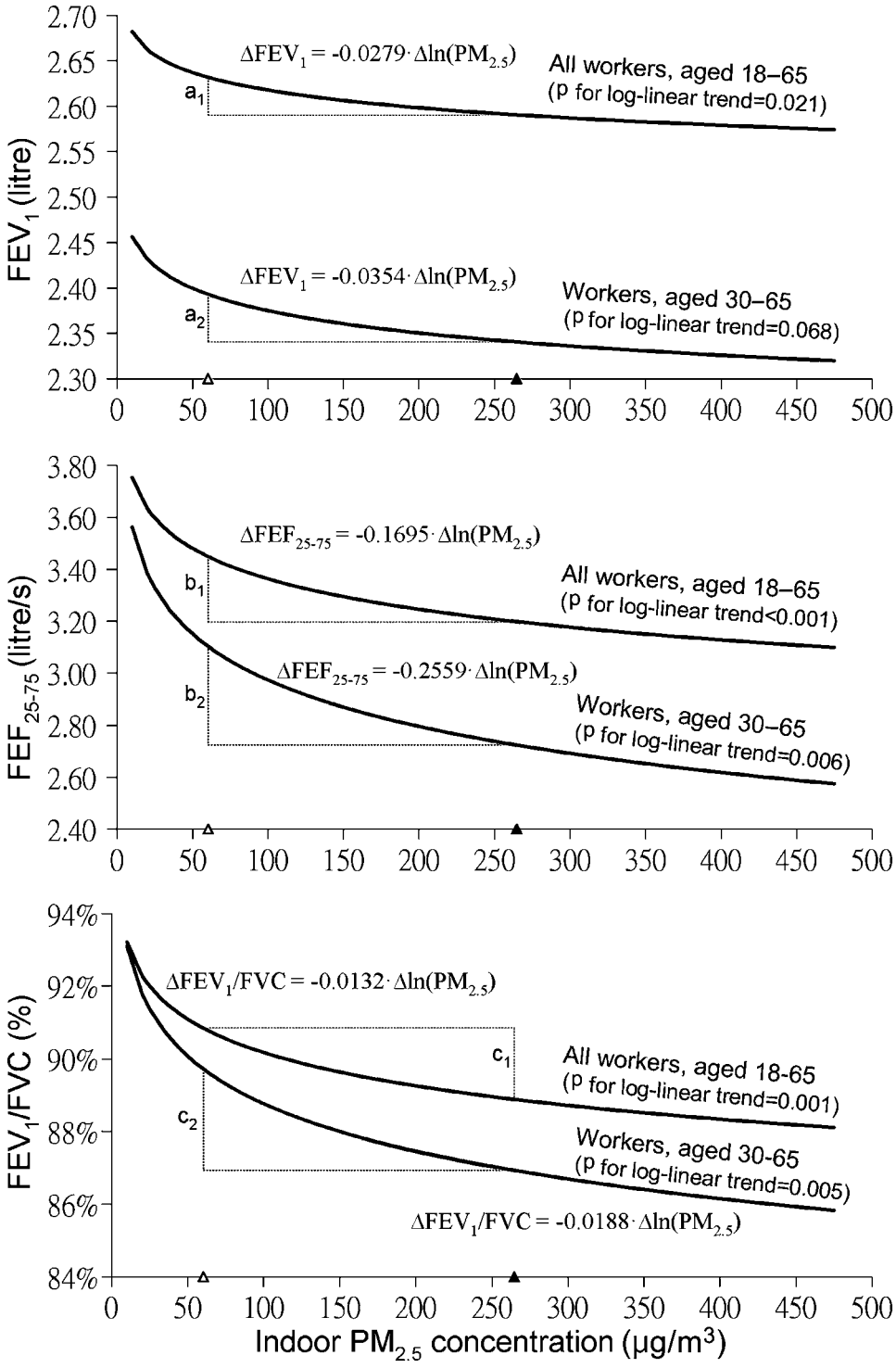


Figure 5 Estimated lung function per unit change of indoor $PM_{2.5}$ concentration among all recruited workers (n=186). a_1=0.04, a_2=0.05, b_1=0.25, b_2=0.38, c_1=2.0, c_2=2.8 represent the difference in the estimated mean lung function corresponding to the mean indoor $PM_{2.5}$ concentrations 60.3 μg/m^3 (open triangle) and 267.9 μg/m^3 (solid triangle) in non-smoking and smoking premises, respectively. Lung function indices were adjusted where appropriate for gender, age, height, duration of SHS exposure at work, daily duration of SHS exposure at home and in other places, education, income, job position, the effect of patio seating and FVC. FEV_1, forced expiratory volume in 1 s; FEF_{25-75}, forced expiratory flow as 25–75% of FVC; FVC, forced vital capacity; SHS, second-hand smoke.

study which reported that newly exposed subjects showed a greater decline in lung function while those who had been living with a smoker showed the smallest decline[24] also supports our finding that the lung function indices in all $PM_{2.5}$ exposure strata consistently became more negative when restricted to subgroups that were relatively less exposed to SHS (table 2).

Although we used a portable office spirometer rather than a standard diagnostic spirometer in a hospital laboratory setting, the MicroLoop spirometer has a precision comparable to that of a standard instrument and is one of the few that has acceptable limits of agreement with standard instruments in all lung function measures.[29]

The downward levelling trends of the estimated lung function per unit increase of indoor $PM_{2.5}$ concentration (p=0.001–0.021) indicate the sensitivity of pulmonary function to SHS with apparent greater reductions in lung function at lower than at higher particulate concentrations (figure 5), similar to the observations on the non-linear effect of both combustion and ambient particulates on cardiovascular disease mortality.[30] Particulates that penetrate to the deepest part of the lung and cause alveolar epithelial injury are associated with both acute reversible inflammatory responses and cumulative irreversible pulmonary damage as indicated by structural changes in airways.[31 32] The observed trends may be related to a saturation

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

mechanism occurring at lower exposure levels[30] where both irreversible and reversible processes may simultaneously exist. The cross-sectional nature of our study limits further interpretation.

To our knowledge, this is the first study showing a non-linear relationship between indoor $PM_{2.5}$ and the probability of smoking being permitted. This method may be particularly useful for tobacco control inspections and enforcement following smoking ban legislation.

Our exposure–response coefficients for lung function also provide a basis for health impact assessments and policy reviews. Lung function is an important indicator of future health and life expectancy.[33] Adverse health effects attributable to the lack of workplace smoking bans include higher risks for respiratory, nasal and ocular symptoms, and hospital admissions for coronary heart disease including acute myocardial infarction.[2] We found strong evidence that the respiratory health of non-smoking workers in exempted premises was damaged by tobacco smoke emissions, and harm to the respiratory system would also occur in workers who were current smokers.[34] In female workers, who comprised two-thirds of the catering workforce, pregnancy could result in retarded fetal growth as a result of SHS exposure.[35]

Our data show that complete prohibition of smoking can reduce indoor fine particulate levels by more than 75% (from 267.9 to 60.3 $\mu g/m^3$) on average, even in locations close to the roadside, and protect the respiratory health of the catering workforce. In Hong Kong's highly polluted environment, mean ambient $PM_{2.5}$ levels (38.4 $\mu g/m^3$ in 2008) are well above the WHO annual Air Quality Guideline of 10 $\mu g/m^3$. There is consistent evidence worldwide that legislative bans are effective in reducing SHS exposure and improving health,[2] but public and occupational health principles have been set aside owing to the influence of vested interests and poor legal draftsmanship on smoke-free legislation.

CONCLUSION

The lung function of workers is inversely associated with exposure to workplace SHS, with a clear exposure–response relationship. The 30-month exemption period of the smoke-free legislation in Hong Kong (2006 Smoking (Public Health) Ordinance) was directly responsible for the impairment of workers' health.

Acknowledgements We thank LY Ho, WL Cheung, J Chen and CH Lee for their help in data collection and LC Wong for her help in study preparation and data management.

Funding AJH received funding for this study from the Health and Health Services Research Fund of the Hong Kong SAR government (HHSRF#05060661).

Competing interests None.

Ethics approval This study was conducted with the approval of the Institutional Review Board of the University of Hong Kong/Hospital Authority Hong Kong West Cluster.

Contributors AJH, JR and SMM conceived and designed the study with RF and C-MW. H-KL, AJH and C-MW analysed the data and drafted the manuscript. AJH, SMM and H-KL were responsible for data collected and managed by CS, Q-YL and others. All authors interpreted the data, critically revised the draft for important intellectual content and gave final approval of the version to be published. AJH is the guarantor.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1. **Wortley PM,** Caraballo RS, Pederson LL, *et al*. Exposure to secondhand smoke in the workplace: serum cotinine by occupation. *J Occup Environ Med* 2002;**44**:503–9.
2. **Callinan JE,** Clarke A, Doherty K, *et al*. Legislative smoking bans for reducing secondhand smoke exposure, smoking prevalence and tobacco consumption. *Cochrane Database Syst Rev* 2010;(4):CD005992.
3. **Health, Welfare and Food Bureau.** *The Administration's Paper on the Proposed Adaptation Periods for Amendments to the Smoking (Public Health) Ordinance*. The Legislative Council Library, 2006:CB(2)1916/05–06(01). http://www.legco.gov.hk/yr04-05/english/bc/bc61/papers/bc610504cb2-1916-1e.pdf (accessed 21 Apr 2011).
4. **Zhang JY,** Cao YX, Xu CB, *et al*. Lipid-soluble smoke particles damage endothelial cells and reduce endothelium-dependent dilatation in rat and man. *BMC Cardiovasc Disord* 2006;**6**:3.
5. **Adair-Kirk TL,** Atkinson JJ, Senior RM. Smoke particulates stress lung cells. *Nat Med* 2008;**14**:1024–5.
6. **U.S. Department of Health and Human Services.** *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2006.
7. **Eisner MD,** Wang Y, Haight TJ, *et al*. Secondhand smoke exposure, pulmonary function, and cardiovascular mortality. *Ann Epidemiol* 2007;**17**:364–73.
8. **U.S. California Environmental Protection Agency.** *Health Effects of Exposure to Environmental Tobacco Smoke—Final Report*. U.S. California Environmental Protection Agency. Office of Environmental Health Hazard Assessment, 1997. http://oehha.ca.gov/air/environmental_tobacco/finalets.html (accessed 06 Dec 2010).
9. **Eisner MD,** Alexander KS, Paul DB. Bartenders' respiratory health after establishment of smoke-free bars and taverns. *JAMA* 1998;**280**:1909–13.
10. **Skogstad M,** Kjaerheim K, Fladseth G, *et al*. Cross shift changes in lung function among bar and restaurant workers before and after implementation of a smoking ban. *Occup Environ Med* 2006;**63**:482–7.
11. **Menzies D,** Nair A, Williamson PA, *et al*. Respiratory symptoms, pulmonary function, and markers of inflammation among bar workers before and after a legislative ban on smoking in public places. *JAMA* 2006;**296**:1742–8.
12. **Anderson HR.** Differential epidemiology of ambient aerosols. *Philos Trans R Soc Lond A* 2000;**358**:2771–85.
13. **McDonnell WF,** Nishino-Ishikawa N, Petersen FF, *et al*. Relationships of mortality with the fine and coarse fraction of long-term ambient PM10 concentrations in nonsmokers. *J Expo Anal Environ Epidemiol* 2000;**10**:427–36.
14. **Wong CM,** Ma S, Hedley AJ, *et al*. Effect of air pollution on daily mortality in Hong Kong. *Environ Health Perspect* 2001;**109**:335–40.
15. **Middleton ET,** Morice AH. Breath carbon monoxide as an indication of smoking habit. *Chest* 2000;**117**:758–63.
16. **Valente P,** Forastiere F, Bacosi A, *et al*. Exposure to fine and ultrafine particles from secondhand smoke in public places before and after the smoking ban, Italy 2005. *Tob Contol* 2007;**16**:312–17.
17. **Ramachandran G,** Adgate JL, Pratt GC, *et al*. Characterizing indoor and outdoor 15 minute average $PM_{2.5}$ concentrations in urban neighborhoods. *Aerosol Sci Technol* 2003;**37**:33–45.
18. **Brutsche MH,** Downs SH, Schindler C, *et al*. Bronchial hyperresponsiveness and the development of asthma and COPD in asymptomatic individuals: SAPALDIA Cohort Study. *Thorax* 2006;**61**:671–7.
19. **Aquilina NJ,** Delgado-Saborit JM, Meddings C, *et al*. Environmental and biological monitoring of exposures to PAHs and ETS in the general population. *Environ Int* 2010;**36**:763–71.
20. **Larsen WA,** McCleary SJ. The use of partial residual plots in regression analysis. *Technometrics* 1972;**14**:781–90.
21. **Künzli N,** Schwartz J, Stutz EZ, *et al*; SAPALDIA-Team. Association of environmental tobacco smoke at work and forced expiratory lung function among never smoking asthmatics and non-asthmatics. *Soz Präventivmed* 2000;**45**:208–17.
22. **Janson C,** Chinn S, Javis D, *et al*. Effect of passive smoking on respiratory symptoms, bronchial responsiveness, lung function, and total serum IgE in the European Community Respiratory Health Survey: a cross-sectional study. *Lancet* 2001;**358**:2103–9.
23. **Xu X,** Li B. Exposure-response relationship between passive smoking and adult pulmonary function. *Am J Respir Crit Care Med* 1995;**151**:41–6.
24. **Carey IM,** Cook DG, Strachan DP. The effects of environmental tobacco smoke exposure on lung function in a longitudinal study of British adults. *Epidemiology* 1999;**10**:319–26.
25. **Chen R,** Tunstall-Pedoe H, Tavendale R. Environmental tobacco smoke and lung function in employees who never smoked: the Scottish MONICA study. *Occup Environ Med* 2001;**58**:563–8.
26. **Eisner MD.** Environmental tobacco smoke exposure and pulmonary function among adults in NHANES III: Impact on the general population and adults with current asthma. *Environ Health Perspect* 2002;**110**:765–70.
27. **Benowitz NL.** Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol Rev* 1996;**18**:188–204.
28. **Wahba WM.** Influence of aging on lung-function—clinical significance of changes from age 20. *Anesth Analg* 1983;**62**:764–76.
29. **Liistro G,** Vanwelde C, Vincken W, *et al*. Technical and functional assessment of 10 office spirometers: a multicenter comparative study. *Chest* 2006;**130**:657–65.

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.

30. **Smith KR,** Peel JL. Mind the gap. *Environ Health Perspect* 2010;**118**:1643–5.
31. **Dockery DW,** Speizer FE, Ferris BG, *et al*. Cumulative and reversible effects of lifetime smoking on simple tests of lung function in adults. *Am Rev Respir Dis* 1988;**137**:286–92.
32. **Flouris AD,** Metsios GS, Carrillo AE, *et al*. Acute and short-term effects of secondhand smoke on lung function and cytokine production. *Am J Respir Crit Care Med* 2009;**179**:1029–33.
33. **Shavelle RM,** Paculdo DR, Kush SJ, *et al*. Life expectancy and years of life lost in chronic obstructive pulmonary disease: findings from the NHANES III follow-up study. *Int J Chron Obstruct Pulmon Dis* 2009;**4**:137–48.
34. **Lam TH,** Ho LM, Hedley AJ, *et al*. Secondhand smoke and respiratory ill health in current smokers. *Tob Control* 2005;**14**:307–14.
35. **Hui LL,** Leung GM, Cowling BJ, *et al*. Determinants of infant growth: evidence from Hong Kong's "Children of 1997" birth cohort. *Ann Epidemiol* 2010;**20**:827–35.

Thorax: first published as 10.1136/thx.2011.160291 on 6 May 2011. Downloaded from http://thorax.bmj.com/ on April 10, 2024 by guest. Protected by copyright.