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

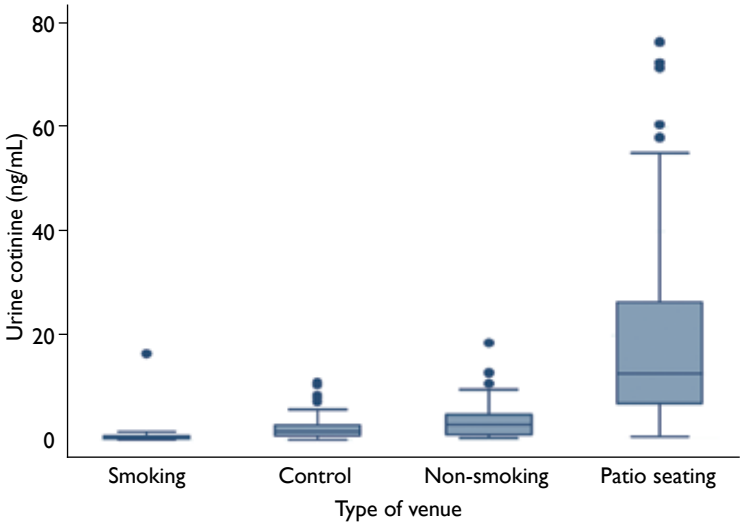

FIG 1. Urine cotinine in non-smoking workers

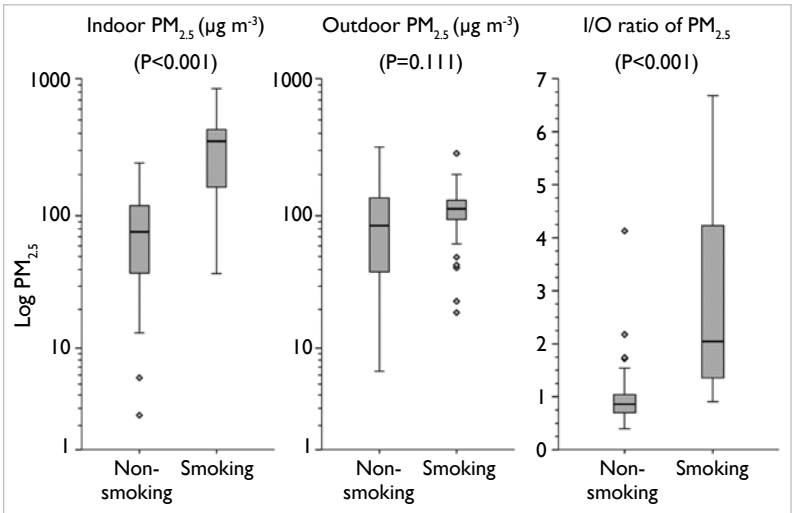

FIG 2. Indoor and outdoor $PM_{2.5}$ concentrations and indoor-outdoor (I/O) ratios at non-smoking (n=62) and smoking premises (n=27)*

* 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%).

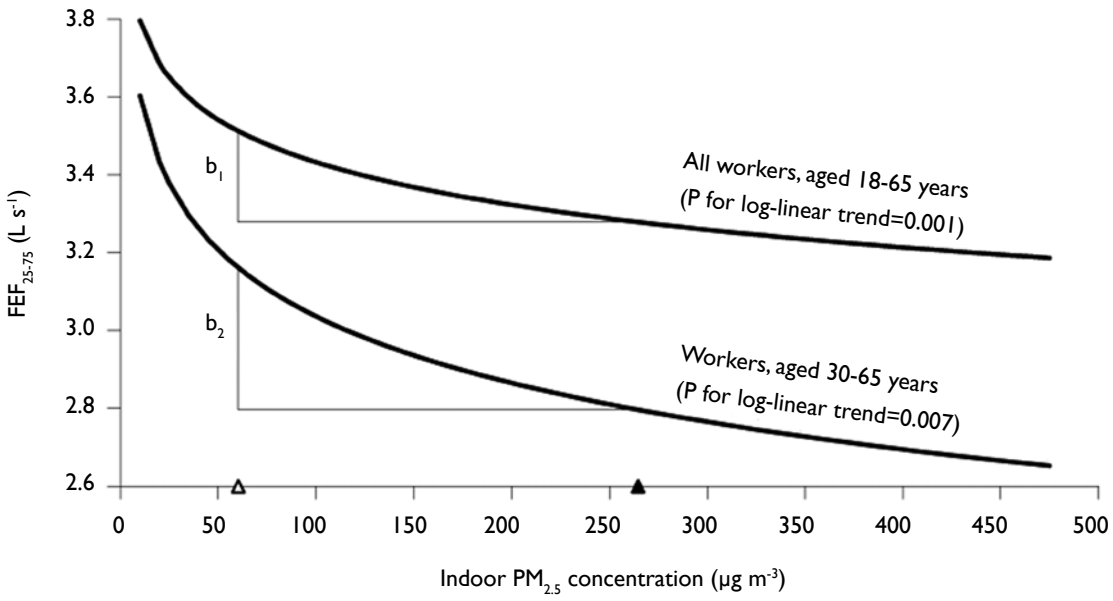

FIG 3. Estimated FEF_{25-75} per unit change of indoor $PM_{2.5}$ concentration among all non-smoking workers (n=186)*

* 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

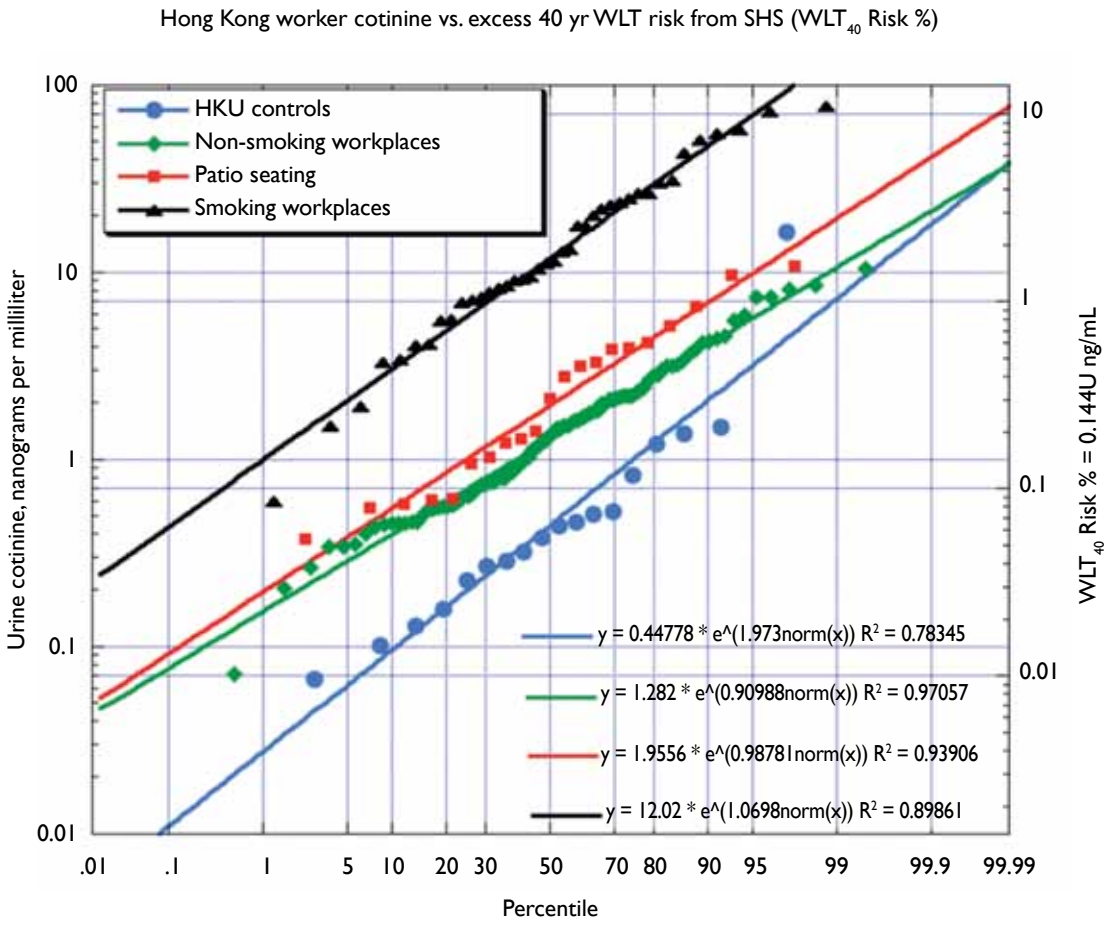

FIG 4. Log-probability plot of estimated working lifetime (WLT) risk for deaths from lung cancer and heart disease in Hong Kong workers versus urine cotinine by second-hand smoke (SHS) exposure group

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.

Acknowledgement

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