indoor workplaces, public indoor places, and some public outdoor places. However, bars, bathhouses, nightclubs, clubs, massage establishments, and mahjong-tin kau premises were exempted until July 2009. The main aim of this legislation was to reduce exposure of workers to SHS. The likely impact of such legislation on health and health care costs was a concern.

We have calculated the cost of active and passive smoking in Hong Kong.[4] To estimate the immediate impact of smoke-free policies on health and costs, we need to monitor the changes in health-related variables over the early intervention period. The Hospital Authority Clinical Management System provides data on mortality and admissions to hospital. We used such data to examine the immediate impact of a reduction of exposure to SHS in the Hong Kong population following the workplace smoking ban. In examining the effect of change in health-related variables following a change in population exposure to airborne toxins, we used statistical methods that had been used to identify the impact of reductions in sulfur dioxide as a result of switching to lower sulfur fuel.[5] In that study, because the intervention took place in 1990, only mortality data were available. The current study was able to make use of health care utilisation data as well.

This study was carried out as soon as feasible in order to provide timely information on the impact of the legislation. It aimed to examine the health effects following the implementation of smoke-free workplaces in Hong Kong by (1) examining trends in deaths and admissions to hospital for conditions associated with passive smoking over the years prior to and immediately following implementation of the smoke-free policy on 1 January 2007, and (2) examining whether there was any discernable change in rates of deaths and admissions for these conditions that coincided with introduction of the policy after accounting for the underlying trends and impacts of other confounders.

Methods

This study was conducted from December 2006 to March 2009. The trends in deaths and admissions between the pre- (1997-2006) and post- (2007-2008) intervention periods were estimated and modelled, and changes between pre and post-intervention periods were calculated. Data on discharges from 31 acute hospitals collected by the Hospital Authority Clinical Management System were obtained for the following conditions: ischaemic heart disease (IHD) [ICD-9-CM 410-414], acute myocardial infarctions (AMI) [410], cerebrovascular disease (430-438), cardiovascular disease (390-459), respiratory disease (460-519), lung cancer (162), and all natural causes (001-799). Injury, poisoning and external causes (800-999, e800-e999), cancer excluding lung cancer (140-161, 163-239), natural causes excluding cardiovascular and respiratory disease (001-389, 520-799), and other causes (001-009,140-161, 163-246, 280-294, 320-326, 520-629, 710-719) were used as control conditions. The year 2003 was excluded in the model for admissions, as the admission trends changed during the outbreak of severe acute respiratory syndrome (SARS) in 2003. Information on deaths obtained from the Hong Kong Census

Annual proportional changes in all ages (%) [95% CI]		Relative change (%) [95% CI]	
Post-intervention (2007-2008)		From pre- to post-intervention	
Hospital admission	Mortality	Hospital admission	Mortality
-6.33 (-11.05 to -1.36)†	-0.63 (-4.61 to 3.50)	-9.00 (-13.59 to -4.17)†	-2.09 (-6.02 to 2.00)
1.81 (-9.46 to 14.49)	-2.76 (-8.64 to 3.50)	0.17 (-10.93 to 12.65)	-1.58 (-7.55 to 4.78)
-0.83 (-6.26 to 4.93)	1.71 (-2.70 to 6.33)	-1.59 (-6.99 to 4.12)	1.78 (-2.65 to 6.42)
-0.39 (-2.86 to 2.15)	1.38 (-1.22 to 4.05)	-1.22 (-3.68 to 1.30)	0.35 (-2.24 to 3.00)
-0.63 (-3.09 to 1.89)	1.27 (-1.73 to 4.36)	2.55 (0.01 to 5.16)†	0.59 (-2.40 to 3.67)
12.67 (4.28 to 21.74)†	-4.22 (-8.35 to 0.09)	14.33 (5.81 to 23.53)†	-5.65 (-9.73 to -1.39)†
2.25 (1.43 to 3.08)†	0.88 (-0.48 to 2.26)	2.30 (1.48 to 3.13)†	-0.55 (-1.90 to 0.81)
0.28 (-2.98 to 3.66)	-1.77 (-8.44 to 5.38)	5.18 (1.76 to 8.72)†	-0.84 (-7.58 to 6.41)
3.28 (0.66 to 5.97)†	1.04 (-1.72 to 3.87)	2.24 (-0.36 to 4.91)	-0.17 (-2.90 to 2.64)
3.17 (2.24 to 4.11)†	0.49 (-1.38 to 2.40)	2.87 (1.95 to 3.81)†	-1.42 (-3.27 to 0.46)
3.28 (1.96 to 4.62)†	1.22 (-1.07 to 3.57)	2.27 (0.96 to 3.59)†	-0.38 (-2.65 to 1.93)

TABLE 2. Seasonal variations in hospital admission and mortality

Disease	Seasonal variations in all ages (%) [95% CI]	
	Baseline (1997-2006)	
	Hospital admission	Mortality
Outcome condition		
Ischaemic heart disease	2.87 (2.21 to 3.52)†	21.12 (19.62 to 22.61)†
Acute myocardial infarctions	13.23 (11.71 to 14.75)†	19.05 (16.97 to 21.12)†
Cerebrovascular	4.21 (3.52 to 4.90)†	17.78 (16.25 to 19.31)†
Cardiovascular	5.49 (5.17 to 5.81)†	20.22 (19.30 to 21.15)†
Respiratory	12.62 (12.33 to 12.91)†	17.61 (16.48 to 18.74)†
Lung cancer	2.96 (2.02 to 3.91)†	2.43 (0.91 to 3.95)†
All natural causes	0.55 (0.45 to 0.65)†	11.65 (11.16 to 12.13)†
Control condition		
Injury, poisoning and external causes	3.26 (2.90 to 3.62)†	16.26 (14.03 to 18.48)†
Cancer excluding lung cancer	2.75 (2.42 to 3.08)†	1.06 (0.09 to 2.02)†
Natural causes excluding cardiorespiratory	2.21 (2.10 to 2.32)†	5.46 (4.80 to 6.13)†
Other causes*	1.04 (0.88 to 1.20)†	4.88 (4.06 to 5.70)†

* Refer to ICD-9 (001-009, 140-161, 163-246, 280-294, 320-326, 520-629, 710-719)
† Significant at 5% level

TABLE 3. Summary of relative changes from pre- to post-intervention in all ages in hospital admission and mortality

Disease	Relative changes in all ages (%) [95% CI]	
	Main model	
	Hospital admission	Mortality
Outcome conditions		
Ischaemic heart disease	-9.00 (-13.59 to -4.17)†	-2.09 (-6.02 to 2.00)
Acute myocardial infarctions	0.17 (-10.93 to 12.65)	-1.58 (-7.55 to 4.78)
Cerebrovascular	-1.59 (-6.99 to 4.12)	1.78 (-2.65 to 6.42)
Cardiovascular	-1.22 (-3.68 to 1.30)	0.35 (-2.24 to 3.00)
Respiratory	2.55 (0.01 to 5.16)†	0.59 (-2.40 to 3.67)
Lung Cancer	14.33 (5.81 to 23.53)†	-5.65 (-9.73 to -1.39)†
All natural causes	2.30 (1.48 to 3.13)†	-0.55 (-1.90 to 0.81)
Control conditions		
Injury, poisoning and external causes	5.18 (1.76 to 8.72)†	-0.84 (-7.58 to 6.41)
Cancer excluding lung cancer	2.24 (-0.36 to 4.91)	-0.17 (-2.90 to 2.64)
Natural causes excluding cardiovascular and respiratory	2.87 (1.95 to 3.81)†	-1.42 (-3.27 to 0.46)
Other causes*	2.27 (0.96 to 3.59)†	-0.38 (-2.65 to 1.93)

* Refer to ICD-9 (001-009, 140-161, 163-246, 280-294, 320-326, 520-629, 710-719)
† Significant at 5% level

Seasonal variations in all ages (%) [95% CI]			
Year 1 (2007)		Year 2 (2008)	
Hospital admission	Mortality	Hospital admission	Mortality
4.99 (3.15 to 6.83)†	9.45 (5.12 to 13.79)†	4.68 (2.88 to 6.47)†	20.86 (16.51 to 25.22)†
9.10 (4.75 to 13.45)†	8.94 (2.35 to 15.53)†	24.40 (20.10 to 28.70)†	21.61 (14.92 to 28.30)†
6.41 (4.41 to 8.41)†	13.18 (8.42 to 17.94)†	6.20 (4.23 to 8.16)†	15.19 (10.51 to 19.88)†
8.64 (7.72 to 9.56)†	12.80 (10.02 to 15.57)†	9.39 (8.51 to 10.26)†	15.59 (12.84 to 18.34)†
10.08 (9.19 to 10.97)†	13.62 (10.37 to 16.86)†	12.94 (12.07 to 13.81)†	20.01 (16.86 to 23.17)†
6.18 (3.48 to 8.88)†	3.17 (-1.45 to 7.79)	6.37 (3.84 to 8.91)†	1.97 (-2.76 to 6.69)
0.32 (0.04 to 0.61)†	8.65 (7.19 to 10.10)†	1.15 (0.88 to 1.43)†	10.70 (9.27 to 12.13)†
2.46 (1.31 to 3.62)†	11.19 (3.69 to 18.70)†	0.80 (-0.34 to 1.93)	11.38 (3.99 to 18.76)†
2.22 (1.32 to 3.12)†	1.77 (-1.18 to 4.72)	2.04 (1.17 to 2.91)†	2.75 (-0.17 to 5.66)
1.95 (1.64 to 2.27)†	4.90 (2.89 to 6.90)†	1.29 (0.99 to 1.60)†	4.84 (2.85 to 6.82)†
0.62 (0.17 to 1.08)†	4.80 (2.35 to 7.25)†	1.78 (1.34 to 2.21)†	4.91 (2.50 to 7.32)†

Relative changes in all ages (%) [95% CI]				
SARS	Air pollutants		Hospital beds and smoking prevalence	
Hospital admission	Hospital admission	Mortality	Hospital admission	Mortality
-5.56 (-10.39 to -0.46)†	-7.39 (-12.48 to -2.01)†	-2.13 (-6.35 to 2.27)	-9.65 (-14.22 to -4.84)†	-2.17 (-6.10 to 1.92)
7.22 (-4.84 to 20.82)	0.22 (-11.96 to 14.08)	-0.84 (-7.29 to 6.05)	-3.92 (-14.60 to 8.09)	-1.71 (-7.67 to 4.65)
-0.75 (-6.28 to 5.10)	-2.43 (-8.29 to 3.81)	2.16 (-2.62 to 7.17)	-2.41 (-7.78 to 3.27)	1.77 (-2.66 to 6.41)
0.64 (-1.90 to 3.25)	0.27 (-2.46 to 3.09)	0.72 (-2.07 to 3.58)	-1.78 (-4.23 to 0.73)	0.29 (-2.29 to 2.93)
2.39 (-0.19 to 5.03)	3.08 (0.27 to 5.96)†	0.45 (-2.76 to 3.77)	2.78 (0.22 to 5.40)†	0.29 (-2.70 to 3.36)
14.05 (5.42 to 23.38)†	17.52 (7.96 to 27.94)†	-4.98 (-9.39 to -0.35)†	14.68 (6.11 to 23.94)†	-5.63 (-9.71 to -1.37)†
3.18 (2.34 to 4.03)†	3.85 (2.94 to 4.77)†	-0.57 (-2.02 to 0.90)	1.78 (0.96 to 2.61)†	-0.61 (-1.96 to 0.75)
3.65 (0.23 to 7.19)†	7.45 (3.63 to 11.42)†	2.07 (-5.38 to 10.11)	5.29 (1.85 to 8.84)†	-0.97 (-7.71 to 6.26)
1.53 (-1.09 to 4.22)	5.53 (2.60 to 8.54)†	-0.44 (-3.37 to 2.58)	2.89 (0.27 to 5.59)†	-0.15 (-2.88 to 2.66)
3.79 (2.83 to 4.74)†	4.55 (3.52 to 5.59)†	-1.69 (-3.67 to 0.33)	2.24 (1.31 to 3.17)†	-1.41 (-3.25 to 0.47)
4.00 (2.64 to 5.37)†	3.42 (1.97 to 4.88)†	-0.67 (-3.10 to 1.82)	1.80 (0.49 to 3.13)†	-0.38 (-2.64 to 1.94)

and Statistics Department for the pre- and post-intervention periods were used to examine mortality for the conditions described above (ICD-10 codes were used in recent years and matched with equivalent ICD-9 codes).

Poisson regression models were used to examine trends in admissions and deaths from 1997 to 2008. Weekly admission and death counts were used as the dependent variables, adjusted with background variables of temperature, humidity, and cycle of seasonality as independent variables to capture the main seasonal variation each year. The regression model was initially fitted for periods of 10 years pre-intervention and 2 years post-intervention separately to estimate the average annual proportional change in admissions and deaths in the pre- and post-intervention periods.

For the relative change in trend between the pre and post-intervention periods, we created a dummy variable (which defined the pre-intervention period as 0 and the post period as 1) and added it as an independent variable in the Poisson models. The coefficient for the variable represented the relative change and the effect of the intervention could be shown in terms of average annual reductions in mortality and admissions.

A measure of proportional change (amplitude) in admissions and deaths was used to examine the seasonal variations to the overall mean in the pre- and post-intervention periods. We fitted a Poisson regression model with deaths or discharges against a pair of trigonometric functions (α sine, β cosine). The estimated coefficients were used to calculate the amplitude of the seasonal curve in terms of sine and cosine in which $\lambda = \sqrt{\alpha^2 + \beta^2}$, with 95% confidence intervals (CI). This amplitude (λ) represented the proportional changes in admissions and deaths in either warm or cool seasons from the relative mean. By comparing the parameters for the 10-year baseline period to the years after the intervention, we were able to evaluate the change in seasonal cycles for each condition that might be associated with the intervention.

We validated our models by examining changes in admission or deaths from conditions, which were less likely to be affected by the smoking ban, such as injury and poisoning, and compared these with the changes in the outcome conditions. For sensitivity analyses, we also adjusted admission trends in pre- and post-SARS periods and tested the background variables including air pollutant concentrations, number of hospital beds, and smoking prevalence in order to identify whether their impact on hospital admissions and deaths might change our conclusions.

Results

After the introduction of the smoke-free law, the annual proportional change in hospital admissions for IHD in all ages dropped by 9% (Table 1). The seasonal peak in hospital admissions for respiratory disease in all ages was reduced from 12.6% to 10.1% in the first year after intervention; however, there was a rebound to 12.9% in the second year (Table 2). For mortality in all ages, only those died from lung cancer immediate dropped by 5.7% after the intervention (Table 1). However, the seasonal peak for deaths from IHD declined from 21.1% to 9.5% and for all natural causes from 11.7% to 8.7% (Table 2). The seasonal peak for deaths from AMI also reduced in the first year but the change was not significant. In the second year, an increase in the seasonal peak for IHD and AMI deaths to the pre-intervention level was observed (Table 2).

Results from the sensitivity analysis[6] are consistent with the main model (Table 3). An alternative model of the post-SARS period for admission was also tested. The changes in admission from pre- to post-intervention were consistent with the main model but the proportional changes in IHD and two of the control conditions became insignificant. This alternative model may add uncertainty to the results and therefore was not considered for use as the final model.

Discussion

Many studies have reported a decrease in cardiovascular disease after the implementation of smoke-free laws. In the present study, the annual proportional changes in mortality were significant in two conditions: lung cancer (which decreased among all ages) and all natural causes (which increased in the age-group of 40 to 64 years). The reduction in lung cancer deaths is difficult to attribute to the intervention due to the long latency in development of lung cancer, except by the development of earlier detection and better treatments. Also the increase in death rates among the age-group of 40 to 64 years is likely to be due to an increase in numbers of this age-group with an ageing population. We examined the data in another way by investigating the seasonal variations in numbers of deaths and found a significant decrease in the peak for IHD, AMI, cardiovascular, and cerebrovascular disease deaths for the 65+ age-group. However, all of these causes of death returned to previous levels the following year, except for cerebrovascular disease (which increased but not significantly). This kind of rebound after a reduction in peak mortality levels has been seen after other interventions, and has been explained as a delay in some deaths that were expected but occurred in the following high mortality season.

Our results showed an impact on hospital admissions for IHD that the rate was significantly lower after the intervention for all ages and the 65+ age-group. In all age-groups, where we expected an

increase in admissions (as found for all causes), we actually had a decrease in admissions for IHD. For the seasonal variation in the number of admissions, there was a significant drop in admission due to respiratory diseases for all ages and the 65+ age-group. The drop might be related to a reduction in SHS exposure but it is difficult to confirm given the other patterns of admissions.

There were limitations in this study mainly related to data availability and complexity. In particular we only had 2 years of data after the intervention and there were a variety of seasonal trends and many factors affecting the data patterns. Therefore further data in later years are useful to confirm the trends identified here.

The disruption of the provision of hospital services due to SARS in 2003 should be taken into account in any analysis. It may have taken some time for admission trends to recover after 2003 and they may not mirror those before 2003. We have tried to adjust for this in our model and sensitivity analysis.

Finally, Hong Kong allowed exemptions to the smoke-free law and thus some of the population continued to be exposed to SHS till mid-2009. Further data in future years are useful to confirm whether the eventual imposition of smoke-free policies in all workplaces contributes to any further benefit in decreasing rates of IHD.

Conclusion

After the implementation of smoke-free policies in most workplaces in Hong Kong, the predicted impact on admissions to hospital and mortality from IHD was in line with findings from around the world. The drop of 9% in admissions for IHD in Hong Kong was on the low side compared with other countries. However, Hong Kong allowed exemptions from the smoke-free policy until mid-2009. Further analyses of future years' data may help to refine the precise benefit to health following amendments to the ordinance.

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Health of catering workers in Hong Kong: impact of the 2006 tobacco control legislation

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KEY MESSAGES

1. Second-hand tobacco smoke is a poison and a major cause of acute illness, chronic disease, and deaths in those exposed.
2. The 2006 Public Health Ordinance conferred enormous benefits in terms of health protection for catering workers. However, the legislation failed to secure the protection of all workers. The law is frequently violated by workers in supposedly non-smoking venues and the implementation of the ordinance did not take sufficient account of the need for clear advice to management on the mandatory nature of the legislation.
3. Non-smokers in exempted premises were continuously subjected to intensive tobacco smoke exposures. The 2.5 years exemption period predictably caused permanent harm to the health of many workers.
4. The delay in amending the Public Health (Smoking) Ordinance and failure to adhere to an evidence-based approach to tobacco control provides a lesson in the translation of public health evidence into policy and enforcement.

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Introduction

This study evaluated the exposures and health impacts of workplace second-hand smoke (SHS) on non-smoking catering workers after implementation of the 2006 Public Health (Smoking) (Amendment) Ordinance. Businesses that could claim their trade mainly entailed selling alcohol to customers aged over 18 years were exempted. This study provides objective evidence of the impact of this legislation on the health of workers in exempted and non-exempted premises.

Methods

This study was conducted from October 2007 to September 2008. We sampled 204 workers (from 99 premises) and 18 controls. Workers were interviewed using a standard schedule in which socioeconomic and demographic information was recorded together with job descriptions, characteristics of their workplace, health, and smoking history. In non-smokers we measured expired air carbon monoxide, lung function, and urinary cotinine concentrations. In the workplace the physical dimensions, sources of pollution, and indoor and outdoor concentrations of $PM_{2.5}$ (particulates matter <2.5 μm) were measured.

Results

The ordinance in 2006 led to a prohibition of smoking in most hospitality venues. The levels of tobacco chemicals from SHS exposures among non-smokers in these workplaces fell by up to 90%, as indicated by concentrations of the tobacco-specific biomarker cotinine in urine. In workers in Chinese restaurants, the median cotinine was 1.4 ng ml^{-1} compared with 9.3 ng ml^{-1} pre-legislation. In Cha Charn Ting workers, the observed level was 1.4 ng ml^{-1} compared with 23.6 ng ml^{-1} pre-legislation, a reduction of 94%. In venues exempted from the ordinance until June 2009, workers were exposed to high levels of fine particulates ($PM_{2.5}$) and tobacco chemicals from SHS. The urine cotinine levels in workers whose workplace permitted smoking were significantly higher than in workers who were protected by the ordinance. Only 2% of workers in exempted venues had cotinine levels of <1 ng ml^{-1} compared with 78% of low-risk controls and 33% of workers in non-smoking venues. None of the controls or non-smoking venue workers had cotinine levels of >25 ng ml^{-1} compared with 28% of those working in smoking venues (Fig 1).

Smoking outdoors generates dense aerosols of chemicals which contaminate those in the immediate vicinity. Workers in non-smoking restaurants with open doors had higher cotinine levels than those in closed-door venues due to customers smoking outside. The mean cotinine level in workers in restaurants with outdoor smoking areas, such as patios, was 4.1 ng ml^{-1}, which was 100% higher than in non-smoking venue workers.

Violations of the ordinance by customers who smoke were frequently reported (17%, 95% CI=12-24%). Co-worker smoking was reported in non-smoking restaurants (52.7%), venues with patios (61.5%) and bars (91%). Co-worker smoking in break periods was a substantial contribution to the health risks from SHS exposures of non-smoking staff as indicated by their urinary cotinine concentrations.

Workers in exempted venues were more likely to perceive poor air quality (odds ratio [OR]=9.3, 95% CI=4.2-20.9), higher risks from poor air (OR=3.7, 95% CI=1.6-8.6), and higher relative risk compared to other workers (OR=21.5, 95% CI=8.8-52.6). Compared to workers in non-smoking venues, workers in smoking venues were less reactive to SHS exposures and were less bothered by SHS (OR=0.2, 95% CI=0.1-0.5), took less protective action, such as discouraging nearby smoking to avoid smoke (OR=0.2, 95% CI=0.1-0.4).

Workers in non-smoking venues had lower median urine cotinine values if they (1) were bothered by smokers; (2) discouraged nearby smoking; (3) discouraged home smoking, and (4) had higher perceived susceptibility of non-smokers to lung cancer. However, there was considerable overlap of the distribution of cotinine levels between the three categories of avoidance behaviour (low, intermediate, high).

The indoor $PM_{2.5}$ levels across all catering venues correlated strongly with urinary cotinine levels in the workers (P<0.0001). Smoking was the most important determinant of indoor $PM_{2.5}$ in terms of variation explained (57%), followed by ventilation type (10%), and outdoor PM2.5 (7.0%). The number of burning cigarettes increased the indoor $PM_{2.5}$ exponentially (P<0.0001, Fig 2). Indoor mean $PM_{2.5}$ levels in non-smoking venues (geometric mean=60.3 μg m^{-3}) were not significantly different from ambient outdoor levels. Both were 500% above the World Health Organization annual Air Quality Guideline for $PM_{2.5}$ (10 μg m^{-3}). In smoking venues the mean $PM_{2.5}$ was 211.6 μg m^{-3} rising to 267.9 μg m^{-3} when smoking was directly observed which was 4.4 times as high as the mean level of 60 μg m^{-3} in non-smoking venues. Hong Kong is a highly polluted environment with poor air quality, but there was no difference in the outdoor ambient $PM_{2.5}$ levels between the locations of the smoking and non-smoking venues. Our findings on health impacts of SHS exposures cannot be explained in terms of outdoor ambient pollution.

Reports of respiratory symptoms were common among all catering workers. Non-smoking workers in smoking venues had the highest prevalence of throat discomfort, cough, phlegm, nasal symptoms and a higher prevalence overall of any reported symptoms. Working in exempted venues compared with non-exempted venues was strongly associated with reports of coughing (OR=3.6, 95% CI=1.1-12.0). This excess risk of 260% indicates that the respiratory system of these workers is constantly injured by their workplace environment. Those with a history of respiratory illness were particularly vulnerable (OR=3.1, 95% CI=1.1-8.4). The association between SHS exposures and symptoms is supported by the significant association between urine cotinine levels and cough (P<0.0049) and a cluster of upper respiratory symptoms including cough, phlegm, sore throat, and nasal blockage (P=0.023).

Lung function was assessed by spirometry

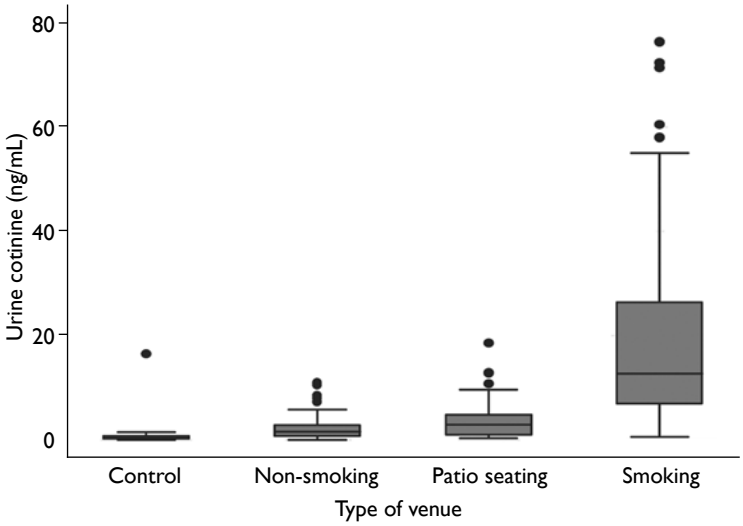

FIG 1. Urine cotinine in non-smoking workers

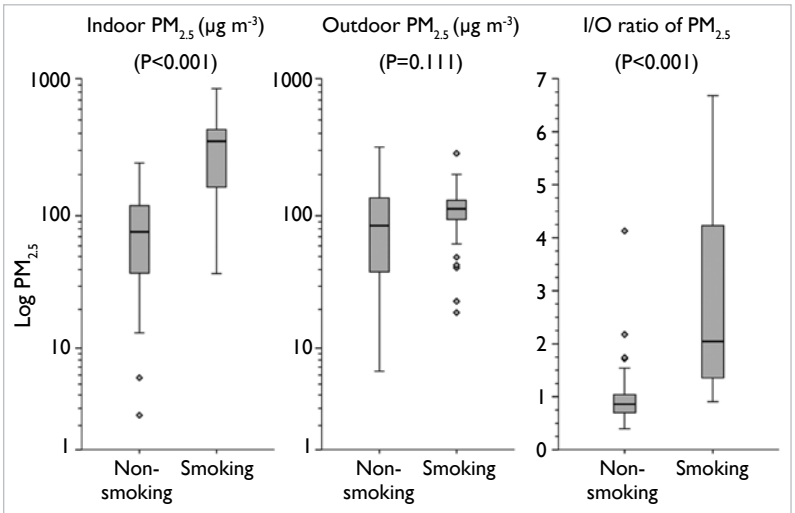

FIG 2. Indoor and outdoor $PM_{2.5}$ concentrations and indoor-outdoor (I/O) ratios at non-smoking (n=62) and smoking premises (n=27)*

* Boxes show the medians and interquartile ranges, and bars are 1.5 times the interquartile range. P values were obtained by ANOVA F-test (2-tailed)

using American Thoracic Society protocols. When compared with indoor $PM_{2.5}$ exposure ≤25 μg m^{-3}, the mean FEV_1 values in all non-smoking workers whose $PM_{2.5}$ exposures were 25-74 μg m^{-3}, 75-175 μg m^{-3}, and >175 μg m^{-3} were lower by 71 (95% CI=21-121), 78 (95% CI=24-132), and 98 (95% CI=14-182) ml, respectively. Similarly on this scale of $PM_{2.5}$ levels, mean FEF_{25-75} values were lower by 0.36 (95% CI=0.08-0.65), 0.50 (95% CI=0.19-0.81), and 0.56 (95% CI=0.23-0.90) L s^{-1}, respectively, whereas for FEV1/FVC, values were lower by 2.9% (95% CI=1.0-4.8), 3.2% (95% CI=1.3-5.1), and 4.3% (95% CI=1.3-7.3), respectively. When the analysis was applied to the subgroup of older workers much larger differences in lung function were observed with increasing levels of $PM_{2.5}$, reflecting their increased susceptibility to air pollutants (Fig 3).

Strong concentration-response relationships were observed between $PM_{2.5}$ and lung function values in all analyses. The tobacco-specific nature of these exposures is supported by the trend in lower lung function values with increasing urine cotinine levels, including FEV1 (P for trend=0.046) and FEF_{25-75} (P for trend=0.022).

After appropriate adjustment for age, sex and other relevant factors, workers in non-smoking venues had higher mean values for FEV_1 and FEF_{25-75} than those in smoking venues. The corresponding benefits were all relatively larger for the older workers indicating the benefit of relatively cleaner indoor air despite very high ambient outdoor pollution by international standards.

The excess mortality risk to Hong Kong catering workers from tobacco smoke can be assessed at two levels. First, by reference to the health based standards applied to air quality and ambient concentrations of particulates. The mean level of fine particulates in smoking venues (268 μg m^{-3}) is 2500% above the World Health Organization annual Air Quality Guideline for $PM_{2.5}$. It is estimated that per 10 μg m^{-3} excess short-term mortality risks are 0.21 to 1.3%, excess long-term all-cause mortality is 4% (95% CI=1-8%), and excess cardiopulmonary mortality is ≥6% (95% CI=2-10%). Second, we can estimate the risk of health outcomes, such as deaths from lung cancer and heart disease mortality from exposures to the total mixture of particulates and gases as indicated by the biomarker cotinine levels in body fluids (Fig 4).

If the catering workforce in Hong Kong totalled 217 985 distributed across non-smoking restaurants (190 970) patio-seating venues (21 219) and unrestricted smoking bars (5796), then the excess deaths attributable to SHS exposures, at the median levels would amount to 191, 83, and 162, respectively, accounting for 2% of all these workers or 2.7% of workers in smoking venues. In our sample of 204 workers, about 11 deaths were expected. The mortality risks for workers in the upper quantiles of the cotinine range are much higher (5-10%).

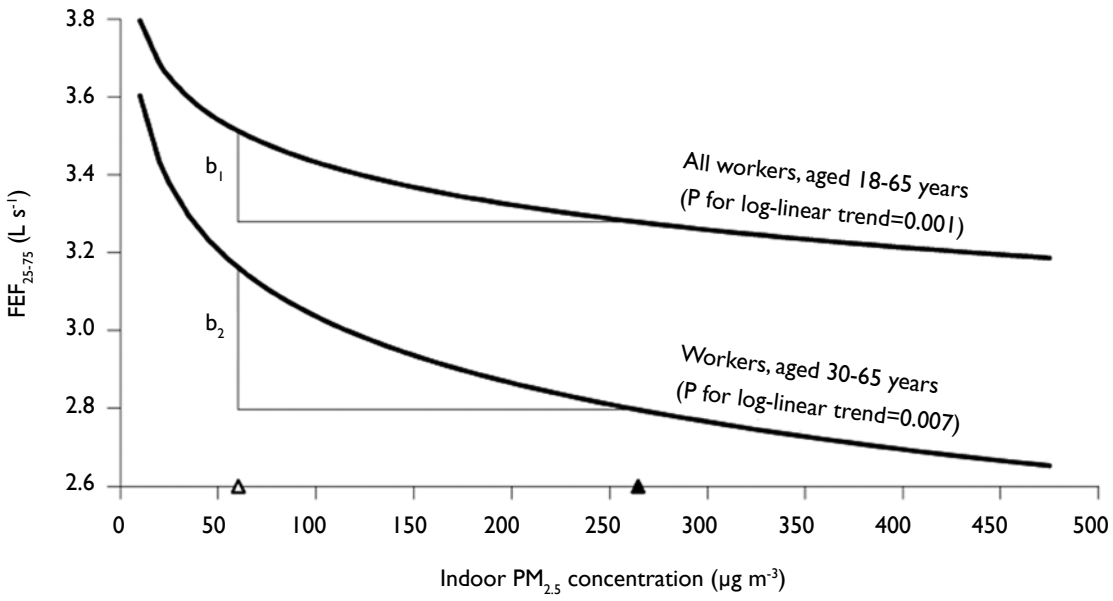

FIG 3. Estimated FEF_{25-75} per unit change of indoor $PM_{2.5}$ concentration among all non-smoking workers (n=186)*

* b_1=0.23, b_2=0.36, represent the differences in the estimated mean lung function corresponding to the mean indoor $PM_{2.5}$ concentrations, 60.3△ and 264.7▲ μg m^{-3}, in non-smoking and smoking premises respectively

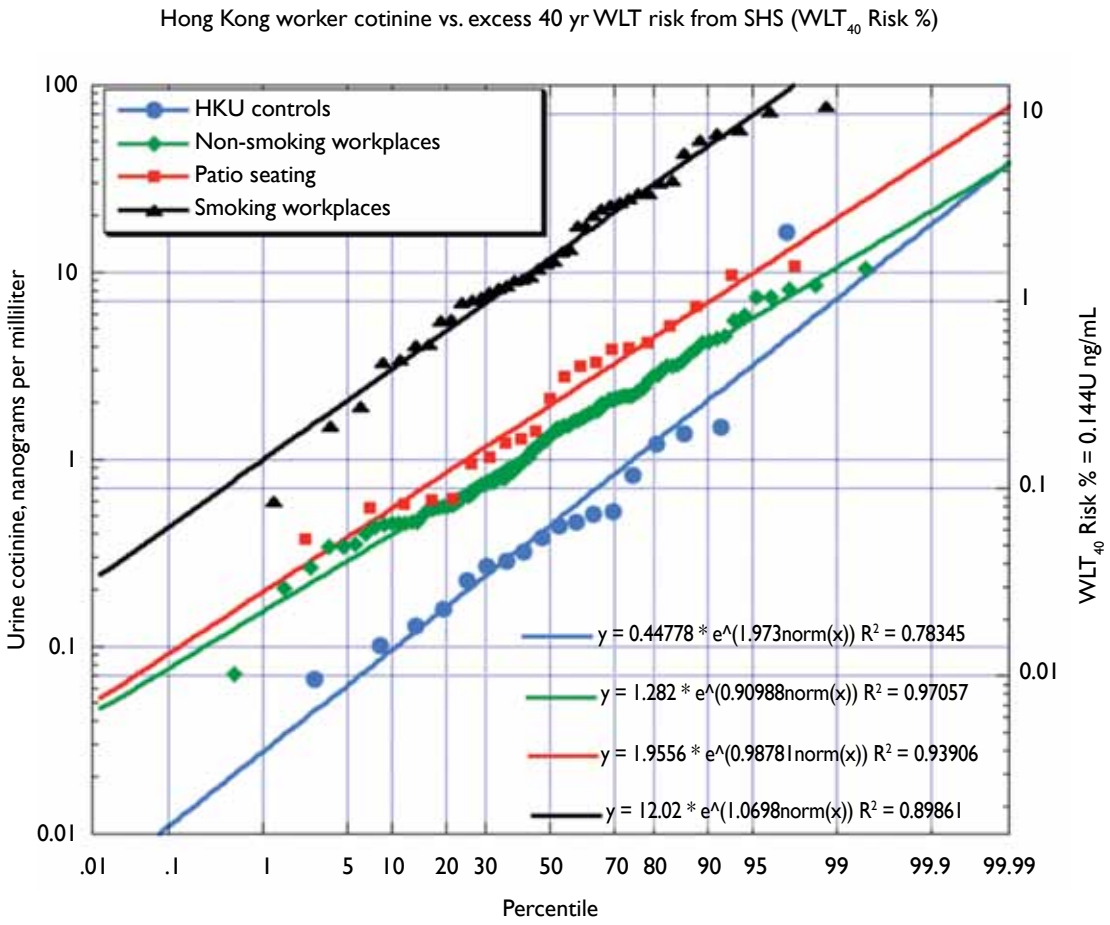

FIG 4. Log-probability plot of estimated working lifetime (WLT) risk for deaths from lung cancer and heart disease in Hong Kong workers versus urine cotinine by second-hand smoke (SHS) exposure group

Mortality risks represent the tip of a pyramid of bad health outcomes. Within the strata of this pyramid, many layers of increased health care needs and actions can be identified, from self-medication, recourse to traditional medicine and western practitioners, referral to specialist care, hospital admissions, and degraded quality of life from chronic illness.

Conclusions and implications

The 2006 Smoking (Public Health) Ordinance made an important contribution to the protection of many catering workers in their workplace. Levels of tobacco chemicals in smoke-free restaurants, indicated by fine particulates and urine cotinine levels, were reduced by up to 90% compared to the pre-ordinance period. However, exemptions from the Ordinance probably increased the intense SHS exposures in workers in exempted premises. These exposures, to chemicals known to cause cancers and diseases of the heart, blood vessels, lungs, and other organs are associated with higher risks of illness episodes, chronic disease, and deaths. The symptom patterns, degradation of lung function, and estimated excess mortality risks which we measured in bar workers indicate that permissive legislation in tobacco control is a causal factor for epidemics of cardiopulmonary disease. These bad health outcomes are a direct result of the exemptions in the 2006 public health legislation. Allowing exemptions was at variance with the established medical evidence of predictable harm. The failure of the public health system in this case study is a clear indication of the need for more reliable approaches to the translation of public health evidence into policy and practice.

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AUTHOR INDEX

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